GASTRIC BYPASS WORKSHOP

April 28 and 29, 1977
Iowa City, Iowa

The University of Iowa
College of Medicine
Department of Surgery
GASTRIC BYPASS WORKSHOP

Presented April 28-29, 1977
Iowa City, Iowa
Under the Auspices of
THE UNIVERSITY OF IOWA
COLLEGE OF MEDICINE and DEPARTMENT OF SURGERY

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June, 1978
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WELCOME, RISK/BENEFIT IN OPERATIVE TREATMENT OF OBESITY.
Edward E. Mason, M.D.

Welcome to Iowa City. I am pleased to see the number of people that are here. We were a little concerned when we sent out a letter earlier that we might have too many people for this room. There was a bigger room that did not have very good acoustics. Then we started worrying a few weeks ago that maybe nobody would show up but now I am glad to see that we have a comfortable number here. You will notice that we had some rain this morning and this is welcome in Iowa because it makes the corn grow. We feed the corn to the pigs and pigs to people and then we get more gastric bypass candidates. Dr. Sidney Ziffren, the Chairman of our Department of Surgery, sends his regards and his welcome and we hope that he will be over for part of the conference. He couldn't get over this morning so I will give the welcome for him.

We knocked heads together a little bit last night. Some of the folks on the program and I never realized that there were so many different ways you could do things. I think maybe one of the highlights of this meeting will be to find out that there are a lot of different ways to do things. Perhaps there is no best way but hopefully before the end of the conference we can find out some better way or, at least, some of the ways of avoiding potential problems.

The title "Risk/Benefit in Operative Treatment of Obesity" is something that I hoped we could really grasp with some mathematical precision. I asked one of our faculty members, Skip Woolson, who is on our panel later, to think about this and we finally decided that we just didn't have enough data. We did not have the various factors to fill in the equations. Nevertheless, I am convinced that is the direction we should head and that eventually we should try to formulate an equation for risk and one for benefit. Of course, this is true of everything we do in medicine. The question comes up as to what to use for a measurement. I know that all of us have some dislike of measuring human life and human activities and happiness in terms of dollars but I suspect that this is the best way to do it. I know that you also don't like to think about legal matters but you are all very aware of the fact that the attorneys
think in terms of dollars. I have talked with one of our professors in the Law faculty and he is going to provide me with a bibliography and help to try to find out how these things are handled in the legal profession. A life is worth so many dollars. Pain and suffering are worth so many dollars and I don't see any reason why we can't use dollars as a scale. Dollars change in value, of course, but that is another advantage. We can always equate what we are doing in terms of dollars for the inflation and situation at hand.

There are certain competing risks that may cancel each other out. In other words, if you will excuse me for using a rather gross illustration of this, if a person has a huge panniculus with an ulcer on the bottom of it he has certain risks but he is not at risk of getting venereal disease. Or if a person has a very bad gallbladder and is very obese and he has to have an operation, he is at risk for cholecystectomy but he probably will not be at risk for gastric bypass. To put it another way, if you put a mortality rate or risk on a certain condition, the occurrence of that feared complication may take that patient out of the risk from other things. You can only die once. You cannot die from several things so there are competing risks. What I am trying to say is you don't add up all of the risks. On the other hand there may be certain benefits that cancel out or that can be included. If a person has a biliary tract disease in addition to their obesity and you do an operation that includes both taking care of the obesity and getting rid of the diseased gallbladder, this really has some advantages. You get the benefit of a single operation for two conditions, both of which may be dangerous to that person. I think this perhaps illustrates the complexity of the situation and also some of the things that have to be considered in setting up these equations. This is the direction we are headed and this is why I think we should be keeping track of these patients. This is one of the times in medicine with a new procedure and new people starting to use the procedure when we can keep track of what is happening to our patients and really find out what the risks and benefits are.

Now let me come to grips with some specific things about obesity. It is very difficult to find out what the true risk of being more than twice
one's ideal weight is as far as life is concerned. The insurance companies do have data indicating that there is a risk to obesity, but most of their information does not regard people that are this heavy. It would be nice if one could set up a randomized prospective study in which you would operate on only half of the people but follow them all for 20 years to see what happens to them. Unfortunately, that may not be feasible because people won't stand still for that. It is very difficult to get people that are at high risk to wait for 20 years to see what is happening while the people in the other group are seemingly being cared for.

The work that was done in Framingham, Massachusetts, over about 20 years following some 5,000 adult individuals has provided us with some information. I would like to observe that for the average weight of Framingham men the morbidity risk is set at 100. Being actually less heavy than the average for the population reduces the risk of angina pectoris. Being approximately normal weight makes the risk somewhat higher, being a little overweight makes it higher still. We don't have any figure here for the patient that is 100% overweight. Of course, that is what we are interested in.

The risk of sudden death begins to show up in people who weigh over 120% of this average population weight. As weight increases the difference in the incidence of sudden death is striking. There is a fourfold increase in risk. This is of interest to us because our patients do not suddenly become normal weight. Sudden death has occurred in some of these patients after we have operated upon them. It has even occurred before we operated on some of them, while we had them in the hospital.

When we started doing gastric bypasses in 1966 we were afraid to operate on people that weighed 400 to 500 lbs. We did not think it was feasible. Consequently, some of those patients were kept in the hospital at considerable expense. We had one patient that was in the hospital on an 800 calorie diet. The patient was receiving heparin in fairly good doses and had a massive fatal pulmonary embolus about 45 minutes after 7500 units of heparin. Of course, pulmonary embolus is one cause of sudden death but there are other people who die suddenly where the
autopsy doesn't show anything. One of our patients died suddenly about
24 hours after gastric bypass and after being ambulatory. This was
about our 60th patient. The experience was so frightening that we quit
doing gastric bypasses for a short time. We had some ideas about this
and so we scrounged through the literature and tried to find information
about the possible toxic effects of free fatty acids.

One of the students worked with me one summer and we measured free fatty
acids and also thrombus formation time with a little piece of polyethylene
tubing on a wheel that rotated. You could fairly accurately determine
when blood clotting occurred by the fact that the blood would no longer
flow in the tubing turning on the wheel. It would rise up to a certain
point and that was the end point for the thrombus formation time. We
found that there was an inverse relationship between free fatty acid
level and thrombus formation time suggesting that perhaps the rise in
the free fatty acids was stimulating blood clotting. The fatty acid
change in mEq/L and the relationship between this and thrombus formation
time was inversely related. We were able to obtain variations in free
fatty acid levels because at that time the patients were receiving
lactated Ringer's without glucose which was pretty common throughout the
country. Carl Moyer had been in our institution and around the country
promoting the use of lactated Ringer's in patients with major operations
and people took this up without realizing that patients also needed
glucose. As a matter of fact, everyone was aware of the fact that
operative trauma causes changes in blood glucose levels. It causes an
elevation in blood glucose but the point of it was that the blood glucose
level did not go up enough from endogenous sources. These patients
probably really needed a higher blood glucose during this post traumatic
period. There isn't very much glucose storage in the body. As soon as
they had used this up their glucose levels were not as high as they
might have been; the free fatty acid levels would rise and then you
would have some effect on the thrombus formation time.

Looking further in the literature we found other things that seemed to
happen with free fatty acids. There was a patient that was followed in
our psychiatric hospital because the doctors over there wanted to treat
the patient with starvation. We asked if we could get blood samples
periodically and we found that with full starvation and weight loss the free fatty acid levels were high. I am not sure what the toxic level is. You can certainly have free fatty acid levels that are higher than 1.5 mEq/L and it doesn't cause any harm. As a matter of fact, people don't die suddenly very often. This is an infrequent occurrence. So all of these things are consistent with but not proof of the hypothesis that high free fatty acid levels effect thrombus formation.

Another effect of free fatty acids is illustrated by a paper reporting the pathological changes in the lungs of humans dying with elevated free fatty acids in delirium tremens. This paper, written by Truman Mays, was based on work that was done at The University of Iowa by friends of mine in the Departments of Internal Medicine and Pathology. Focal atelectasis was present in humans dying with delirium tremens and in animals injected with high doses of free fatty acids. Intra-alveolar hemorrhage was also present in both situations as well as hyaline membranes, edema, and congestion. I wouldn't want to think for a minute that this proves anything but the suggestion is there that there are toxic effects of too rapid mobilization of fat or triglycerides, too rapid hydrolysis of those triglycerides to triglycerol and free fatty acids, and too high levels of free fatty acids so that they exceed the carrying capacity of albumin.

Other effects have been noted by a student in our institution who perfused isolated rabbit hearts and measured the effluent from the sinus venosus and observed that when free fatty acids were in the perfusate, there was no circulation through the myocardium. If the free fatty acids were in the perfusate with albumin so there was something to carry the free fatty acids, the myocardial perfusion was normal. You can have complete shutdown of myocardial perfusion, at least in experimental situations, when the level of free fatty acids is too high. Obviously this could cause death.

Another study that was carried out in this institution dealt with the injection of free fatty acids into dogs. Death with thromboses was observed. Another group of animals were heparinized and the experiment was repeated. The dogs still died. They had EKG changes and they died
but there were no thromboses. There were no findings pathologically and this was the stimulus for the study of the isolated rabbit heart. Excessive levels of free fatty acids conceivably can shut off coronary blood flow. There are other experiments with isolated strips of heart muscle which show that the heart muscle does not contract properly when free fatty acids are present.

Other studies show that free fatty acids are taken up with the cells in the lung that produce surfactant. The interference of the ventilation as the result of the loss of surfactant is probably the mechanism whereby focal atelectasis and some of these changes occur. A paper by Moore and colleagues indicates that after major trauma there is a relationship between plasma glucose and free fatty acid levels. It isn't a very good relationship. It's a scatter. Nevertheless, after major trauma you do have elevation of free fatty acids. The reason that I am stressing this is that we are subjecting our patients to major trauma. The effect of epinephrine is to cause a rise in free fatty acids. I remember a tragic incident with one of our patients. We take nude photographs of our patients preoperatively. We always ask permission for this because we feel it is worthwhile to have in the chart. I discovered that this patient had not had her photograph. So she went to the photographers and had her nude photograph on the morning that she was supposed to have her gastric bypass. She had a cardiac arrest during anesthesia induction and died. That doesn't prove anything but the suggestion is there that excitement, epinephrine release and maybe a lot of factors that we don't understand contributed to her demise. This is compounded by the fact that she had a lot of fat and triglycerides that could be mobilized. The combination of the starvation of the patient who is being prepared for operation plus the excitement, concern, and epinephrine release perhaps may have been the factor.

There is information about fatty acid levels in patients of various kinds. Again Truman Mays' paper shows that those patients that died with delirium tremens had high fatty acid levels, those that survived had lower fatty acid levels. I don't know if that is cause or effect. Somebody sick enough to die is likely to have higher fatty acid levels. Maybe it is just a coincidence. There are other studies in patients
with myocardial infarction that show the same thing. A high fatty acid
level is a bad diagnostic sign.

I have been talking about a risk of sudden death and maybe suggesting an
explanation. The risk is there. There is no argument about the risk. There is a risk of being obese and the risk can be great enough to
justify even an operation. I don't need to convince you of that or we
wouldn't be here. Another point that Dr. Soper will be talking to you
about later in the program concerns children with Prader-Willi syndrome
who do not survive to adulthood. The only thing that we know is wrong
with them, is that they eat themselves to death. They have lower intelli-
gence, they have poorly developed genitalia, they have things which Dr.
Soper will tell you about but the point is there are groups of people
who inevitably will die unless you find some way to control their eating.

Obesity is a disease that can be fatal. Why the insurance companies
will charge extra or not even give insurance to people that are grossly
obese and then turn around and refuse to pay for an operation that
treats that condition is beyond me. This is not cosmetic surgery. This
is a disease. It is potentially lethal.

There are other facets to the risk benefit equation and this, of course,
is what the whole conference is about. Let me just go into a few of
those things. We have repeatedly sent questionnaires out over the years
and we think we are beginning to be able to prepare a questionnaire that
may provide some useful information. Our most recent questionnaires
just came back in the last month or so. It compares patients having had
gastric bypass for varying periods of time. Preoperatively they remember
that they used to eat because of hunger about 12% of the time. Post-
operatively they eat because of hunger 36% of the time. They eat just
because it is a habit to eat at meal time or they eat to stay alive.
During the first year postoperatively they have had a change in reasons
for eating. They no longer eat because of nerves. I am sure that you
have elicited this from your patients. They eat when they get upset or
when anything makes them nervous. It is like having a security blanket.
After about a year this has changed completely and they no longer eat
because of nerves but rather because of hunger or to stay alive. I
don't know whether we could put that into an equation as a benefit but I really think that perhaps it is a benefit.

Before surgery the patients claim that they were never hungry 21% of the time and after surgery, 29% of the time. With respect to being hungry at meal time but not after, that changes from 14 to 64% and has persisted over the years. They seem to have a normal hunger at meal time which is something they didn't have before. They used to always be hungry and now they say they are just hungry at meal times. That is a change that perhaps you would want to put into the equation as a benefit. At least it is more consistent with why the normal weight people eat. In regard to the frequency of hunger some of them are hungry every two hours, some every three to four hours, some every five to six hours. During the first year 35% responded they were almost never hungry, while 7% were actually never hungry. In subsequent years they do develop some sensation of hunger.

During the first year they have trouble with meat. It appears that the meat fiber does not become liquefied and it plugs up the stoma. Over a period of time this improves. This is a negative benefit during the first year but it is not a permanent problem. Nutritionists would probably feel that the more fruits and vegetables that they eat, the better the score on the equation, the better the benefit. During the first year they don't eat very much in the way of fruits and vegetables but as time goes on they eat more and more fruits and vegetables. This is also an advantage that improves with time. We asked them how many snacks they eat a day. Again, during the first year they don't snack very much but as time goes on they become snackers. Of course they were snackers and volemics and ate all of the time and in every way before operation but during the first year after operation they don't eat much and they don't snack. Snacking is one of the things that can defeat the operation to a certain extent. So this is why some of the patients may seem to get a good response and then have to have a revision later on.

Other responses in the questionnaire indicate that dumping is not very common but it happens occasionally. Nausea and vomiting after meals
occur in almost all of the patients at some time. Although they are not frequent, they do occur occasionally in half of the patients. The striking thing to me about this is that a lot of these patients never learn. They continue to vomit even after several years. That is a warning that if you put the stomach back together they will just resume their former weight. I used to tell people that this was a Pavlovian training operation. That sounds nice but in reality the patients don't seem to learn.

Because of the interest of the medical profession in intestinal bypass I felt it desirable to ask about bowel habits. During the first year, the pattern is more one of constipation, less of diarrhea. As time goes on the constipation becomes a little less striking and some of the patients begin to have a little diarrhea. I think this is because they are able to eat more, the stoma stretches and they have a little more dumping. The dumping gives hyperperistalsis and, consequently, they have some postprandial diarrhea.

Other miscellaneous responses indicate that about half of the patients are on diets. About a fourth to a third of them are having some ankle swelling. About two-thirds of them are exercising regularly.

I have a series of results comparing the operation as we did it with the operation as it was done in another city. The other surgeon uses a big gastroenterostomy stoma, we have a small gastroenterostomy stoma. His patients have more dumping and diarrhea; our patients have less dumping and more constipation, and so on and so forth. You can get some valuable information in this way that will help feed into the risk/benefit equation, not only with regard to the effects of the operation but also the effects as to the way the operation is performed. A lot will be said about the varying ways of doing the operation during the next two days. What we also need to find out is what the real effects are on the patient, on his function, on his eating, on his bowel habits, and on his life.

QUESTION: In the Framingham study there was an increased mortality in obese males as compared to females. This was pointed out to me by some
of my medical colleagues. Do you have any comments as to why this is so?

Mason: I can't give you a good answer to that question. I think that it is illustrative of our ignorance and a need for good data. I would refer you to a book on obesity by Bray from UCLA that has recently been published by Saunders and Company. He has an excellent chapter about the risks of obesity. The folks at the University of Minnesota have written a lot about this but I suspect that much of the lack of information about this subject is due to the fact that there isn't any data available for people who are morbidly obese. They are usually talking about 20% overweight.
ANESTHESIA AND RESPIRATORY PHYSIOLOGY OF OBESITY
Azmy R. Boutros, M.D.

I intend to discuss the two major points. First, I shall talk about some cardiopulmonary physiology that might be pertinent to obese people. Then I will try to see how we can utilize some of this knowledge or information to appropriately and reasonably manage these patients preoperatively, intraoperatively and postoperatively.

Starting with the physiology, one question we wanted answered was what would be the effect of posture on very obese people. This was of interest to us because of the clinical impression we and other people had that these patients do much better sitting up than lying down. We had about eleven or twelve of these patients essentially wired for sound with Swan-Ganz catheters, arterial lines, etc., preoperatively. We measured various variables with them sitting up vs lying down. When people who are not obese assume the supine posture the cardiac output increases, there is a shift of the blood to the central channel and the peripheral resistance diminishes. However, oxygen consumption does not change. That is to say, when you and I lie down we get a shift of blood to the central channels, peripheral resistance drops but we do not really increase our oxygen consumption.

When we had the severely or morbidly obese people assume a supine position, we found that the peripheral resistance did drop significantly by about 22 or 23 percent. Heart rate also dropped, and although the fall in rate was small it was very consistent and significant. Pulmonary vascular resistance as opposed to peripheral resistance did not change significantly. As the cardiac output increased the implication was that the pulmonary arterial pressure also increased. We found the cardiac output increased by about 35%, that was very significant. The peripheral resistance decreased by only 23%. The reduction in peripheral resistance could not have explained the total rise in cardiac output. Oxygen consumption increased significantly by about 12%. It is our hunch that the combined effect of the reduction of peripheral resistance which occurs normally plus the increased oxygen consumption which we attribute to increased work of breathing was responsible for this fairly significant
rise in cardiac output. The effect of this on various hemodynamics was noticeable. The pulmonary artery wedge pressure increased by about 35%. In actual number, we were running a mean of about 9 mmHg in the sitting position and about 15 mmHg for people in the supine position. Fifteen mmHg is a fairly high figure. It is equivalent to 18 to 19 cm of water and is compatible with impending congestive failure. The QS/QT which is a measure of the shunt fraction, increased significantly indicating that the shunt in these people did increase. However, the A-a gradient, which is the easier thing to measure (the same applies to ordinary PaO₂), did not change significantly. The reason for this is that it is well known that the A-a gradient and straight forward PaO₂ are very dependent on cardiac output. We assume that the significant rise in cardiac output offset the shunt increase which was thus not reflected on the A-a gradient. A-v oxygen consumption was decreased in the supine position which is the expected thing.

I think we ought to realize that these changes are over and above the basic changes in obese people. These people have a higher cardiac output. They have increased work of breathing and all the changes described before are on top of that. No wonder they are very borderline. In our estimation we felt that the key thing that really made the difference with these people was whether their hearts were capable of taking care of that increased filling pressure. There is a marked increase in venous return and if the heart can take care of that through the usual compensatory mechanisms by increasing stroke volume or increasing pulse rate then all is well and good. In such a case the heart will be able to cope and not only will the patient be protected from sudden congestion of the lungs but he will be able to cover up any tendency towards a drop in PaO₂ due to the increased shunt. That is to say, if the heart can take care of the preload the cardiac output increase will cover up the hypoxia. I feel that in patients who have borderline myocardial insufficiency the heart might not be able to rise to the occasion and take care of their increased preload, the cardiac output would not rise that much. The increased shunt would immediately reflect on the A-a gradient and PaO₂ causing manifest hypoxia. This in turn depresses the medullary centers or other respiratory centers and produces the increased PaO₂ or CO₂ retention. This is a rationalization of how a Pickwickian becomes
stuporous and hypercarbic. I don't know whether this is true or not. It is just another hypothesis.

The second item I want to talk about is, I think, very pertinent to the management of these patients. This is the closing volume. Closing volume is a term that has become fashionable in the last few years. It is the volume of the lungs at which small airways close during expiration. At that point small bronchules and small alveoli and alveolar ducts start to collapse which increases the work of breathing and causes wheezing and so on. The thing that is significant here is that the closing capacity at any one time does not change really from minute to minute or from hour to hour. What does change is the functional residual capacity (FRC). Anything that shrinks the FRC is going to allow the closing volume to be higher than the FRC. In such a case, instead of wheezing during exhalation of a large breath, the patient is going to start to wheeze when he is in the normal resting expired position. As the FRC drops further that patient is going to have the majority of his airway closed and this constitutes a major resistance to breathing. Worse still, as you close the airway you eventually produce alveolar collapse which really cannot be reopened by just a regular breath but must be reopened by a big inflation. The method that I give our residents and the method that I follow myself in obese people is to try as much as possible not to let the FRC drop.

What causes the FRC to decrease? Obesity by itself decreases the FRC and increases the closing capacity. They are already fairly close to the point at which normal ventilation is within the closing capacity range, and this is bad. Pain, intraoperative retraction, packs under the diaphragm, and a whole host of things produce a reduction in FRC. This is something that I think we ought to keep in mind at all times. By changing the posture from the sitting to the supine position, the expiratory reserve volume (ERV), shrinks dramatically. Once this shrinks the patient will start to wheeze. This is another thing with which we ought to be careful.

In regard to anesthetic management, preoperatively we like to do pulmonary functions on these patients. Although we measure vital capacity the
things we go for are the flow measurements. The one second vital capacity, maximum expository flow rate, etc., are also important because these indicate obstructive elements in the patients. We like to get blood gases. We like to document cardiac status mainly because of the previous assumption that the heart is a key factor in taking care of the preload and if this is not taken care of the patient, in addition to being hypoxic, is going to be in borderline congestive failure as well. We try to eliminate or make sure that we do not have a patient with very borderline cardiac status.

We try not to premedicate too heavily. If the patient has chronic obstructive lung disease or if he is running a high PaCO₂ he will probably be borderline and you do not want to knock him out too much with morphine. If the patient is a chronic obstructive lung disease patient, I think I would like to check the SMA 6/60 to see what the serum chloride is. As the CO₂ increases in chronically hypercarbic patients the serum bicarbonate also progressively increases as per the Henderson-Hesselbach equation. At the same time the chloride keeps falling and in most cases you will find that this is a mirror image of the rise in bicarbonate. For example, mEq for mEq you might find a serum bicarbonate of 34 and a serum chloride of 90. In acute hypercapnea, if this has been due to a sudden rise of CO₂ the effect on pH is fairly devastating, but if this is chronic hypercapnea, as with obstructive lung disease or obesity, you will find that at the same PaCO₂ level, the pH is reduced less because of the bicarbonate-chloride exchange in the kidney. I am telling you all this because sometimes people get compulsive about correcting a chloride of 85 or 90 preoperatively. I think this is wrong because as soon as you correct the chloride you are going to lower the bicarbonate and let the patient be a sitting duck for a high PaCO₂. It is also important to know how much bicarbonate there is because if you start to ventilate these patients heavily you will have to expect that the PaCO₂ will drop, leaving a high bicarbonate level, with a very high pH which might produce arrhythmias.

As Dr. Terry indicated, we too like to put in arterial lines for blood pressure monitoring and for blood gas measurements.
Intraoperative Management and the Airway: The majority of people like to intubate these patients while awake because of the fear of putting them to sleep and then not being able to get an airway. I think this is reasonable and valid although sometimes it is rather difficult to stand and watch these patients suffer, especially if the anesthesiologist is not trained well enough to intubate blind without too many problems. At times, if I can put a mask on the face with the patient awake and I know I can get a good mask fit, I take them down by inhalation agents without paralyzing them. However, if in doubt, it would probably be safer to intubate awake rather than run the risk of having a patient who is obstructed with no airway.

The Anesthetic Agent: I don't think that anybody can dictate what anesthetic agent to use. The best anesthetic agent is that one which is best in the hands of that particular anesthetist. Muscle relaxants are helpful, not as much in producing muscle relaxation which is useful, but in trying to diminish the possibility of myocardial depression. Again, we have the patient in a supine position with a low FRC. If you suppress myocardial contractility with a deep anesthetic like Halothane you might find the pulmonary artery wedge pressure going up quite a bit and you might border on pulmonary edema.

Another thought is ether. I know that ether is a dirty word and maybe we shouldn't use an explosive agent. However, if you have somebody who knows how to give ether anesthesia, the patient will not be curarized or muscle relaxed. It is not cardiac depressant in anesthetic doses and it produces excellent working conditions. But, I suppose if I stood up at a national meeting and said that, they would probably throw me out of the meeting room and maybe the society altogether.

Pattern of Ventilation: With respect to the pattern of ventilation, we like to ventilate the patients mechanically intraoperatively. It is very hard to be able to deliver by hand all the tidal volume that is needed to maintain a PaCO\textsubscript{2} around 35 to 40. We like to use a tidal volume of about 10 ml/kg. We usually use more than that in thin people but considering that the body weight in fat people is not all lean mass, we go by the lower level. If you have somebody weighing 100 kg, you are
going to be ventilating with a 1,000 cc. We like to use PEEP to keep the FRC expanded. We are saying to the lungs, "O.K., we are not going to let you breathe out all the way -- we are just going to hold you a little inflated so that the FRC does not reach the level of critical closing volume and result in a collapsed airway."

Postoperatively, I think we are learning very fast. I was pleased and encouraged that most people that I talked with have almost identical ideas about the necessity for early extubation. As to the necessity for early ambulation, there is no disagreement. If the patient is awake and uncurarized and I ask him to cough and he does, I would be tempted to take the endotracheal tube out and let him have his own airway. If there is any doubt in my mind, I would leave the endotracheal tube in place. However, we have found that if you leave an endotracheal tube in the trachea and allow the patient to breathe against no resistance, these patients are at a great disadvantage. They cannot cough, they cannot purse their lips, they cannot do anything. They are sitting ducks to an open airway and that is devastating. Instead of this we recommend that rather than using a T-tube adaptor or something similar, we put them on a tight bag. We hook them up to the attachment of the endotracheal tube with a line of oxygen under an ordinary underwater seal for a chest drainage. This produces about 5 to 8 cm of expiratory pressure. You need about 15 liters of flow so that there will be no CO₂ retention as the patient is breathing through the system. As you increase the airway pressure obviously you are increasing the FRC but at a price. The price is that you might increase intrathoracic pressure and reduce venous return. This might be a good thing in a patient who has a high venous return but in a patient who might be shocky it is not very wise. If the patient is still curarized or we have any doubts in our minds, we continue to ventilate them the same way we do intraoperatively with a volume ventilator rather than a pressure ventilator.

Posture: We try to sit the patients up and ambulate them as early as possible. Pain is one of the things that reduces FRC tremendously. We have noticed in these patients and in other patients with chronic obstructive lung disease that whereas giving morphine preoperatively in the absence of pain can produce a rise in PaCO₂, when given in the
presence of pain morphine makes them able to breathe a little bit better and they improve both their \( \text{PaCO}_2 \) and \( \text{PaO}_2 \). Fluid intake is also very important. We lost a fair number of patients, not obese patients but other patients, because we did not realize earlier on that patients on the mechanical ventilator cannot handle fluids as well as patients breathing spontaneously. If the patient is breathing spontaneously give him whatever you want to give him as usual. If the patient is not breathing spontaneously, I think you ought to restrict the intake for the time being. The formula we go by is 1 mL/kg/hr so that if you have somebody weighing 100 kg you would give them about 100 cc/hr for the duration of the time when they are on the mechanical ventilator. Finally, we like to follow closely the blood gases, serum and urine osmolality, free water clearance, and osmotic pressure measurements which are currently fashionable in place of serum albumin levels. Obviously we need to know what the electrolytes, both in serum and in urine, are, to allow us to know what kind of drugs to give. Thank you.
RESPIRATORY INSUFFICIENCY
Boyd E. Terry, M.D.

I would like to refer to a specific group of patients who have respiratory insufficiency, the typical Pickwickian patient. We, who have done this procedure, recognize certain elements of Pickwickian disease or symptoms in many of our patients. This particular group of patients has exhibited a special challenge to me and my experience in terms of getting them ready for an operation and getting them into a position where my anesthesia colleagues feel comfortable about working with them. In our experience this has been about 5 to 7% of the patients that we have seen out of approximately 100 patients. Morbid obesity complicated by pulmonary and right heart failure and the profound effects of obesity on pulmonary cardiac function that was first noted 25 years ago presents a challenging problem to the physician. This is a group of patients who were obese, somnolent, plethoric and edematous with alternating periods of cluster breathing and apnea. They had cardiac enlargement, polycythemia, hypoxemia, hypercapnea, loss of vital capacity and maximum voluntary ventilation. It appears that the pulmonary and cardiac symptoms improve with weight loss. It is noteworthy that in this group of people as they become heavier, they exercise less. Even though we can put them in the hospital and starve the weight off them, they always regain it after discharge. Perhaps a vicious cycle is initiated because of the inactivity.

The cardiac failure makes them inexorably more weak. Medical therapy is usually ineffective on a long term basis. Sometimes when these people come in it appears that they may have an actual structural heart disease. They may come to cardiac catheterization and they are found to have extremely high wedge pressures and pulmonary artery pressures. This is in contrast to the usual patient that may be having dyspnea and exertional problems in the typical morbid obesity that we see. Individuals managing these patients tell me that when they see pulmonary hypertension associated with structural heart disease or pulmonary disease of a significant degree they give a patient perhaps six months to live. Their mortality would be at least 50 percent within six months. I think that this might be overstating it for this particular group of patients because they are being compared to patients with structural cardiac disease or fibrosis.
Nevertheless, they rival this group of patients in terms of pulmonary hypertension.

Many surgeons and anesthesiologists, therefore, are reluctant to accept the risks of this type of patient. It is our contention that this patient can be helped if we strictly adhere to certain principles in preparing the patients. They can be operated on with a significant improvement that guarantees a certain weight loss that is not reversible and which gives them a new lease on life. We find that this can be done with reasonable risk. Dr. Mason has talked about risk vs benefit and when I talk to patients about this I put out the scale of justice. On one side is obesity and the other side the risks that I would inflict upon them in doing the procedure. I think in this group of patients it has been very easy to recognize that the risk of obesity and complications are high. At the same time it would appear on the surface that it is extremely risky to do an operative procedure on them.

Within the past five years we have encountered about seven of these individuals that have had significant degrees of pulmonary and cardiac failure. Their obesity is at least two times normal body weight. They have hypercapnea with a CO$_2$ greater than 45 mmHg. They have hypoxemia with a P0$_2$ less than 60 and we frequently see these people with a P0$_2$ in the 40s. Often we can't get it up even if we give them oxygen. It will just not go up even with strict respiratory therapy. They have right heart failure, exhibited typically with the edema or with clinical evidence of right axis deviation and elevated right heart pressure. Recently when we encounter this condition we put in a Swan-Ganz catheter through a supraclavicular route and monitor the patient preoperatively, intraoperatively and immediately postoperatively. We have one individual that is 36 years old, female, and has had several admissions because of severe right heart failure. She was spending a good deal of her time in the hospital. Her weight was 300 lbs. Much of this was wet weight that was recognizable. She had right axis deviation.

Chest x-ray showed cardiomegaly. Cardiac catheterization was done showing the pulmonary ventricular wedge of pressure of 12 mmHg, pulmonary venous pressure of 55/10 and PA pressure, 55/30. She had evidence of
chronic stasis in her legs. After a prolonged hospitalization and
treatment with digitalis and diuretics we were able to get her from 49
up to 59 PO₂ on room air and felt at this time, if any, was an optimal
time to intercede. She had an intestinal bypass procedure and has done
well subsequent to that time. She has not required further admission
for cardiac failure.

You might say this is a reasonable way to approach this, to set these
people off to the side and let them get into a position where it seems
safe to operate on them. My experience has been that some of them are
so acutely ill that you set them in the hospital for a while at con­
siderable expense. They improve enough to leave the hospital. People
breathe a sigh of relief thinking we have gotten over this hurdle. But
a couple of weeks later they are right back again. This is their life
for years and years.

I am trying to make the thesis that even though the patient may look
very bad, if one takes certain precautions, one can interrupt this cycle
at an appropriate time without undue expense and hospitalization.
Another individual came to us after having an upper respiratory infection.
He is a long term smoker and has pulmonary emphysema in addition to the
problem of right heart failure associated with obesity. He was given a
tracheostomy because of his extreme condition. He arrived at our hospital
weighing about 380 lbs. I was quite concerned about even considering an
operation on him. It took a certain amount of time to get him in optimum
condition. He felt that he could never reduce. We said that it was
worth his life to reduce and that he better do it. He said that he
would just as soon die because he had tried losing weight for years and
years and he could not do it. That is a familiar story and you know
that it is difficult to make judgments on that. Sometimes we are tempted
to take the bait but this individual was operated and lost over 100
pounds to 230. At this time he was in a much better position and was
able to work and get around. His PO₂ increased two years postoperatively
to the 61 level, PCO₂'s were down to normal range and his pulmonary
artery pressures had returned to almost normal. His wedge pressures
also seemed normal.
Rochester, at Columbia University, has studied some of these morbidly obese patients and he has been able to categorize them into two groups. You might look at them and they would appear the same in terms of pounds per inch but they behave differently in terms of their cardiac and respiratory functions. Those who have this cardiac condition are those who are Pickwickian and it doesn't seem to be purely weight dependent.

Some of our patients have had gastric bypass, some of them intestinal bypass. More recently we have been doing the gastric bypass. We see an improvement in the vital capacity in virtually all of the patients as their weight drops. Dudley Rochester looked at the obese group which, pound for pound, is about the same as the group that has obesity hypoventilation syndrome. As they lose weight the vital capacities change upwardly in this group. At the same time there is very little change in the merely obese group. I don't want you to derive that these people (the morbidly obese) don't do well; it is just that they weren't having a problem to begin with in their vital capacity and consequently, don't improve that much. I think this as well as anything depicts the type of problem that I am trying to illustrate in this talk today. The forced expiratory volume at one second (FEV1) has been shown to improve similarly with weight loss except in one patient. In the case of this specific patient improvement did not occur and seemed to be related to a restrictive element of his pulmonary disease which was similar to pulmonary fibrosis. Most of these people do not have increased respiratory resistance associated with emphysema. This particular patient did. His vital capacity did improve but the FEV1 did not. The PO2's likewise improved and the PCO2's declined in all cases.

Morbid obesity complicated by pulmonary failure permits only a sedentary life, thus leading to even greater obesity. The etiology of the pulmonary failure seems to involve several concepts, none of which are completely pinned down right now. The chest wall compliance has been measured to be one-third of the normal in this group of patients. Lung compliance likewise has been shown to be markedly depressed. This could be due partly to the increased pulmonary blood volume and the increased total blood volume. I noticed, and perhaps some of you have, that most of my patients have spleens that are two to three times enlarged. I think it
is a reflection of the total blood volume. Actual measurements on pulmonary blood volume and total blood volume have shown them to be increased. If one exercises the person with something such as leg lifts one can overload the pulmonary circuit fairly easily which already has a maximum volume and thus encroach upon the ventilatory space. I think this is an attractive concept which is fairly simple. The work of breathing and the oxygen consumption are both increased. Whether or not there is a respiratory muscle weakness is unproved.

Rochester at Columbia University has measured static inspiratory pressures and they were 60 to 70% of normal in the hypercapneic obese in spite of the absence of other specific disorders. The role of the respiratory center in producing hypoventilation is poorly understood. Ventilation perfusion defects are present in people with Pickwickian syndrome quite common in the obese and could partially account for the low \(\text{PO}_2\) and the hypercapnea. Once hypoxemia and acidemia are established, they lead to increased red cell mass and to salt and water retention which further aggravates the already overexpanded circulatory system. Acidosis also produces pulmonary artery vasoconstriction in the small muscular arteries. The vasoconstriction plus the increased load contributes to the pulmonary hypertension and right heart failure. Weight loss can specifically reverse these factors and we have witnessed this to be very helpful.

Operative intervention can be undertaken by strict adherence to certain principles. As mentioned before, the preoperative stabilization of pulmonary and cardiac functions is mandatory. An adequate airway is essential and must be assured from the very beginning of the operation. I always like to be present when this step occurs. My anesthesiologist colleagues who do a very fine job do an awake intubation, particularly on the larger individuals. The tube is passed in with topical anesthetic and before any kind of anesthetic induction we can have control of the airway. It is also very important to have intraoperative monitoring of blood gases. We do this on all of the patients.

We have had one early postoperative mortality in our group. He had a myocardial infarction in the late postoperative period. He had had wound separation. We returned him to the operating room on the tenth
postoperative day and fixed the wound separation. He required several
days on the respirator and was just getting ready to come off the respira-
tor when he had a myocardial infarction. None of the others have suc-
cumbed to this procedure.

Another of our patients, a truck driver, was referred by a friend. The
friend said that he wouldn't drive across town with that fellow because
he went to sleep so frequently. He had wrecked two or three large truck
rigs. This individual had the gastric bypass. He had a PO₂ in the 40s.
We got him up to the low 50s. Postoperatively after losing 100 lbs his
PO₂ and pulmonary functions were markedly improved. His PO₂ was in the
70s. He is now back to work.

What I have tried to show in this rather trepidaceous group of patients
with alarming parameters of cardiopulmonary function is that if we pay
attention to them and do the best we can preoperatively in drying them
out, that we can operate on them. We can do this without greater risk
as one might anticipate by the actual values and see improved function.
Thank you.

QUESTION: Have you tried other nonoperative methods?

ANSWER: Other people, Alexander and Rochester, have shown that you can
starve these people or fast them, it may take six months or more. If
they lose 30 or 40 lbs frequently their hypoxic index will improve. But
the point I am trying to make is that you can't hold them that way
forever. You can't set them off and say that this is going to cure
them. They just keep coming back and it is very expensive. I am aware
that the first criticism will be that you don't have any right to operate
on these people, you can fast them and they will get better. Maybe you
should operate on them after you have fasted them. I think that that is
a valid thesis if the patient can afford it. I happen to feel that one
can go ahead with operation once you get initial stabilization and do
quite well.

QUESTION: Dr. Terry, could you more specifically indicate what you do
to stabilize them before operation?
ANSWER: They may need digitalis and diuretics. When they are in bed their legs are elevated. We stop their smoking and often give them inhalation therapy treatments. At the same time they may be on a reduction diet. These are fairly straight forward things. I think you might measure my eagerness to do these as being an impatient surgeon, wanting to get things over. However, some of these people just do not get the attention that they might require from internists. They will say, "Well, we can make them better by losing 20 or 30 lbs and we will do just that," then the patients go home, and before long comes back and the same thing happens all over again. The patients do not get good care, they get worse in a spiral of complications in addition to the morbid obesity which we deal with every day.

QUESTION: Do you do anything about the polycythemia if this is severe or do you just use this as one of the criteria of readiness for operation?

ANSWER: I just regard this as a criteria that fits them into this group and I look upon the polycythemia as a physiologic necessity. I don't want to diminish it in any way. We try to do them without much blood loss and as far as thrombotic problems associated with polycythemia I haven't given it as much concern as one might. I haven't seen any problems with it. It is a small group to make any conclusions about.

QUESTION: Do you use anticoagulants in your patients?

ANSWER: Not generally. It is not my routine to use them. I may want to use them in some patients who have a venous ulcer or significant stasis disease or evident advanced disease. I have, on occasion, but I have had as many complications with the anticoagulation as I have had with not using it. Actually, I have more in terms of wound problems.

QUESTION: Do you use intermittent positive pressure breathing?

ANSWER: The patients on a respirator don't mobilize very well. I don't use IPPB because people take a volumetric respirator. IPPB is hazardous.

QUESTION: I mean after they are operated do you give them IPPB?
ANSWER: They may be assisted with that. Remember that they have a gastric bypass. You don't want them to swallow lots of air and I tend to stay away from that. We just get them up early, ambulating them using deep breathing exercises, percussion and other things like that.
You may well wonder what business a pediatric surgeon has addressing a conference on morbid obesity and I guess there are at least two lines of defense I could mount to justify that. The first is that we all know morbidly obese young adults don't get that way overnight, but often after they have been morbidly obese juveniles or adolescents and perhaps even grossly obese school children. Dr. Filer and Dr. Fomon in our Pediatrics Department are among a number of people trying to correlate infant formulas and the fat American baby with later cholesterol and obesity problems; this is as yet an incomplete chapter in the story that we are going to unfold today. I don't plan to address myself to this aspect of the input of the pediatric surgeon to the problem of morbid obesity, but rather to the second input that we might have which relates to a fortunately rare and very difficult childhood syndrome of which morbid obesity secondary to hyperphagia is one of the clinical expressions, perhaps the one of greatest threat to their life. This syndrome is referred to by a number of different names. As listed in your program, the term Prader-Willi syndrome honors the two pediatricians in the Zurich Children's Hospital who first recognized and wrote about this syndrome. It is also referred to by other mnemonics according to the different clinical expressions that it carries with it, which we will go into later on.

We are fortunate in having Dr. Hans Zellweger at The University of Iowa in our Pediatric Department. He was a colleague, classmate and teaching associate of Prader and Willi during their early Zurich years. He is an internationally renowned geneticist interested in all sorts of peculiar childhood syndromes of which the Willi-Prader syndrome is one. By virtue of his stature and presence, we have an unusually large number of these children. About seven years ago we decided that it would be a reasonable clinical experiment to evaluate the effect of gastric bypass in ameliorating or controlling the morbidly obese part of this syndrome which is the most lethal to their well being and longevity.
This morning I plan to summarize for you the experience that we have had with eleven children with the Prader-Willi syndrome, and try to put into perspective the place of surgical treatment for controlling the obesity in this unfortunate group of children. The Willi-Prader syndrome is characterized by hypotonia, hyperphagia, hypogonadism and obesity; because of mnemonic overtones it is referred to in some circles as the "H_3O disease" or the "H_2O disease" if you drop the hypotonia, or the "water syndrome". I will refer to it for better or worse as the Prader-Willi syndrome. Hyperphagia and obesity are two of the cornerstones of this syndrome, and it is to these that we shall address ourselves this morning. Since this syndrome was first described by Prader and Willi about 20 years ago at least 100 cases have been recognized and recorded in the literature in Western Europe and in the past decade or so a good number of cases have also been reported in this country. This syndrome, because of the hypogonadism and location of the gonads in the male is better recognized in males than females and this probably accounts for the sexual predilection toward the male in the cases that have been recorded. Females, indeed, have hypogonadism, but it is somewhat more difficult to identify in the gonad of the female.

The etiology of this syndrome is, to say the least, obscure. There are at least two sets of siblings reported with the Willi-Prader syndrome, which of course suggests some kind of autosomal recessive means of inheritance. The chromosomes have been looked at and in about 10% of these unfortunate youngsters there are indeed some major chromosomal aberrations. On the other hand, in the other 90% the chromosomes appear to be perfectly normal. Because of the hyperphagia this syndrome suggests a hypothalamic disorder, and some individuals are looking into this to explain the hyperphagia and obesity. Anatomically the thalamus in these youngsters at autopsy is perfectly normal, so I think we will have to rest the case by saying we really don't know the exact mechanism of inheritance, if it indeed is inherited. It is a rare enough syndrome so that we don't have enough kindreds to study to really map out the inheritance pattern.

There are two different methods of expressing this syndrome. In infancy it presents a totally different picture than it gradually assumes during
childhood and adolescence. During the neonatal period of life these are very floppy babies. They are depressed. They breathe poorly and with difficulty, and occasionally require respiratory support and sometimes succumb to this element of their problem. They have diminished reflexes, both the marrow reflexes and the deep tendon reflexes. The suck is weak, they swallow poorly and so, paradoxically early in life nutrition is a problem with them. They do not exhibit the normal movements that babies ordinarily do and the body temperature is erratic. In the male babies these symptoms can easily be correlated with the very underdeveloped scrotum, small penis and absence of testes so as to correctly label the male babies who have this syndrome. Lacking these characteristic external genital markings, the females are less easily recognized during infancy. Respiratory support is often necessary, as well as nasogastric feedings to support them nutritionally to tide them over the stormy infancy period.

However, during preschool years the syndrome changes. Although they still retain diminished psychomotor development, they begin to breathe satisfactorily and certainly their eating patterns change. They all are subnormal mentally, with IQs ranging anywhere from 40 to 80; and their mental retardation understandably makes managing the obesity very difficult. Their eating habits quickly get completely out of hand; these youngsters will go to any extreme to get food to eat. If they can't get into their parent's pantry or refrigerator they will get food from garbage cans or steal it from neighbors. They are absolutely compulsive eaters, which has been a continuing problem after gastric bypass that I shall come back to later on.

Prader-Willi patients have diminished body stature with delayed bone development and growth, and almost invariably they have many tooth caries. About a third of them are diabetic and this, of course, associated with their mental retardation and obesity, represents a significant therapeutic problem. The morbid obesity and late respiratory and Pickwickian changes which Drs. Terry and Boutros have talked about this morning become, as time goes on, an overriding and continuing problem of increasing magnitude that ultimately results in their death. The majority of Prader-Willi patients die in the second or third decade of life; the
oldest recorded patient that I am aware of expired at the age of 31 years with a Pickwickian type of problem. A look into the unwholesome looking mouth of one of these youngsters will usually show that almost every tooth in his mouth has been decimated by the caries which is typical of this syndrome. The oligospermia is invariably a part of the gonadal biopsy in boys and there are analogous dysplastic and hypoplastic changes in the female gonad. A prototype of the Prader-Willi syndrome in an early publication by the man who first described it shows a type of body fat that is developed mostly around the torso, less so on the extremities. The facies reflects mental retardation. Again, the grotesque bodily proportions are evident at the many different ages. Growth charts of these patients indicated that most of them fall below the 50th percentile for accepted weight during the first two or three years of life, but rapidly thereafter there is a sharp upward inclination in the weight curve that can assume staggering proportions. In contrast in terms of their height for age they almost invariably fall below the 50th percentile, reflecting the small, short stature which is part of the syndrome.

We had one 19-year-old female with the Prader-Willi syndrome who was in desperate straits with hypercarbia, acidosis, heart failure, fluid retention and the Pickwickian type of picture that we have talked about today. She was in the hospital for many weeks before she became even an acceptable operative risk for gastric bypass. As an example of some of the problems related to their mental status, this youngster on one occasion postoperatively picked up a full tube of K-Y jelly and ate the whole thing. This is representative of the intense appetite drive which is absolutely uncontrollable in these youngsters.

Six of our eleven Prader-Willi patients who underwent gastric bypass were male. The mean age at operation was 14 years; please bear in mind that youngsters between 8 and 14 or 15 normally have increments of body weight gain which are to be expected. This is part of the problem in interpreting our gastric bypass results, to which I shall return later. Their weight at the time of operation averaged two times normal, if you consider the 50th percentile of weight for age as accepted normal,
ranging from $1\frac{1}{2}$ to 3 times normal; the mean preoperative weight in kilograms was 100, ranging from 42 to 163.

In regard to the technical details of the gastric bypass operation on these eleven patients the anesthesia time averaged just under four hours using the old fashioned technique for gastric bypass. We will get into technique this afternoon, but these eleven patients underwent the standard gastric bypass techniques before recent modification. Three of the eleven required blood transfusions during or after operation. The pre- and postoperative hospital stay ranged from 14 to 70 days; the patient who was in the hospital the 70 days of course was the patient mentioned earlier. She required about 50 of those days preoperatively. The hospital stay averaged 23 days. Excluding this youngster with the excessively lengthy stay, the other ten averaged 18 days in the hospital for this operation. Four of the eleven had concomitant procedures carried out, including cholecystectomy, liver biopsy and removal of the appendix.

Three of the eleven patients had some kind of early postoperative problem, including one patient with a Streptococcal wound infection that was horrendous. It occurred in the same 19-year-old female who had all the aforementioned trouble. One patient had a urinary tract Coliform infection which recurred two or three times postoperatively. Another youngster had a diarrhea problem which was not clinically difficult to treat but was recorded as significant. Late complications included two wound hernias, one in the patient we have alluded to several times earlier. One patient had to be rehospitalized twice because he simply overate to the point where he completely obstructed the outlet from the gastric pouch, requiring short term nasogastric decompression. It was no treatment problem, but was representative of the problems peculiar to youngsters with mental retardation and lack of motivation. In conclusion, our problems were not major, but when they did occur they were significant.

Now, how do you express postoperative weight loss in a group of patients who have some growth potential left? I don't really know any better than you. In any event, I have chosen to plot their weight against the normal growth standards for age and sex. I plotted each of these eleven
patients at their age at operation with their weight in kilograms, and then plotted their postoperative weights for the entire period of followup. All of these youngsters were reviewed within the last three months to bring them up to date. Our track record is spotty to say the least. For instance, one youngster was operated at 8 years of age, at a time when he weighed 50 kg which represents $2\frac{1}{2}$ times what he should weigh at this age. He lost a significant amount of weight early on and now two years later he weighs just about what he did at the time of operation. Knowing the natural weight history of Prader-Willi youngsters, we can say that the operation has indeed favorably influenced the weight we would expect to see in an 8-year-old Willi-Prader child without operation. Another example is a youngster who was operated upon at about 11 years of age at which time he weighed 53 kilograms. Two years later he still had a remarkably good weight control. This patient has an unusually cooperative and understanding set of parents, and you can appreciate that this is an important variable in the postoperative results in these youngsters. Two other patients have weight changes postoperatively which are similar to the curves expected at that age, and certainly are different from the unoperated Prader-Willi children. The girls are plotted in a similar fashion. Again, the results are not everything that you and I would want them to be, but certainly represent a respectable control of what otherwise is a severe and continuing problem.

To express our results in a different way, we have plotted the preoperative weights of these eleven patients against the median expected weight for the age at which they were operated; we have plotted their weights in terms of multiples of these expected normal means. In other words, patient JJ had a preoperative weight which was $1\frac{1}{2}$ times the expected or normal body weight; in the two years since operation he has decreased significantly in weight. Another patient was 3 times expected weight preoperatively and now five years after operation is only 2.3 times her expected weight for that age. This method of evaluating results is an attempt to crank into the evaluation the effect of age, which I think is significant. Not shown although definitely implied, is the mental retardation which makes management and postoperative evaluations extremely difficult.
Thus, we have current data on all eleven operated patients. At the present time their average weight is 1.7 times normal, and they average 9% less body weight than at the time of operation. Perhaps most significantly of all, none of them has developed a major metabolic problem postoperatively. Further, linear growth in the patients young enough for growth to occur has continued in a fashion which you would expect in patients with this syndrome; it is not normal for normal children, but is normal for the syndrome. Again, we have experienced no metabolic complications as a result of the altered physiology of the operation.

In summary, I think we can say that gastric bypass is an effective way of changing the expected gargantuan increases in weight in children with the Prader-Willi syndrome. Our postoperative results are modest, and must be interpreted with a sympathetic view toward the mental retardation which characterizes patients with this syndrome. In our experience, gastric bypass is an acceptable operation for controlling weight in these children, providing the expectations of the parents don't exceed the capability of the operation or the stringencies of the disease.
OLDER PATIENTS
Kenneth J. Printen, M.D.

The patients who are in the prime weight loss group, between the ages of 20 and 40, tend to do reasonably well and as you the see various slides shown during the course of this meeting you will see that this is the optimum weight loss period. The patients over the age of 50 definitely don't do as well. In our 52 patients over fifty we find a 4 to 1 ratio of females to males, which is pretty much the same as what we see in our total series of over 600 patients. The patients ranged between the ages of 50 and 68. We operated basically on anyone whom we felt was a candidate except for the fact that they were over the age of 50. Median weight of these patients was about 300 lbs. Preoperatively we evaluated these patients and excluded some according to the same criteria that we use to evaluate the younger age group patients. By this I mean severe hypertension and diabetes that have produced irreversible sequelae. The fat patient with Kimmelsteil-Wilson kidney disease is not going to get better because of his gastric bypass nor will the hypertensive patient who has had a stroke. Those sequelae are irreversible and losing weight isn't going to help them. So systemic diseases disqualified patients over the age of 50 as did hypothyroidism which we didn't find at all, Cushing's disease which doesn't exist in patients this fat and a variety of other medical conditions which we looked for but never found. However, the things that we couldn't really exclude in patients over the age of 50 were heart disease of varying types and of varying severity both treated and untreated or a diagnosis of carcinoma in a patient who had been treated perhaps five or six years prior to consideration for gastric bypass. These were things that did not constitute contraindications to gastric bypass in the patients over the age of 50.

At the end of five years the patients over 50 who have had a gastric bypass certainly have not lost as much weight as their counterparts in the younger age group. If you look at the six month figures you will find that the same holds true although perhaps to a lesser degree. These older individuals do not maintain as great a weight loss as their younger counterparts when you get out past the two year period. What we have found then is that individuals who are over the age of 50 for one
reason or another do not lose weight as rapidly or to the same degree as patients who are the same weight but of a much younger age.

The reasons for that are not clear but I suspect they are related to what I consider philosophical considerations, in that the morbidly obese individual who is over 50 has been morbidly obese for 45 years. I think there is no question about that. That is one of the differences between the morbidly obese patient and the patient with Cushing's disease. The patient who is morbidly obese was a fat baby, a fat teenager, a fat young adult and at age 50 is a fat mature adult, not an older patient, just a fat mature adult. The individual with Cushing's disease of course is different. He has rapid weight gain which is not nearly as marked as the patient who is morbidly obese. The people who are morbidly obese have accommodated to being so fat. Although fat housewives do just as much work as skinny housewives, it takes them a lot longer to do it. It takes them all day where it might take the thin lady two or three hours. I think in general obese patients have accommodated. They operate by the time honored principle that God only gave you so many heart beats and if you waste them doing silly things like exercise you haven't got enough left when it really counts, such as at meal times. They move slowly and I feel that restricting caloric intake, without providing some means for an outlet: such as a goal to be accomplished after the weight is lost, is doomed to failure. These people must be willing to do something once they lose weight. The younger patients do that. The older patients really have no longer term goal in many instances except that they realize they are overweight and would like to get thin. I think that is one of the reasons that the operation fails and it is one of the reasons that we don't do it in individuals over the age of 50 except for some of the specific reasons which will be mentioned below.

Another reason we don't do gastric bypasses in patients over 50 is because of their reduced longterm survival. These older patients constitute 27% of the total mortality of our group of gastric bypass patients. 27% of the early and late mortality combined comes from these patients over 50 but they only comprise 9% of our total operative experience. These people suffer from the same kinds of disease from which everybody
else over the age of 50 suffers. The only difference is that they have been morbidly obese for 35 or 40 years and consequently may have multiplied some of the risks as far as heart disease are concerned. We have had patients who we have successfully nurtured through a gastric bypass who have been discharged from the hospital only to have a myocardial infarction two months out of the hospital. Other people have gone into unremitting congestive heart failure. Now they might have done that whether they had the bypass or not. If they did it without being a bypass statistic we wouldn't have known about it but the fact remains that one of the other things that you have to think about when you do an operation on these people is "will this operation get you up to a more normal life span?" Can you achieve that with people over the age of 50 who have been morbidly obese all of their life? I don't know. I think not and that is why I don't operate upon them.

From a more pragmatic standpoint of course, you can not guarantee them any more than a 50 lb weight loss at this age, at least in our experience. For most patients that really isn't enough to get them out of trouble. Nevertheless, we do have one group of patients, a really striking standout group, in which this 50 or 60 lbs seems to make a lot of difference. These are the people who were referred to us by orthopaedic surgeons for difficulties relating to joint replacement, usually because of osteoarthritis. We currently have about nine such patients. They were all on the projected orthopaedic operating schedule after they had lost enough weight to be manageable from the orthopaedic standpoint. All of them have lost enough weight to be rendered so much less symptomatic that none of them has yet been operated for their orthopaedic problem. Some of these patients have now been followed for five or six years. I think in this group of elderly patients a 50 or 60 lb weight loss really does mean something. We were able to take that weight off and keep it off and get grandpa back on the tractor where he really wants to be, working in the springtime.

For the rest of the patients over the age of 50 you really have to sit down and do a lot of talking with the individual patient, with yourself, and look very closely into the condition of the patient overall and what he might expect from a 50 or 60 lb weight loss before deciding whether
or not you can reasonably go ahead. It is an individual thing. Just the fact that they meet all the other height-weight criteria does not automatically mean that they can be accepted into the gastric bypass program. There has to be some real reason to accept an individual over the age of 50. That is the way we operate based on our experience and, as we have gotten more patients into the series, it seems that our experience with weight loss in the older group of patients has maintained itself. I think that here at The University of Iowa we will probably continue to routinely not operate upon people over the age of 50 unless, as mentioned before, they present with special indications.
I am going to start the Symposium by asking Dr. Robert Woolson, one of our statisticians, to answer George Blackburn's question which I ignored earlier. George said that he expected this morning to hear something about the mathematical treatment of risk/benefit. Would you say something about that.

DR. WOOLSON: We looked at some of the data on gastric bypass patients. One of the real problems in looking at any sort of risk/benefit analysis is that in order to weigh the risk or benefit, it is nice to have a set of individuals who have not been subjected to the actual surgical procedure and yet fall into the same patient selection criteria, that is they are eligible for the surgery but simply we have not given them the surgery. Hopefully, this would be done in some sort of randomized fashion.

It is very difficult to interpret uncontrolled or nonrandomized data. It is much like the response of the biostatistician who was once asked how his wife was and he responded, compared to whom. It is much the same way when you are looking at any particular medical or surgical intervention. I think that any time you are looking at an effect, whether it is a risk or whether it is a benefit, there are four possible explanations for the effect. The first possibility is that there is no real effect of the treatment and what you are seeing is a chance outcome. It is here that your local statistician can help you assess whether or not this is indeed a very rare event. Another is that there may be some difference in handling the treatment group versus how you would have handled the control group. Another is that there may be some differences in the make up of treatment in the control group. For example, the individuals who are eligible for the actual surgery might be different from those who would be in the control group. Finally there may really be a treatment effect. To decide whether or not a particular procedure
is beneficial, I think the randomized control of clinical trial is really the best way.

Unfortunately, there is no control information. A number of questions have come up today regarding individuals who are obese and no intervention has actually taken place. There is no good control information available. I think it is very difficult to assess the risks and benefits. I was not able to come up with any sort of statistical model for those very reasons.

QUESTION: Preoperative psychiatric evaluation wasn't mentioned. Do you use this or have you been happy with the results of your preoperative psychiatric consultations or do you feel that you could have done just as well yourself by talking to the patient?

DR. TERRY: I obtain psychologic testing on every patient. I reserve the right to be the psychiatrist. A psychologist is actually the one who helps me on these and I don't look at them preoperatively but compile them so that I can compare serially what happens to their psychologic test pattern. I don't believe that psychiatrists like to treat obese patients. I don't believe they know anything about them or what is going to happen to them because they don't have any experience and tend to shun that experience. I have relied upon my own judgment which is accumulating. I haven't made very many errors in terms of admitting severe psychiatric misfits into the program. Our initial analysis of the psychologic testing has not shown any big deviation from the usual patient who has some concerns and anxieties about body hampering illness.

DR. SOPER: We haven't had a lot of benefit from the psychiatric evaluation in children, especially of course the Prader-Willi children who are investigated psychologically and intellectually. There are several non-Willi-Prader youngsters in our series who were referred to us by psychiatrists. Although we haven't looked specifically into this in a statistical sort of way, a few of our patients have just dramatically improved in their outlook upon themselves and upon life after changing their body image by gastric bypass. Unfortunately, I have no controlled series that would shed any valid light on that.
DR. PRINTEN: Our psychiatrists just plain aren't interested in these people. They were interested when they thought there were going to be two a month but when they found out how many there were, they rapidly lost interest. We have one psychiatrist who really feeds the program as it were. He deals in adolescent psychiatry and most of the patients I have operated upon who are 20 and under have come from this particular psychiatrist. He has documented their psychiatric changes as far as personality is concerned and their ability to get out in the world and function again once they lose weight. If you were to look at a large series of patients who were morbidly obese, and this has been done, preoperatively as far as their psychiatric problems are concerned, they are no more crazy than the rest of us. They have some personality disturbances but they do not have gross psychoses as a general rule and when they do lose weight they again don't become any more crazy than the rest of us.

DR. MASON: I do think you need to be a little leery of what you might call the autistic type or the patient that you can't talk to. There are some patients that are not only unmanageable for their eating but postoperatively they may sneak to the sink and swig a pitcher full of water or steal it from their neighbor. We have had some blowout their stomachs by doing this.

QUESTION: The concept of motivation was on one of Dr. Printen's slides. How do you determine proper motivation?

DR. PRINTEN: Mrs. Gabel, a nurse, sees all of the patients preoperatively and really visits with them about things that are important to the patient. She sees them all postoperatively too every time they come back to the clinic. Besides that she calls them on the telephone in between times and they call her when their husbands run away and all of the rest of that stuff. She is able to assess their motivation pretty adequately insofar as having a goal in mind once they lose weight. I get very leery of the patients who come to me and sit down like great big amoebas and say "I want to be skinny." You ask them why, and they don't know they just want to be skinny because they know they are too fat. Those patients are marginal candidates. I have been in the practice
of putting them at the back of the waiting list and sooner or later they get the message and they either don't come back or next time I talk to them they have got something in mind that they want to do. The ones who want to do something really have something to gain by losing weight and being able to move around. The mothers who have to chase kids around or work, the men who are students or who are being discriminated against in jobs, that is a real thing for men.

QUESTION: I would like to make a comment on what Dr. Boutros said and ask a question. He pointed out the difficulty in giving anesthesia to these patients. At our hospital we use a fiberoptic bronchoscope and they feel that this is going to take care of the problem, because you can slide it in easily without trauma and it saves time. At least in looking at it in this early stage of our experience, it seems to be the answer. The question I would like to ask is this. We have recently had a very serious complication secondary to subclavian cannulization. This patient has a thrombosed subclavian vein and right now is very swollen and edematous which gives a lot of trouble and so on. Obviously it will be two or three months before she will recover from that if she does. The question has been raised with relevance to a good risk patient, for example a 300 lb patient without pulmonary insufficiency, is it necessary or indicated to do central venous monitoring by placing a catheter in the central venous system, or can we go with a peripheral venous line?

DR. BOUTROS: What did they use to infuse through that line?

QUESTION: I think it was due to the trauma of cannulation. This is the only one that we have had, and it is a well recognized complication.

DR. BOUTROS: It does happen. When we used Swan-Ganz catheters on these patients we did a cut down. However, since the study we are more and more ambitious and we have been using subclavian sticks, in fact putting Swan-Ganz catheters through a #12 gauge Medicut. We have not had problems of that kind except in one patient in whom we were using HCl to control severe hypochloremia and we did get the subclavian thrombosis on both sides. I don't know what the answer is.
QUESTION: Do you think we need a special line in these patients?

DR. BOUTROS: I might get stomped upon by some people, but I personally do not have much faith anymore in central venous pressure per se. If I want to measure something, then I would like to measure the wedge pressure rather than central venous pressure.

DR. TERRY: I had some compulsions to use a central venous line initially when I started out with this and I guess for the last 80 patients we have not used that except if they were someone who had respiratory insufficiency. Then we may put a Swan-Ganz in and we do that in the easiest and safest way possible. Sometimes that is by direct puncture. I think that these people generally do extremely well intraoperatively by carefully monitoring their blood gases, having a suitable way to monitor their blood pressure, and having some experience on your operative and anesthesia team with managing them. You can manage them postoperatively. They have a very short period of time in which they are unstable, in fact they usually are sitting up frequently from the very time you take them off the operating table.

DR. MASON: You don't use arterial sticks on all of your patients? You just use those on the people who have had cardiorespiratory problems, or do you use it on all of them?

DR. TERRY: We tend to use it on most of them. There are some that the anesthesiologist will not be as concerned with but its a challenge for the anesthesiologist to be able to put in an arterial line in these patients and they tend to like to do that and we derive important data from it. I think it may not be entirely necessary. We do do it in most of them.

DR. MASON: Azmy, on the patient that is in pretty good shape and only twice ideal weight, would you be willing to go along with a Doppler type of blood pressure recording and skip the arterial gas analyses?

DR. BOUTROS: I suppose, to be conservative that would probably be acceptable. Putting arterial lines in fat people is, to my mind, not a
very difficult chore. If you put in an arterial line easily, then I would much rather do that. If it is going to be a hassle, I would think first of the Doppler. We need blood gases.

DR. HOSIE: Does the way an individual takes his calories preoperatively influence your decision as to whether to do a bypass or not?

DR. MASON: I guess what you are getting at is can you avoid operating on someone who is going to exist on malted milks afterwards.

DR. PRINTEN: I have one patient who has lost 4 lbs in four years. She gets up at 7:00 with her husband, who makes breakfast for both of them, then he sets her chair in front of the television set and puts a case of wafers and a jar of peanut butter next to the TV set. She sits there all day except for I am sure when she has to get up and go to the bathroom. He comes home at night and cooks supper for her. She gets up, eats supper and then they both sit in front of the television set and eat ice cream and popcorn until the news goes off or they fall asleep. If we had known before hand, that that was their eating habit, we wouldn't have operated on her.

DR. TERRY: In my experience these people uniformly fall into the category of eating a high carbohydrate diet, particularly the carbonated sodas, sometimes up to a case a day. This seems to be a habit that has been inured in these people by our television commercials and the way we live. If they don't drink sodas, they are eating something else that is high carbohydrate. I think it is a problem that we can't dismiss by saying we won't do them. I think a large number of them fall into that category to some degree and I tell them initially and then I tell them after gastric bypass that I want them to change the habits they have. First of all the soda has to go, even if they use no calorie soda. I don't want them to get in the habit again of putting something in their mouth. I am somewhat successful, probably not very successful, in changing them but I try.

DR. MASON: I think you have to make efforts along these lines but I also have the basic underlying feeling that if these people could be
talked to we wouldn't be doing the operation. I can't pick them out. There are some fantastic failures.

QUESTION: Some of these young ladies want to get married and have babies after operation. How do you manage pregnancy confinement in regard to control of the digestive process?

DR. MASON: That is another thing you cannot control.

DR. PRINTEN: We really don't tell them much of anything except that you know within the first six weeks it probably isn't a good idea.

DR. MASON: What isn't?

DR. PRINTEN: To be pregnant. Practicing is all right, but they shouldn't be pregnant. What we do do though is to warn them or counsel them that if in fact they do become pregnant after a gastric bypass, they should, by all means, let the fellow taking care of the pregnancy know about it. We have collected some of the data from approximately 13 of our patients who delivered babies after having gastric bypass and the only thing we found is that about 3 of them really had increased iron requirements during the time of their pregnancy. They were not able to be maintained at a normal level of serum iron or normal level of hemoglobin with the usual supplements that are given to pregnant women. When the supplements were doubled in one case, the girl was able to maintain an adequate hemoglobin so I think from our experience, that is really the only thing metabolically that has been influenced as far as the pregnancy is concerned. The babies have been normal just as they are with intestinal bypass. Babies born to mothers after intestinal bypass are almost always normal.

DR. MASON: I think that a woman who is in the rapid weight loss period probably is not the best candidate for pregnancy but on the other hand they seem to be able to handle it perfectly well.

QUESTION: As far as weight loss, what do you tell a patient?
DR. PRINTEN: We don't really guarantee anybody anything, of course, but what we do is sit down and talk with the patient. We have our own little book that we have for patient information. We have stated the usual weight loss is between 30 and 35 percent.

QUESTION: Is there any difference between the morbidly obese adolescent and the morbidly obese adult?

DR. SOPER: Excluding the Willi-Prader children, I see very little difference. It is a gradation from the one into the other. We now have experience with 30 patients subjected to gastric bypass under the age of 20 and the average age at operation in this group is about 16. Their treatment results correspond to the 20 to 40 age group patient after gastric bypass. In other words, they respond quite well in terms of weight loss. Our clinical reservation of course is related to what this would do to later growth. The evaluation that we have to date indicates no alterations in linear growth. This was one of our concerns. Nor did we see any hepatic or other metabolic changes that might have been influenced and to which the adolescent might be peculiarly susceptible because of his age.

QUESTION: What was the lower age limit?

DR. SOPER: Well, the youngest is a Willi-Prader who was, I think, 7 years of age at operation. The youngest normal, intelligent child was 13. The average of the Willi-Praders was 14, the average of the non-Willi-Prader adolescents was, I think, 16.

DR. BLACKBURN: I wonder if we couldn't solve this statistical problem of people who are shut off because of 50 years or older. I wonder, couldn't we randomize those groups? It looks like you have 9% of your patients with 25% of your complications. Why not randomize these patients rather than shutting them off without any good data?

DR. PRINTEN: I think that is a really good idea. It certainly makes a lot of sense because you could accumulate an adequate patient population in a short enough time.
DR. TERRY: Let me just make some brief comments here. They usually are brought in by another patient that I have done, more frequently than being referred by a physician. I sit down with them and I have what I call a fortitudinous confrontation. That is, I try to find out what it is that is bothering them most about their obesity. I need to know important facts and need to get a feeling for their motivation and other factors we have talked about. I may upset them a little bit. I try to be endearing and understanding but I really want to get down to the brass tacks. I always have a dietitian see them. They come in with a diary of what their intake was for a week and the dietitian provides me with a calorie count. If they say they have been on a diet and I know how to diet, I say, "well, let's see. Do you know how much you have been eating lately?" Then I am able to confront that issue. Usually they are eating twice what they thought they were eating even though they claimed to be good calorie counters and that is helpful in the borderline case. I am not so likely to be tempted by someone who is less than two times their normal weight for height.

I assess their previous dietary effort and that is hard to do. As we have seen, they are usually obese in early childhood. I get a profile on what their weight has been with the years and keep it in the chart, a graphic profile. I sit down and talk about the operation, describe it to them and I tell them that there is one chance in 20 that they will die and that is a hard thing to tell somebody. I tell them that there is probably a 5% risk in my series of around 100. I have a 3% mortality and I just have to say that on the average this is the safe way to explain the risk. I warn them. I tell them very graphically one chance in 20 and I say if you were thin and I was doing a similar procedure maybe it would be one chance in 100 or one chance in 1,000. That is the difference and they understand that. I tell them that I expect a functional weight loss, a third to a quarter. I disagree with Dr. Printen. I am not sure the super-large individual in the gastric bypass group will lose proportionately to his size. I have some concern that they may peter out before they have reached the same proportion of weight loss as the smaller individual.
We talk about side effects or the lack of them contrasting it to intestinal bypass. We make a contractual agreement which guarantees certain things such as followup. We do a five day preoperative workup. They are prescheduled and they go like clockwork but it takes five days and they are busy. They have pulmonary function tests and arterial blood gases. They have complete gastrointestinal x-rays. I haven't detected very much with this but I have, on one occasion, found a malrotation of the colon which gave me no end of trouble. The radiologists read it out as partial malrotation. When I got in it was complete malrotation and I had to do some make shift arrangements to my usual technique. Augmented histamine tests are done, with gastric analysis on all patients, BSP and 12/60. My gastric analysis shows some of them to be in the ulcer range, some of them not and I haven't seen any difference in the two. Lipid profile is done. I find that there are a few Type IV hyperlipidemias. I was interested to see if gastric bypass would reverse this. It has and I continue to be interested in this. It has reversed a couple of them, I guess, just related to their decreased intake. Furthermore, we do glucose tolerance, endocrine evaluation which is a urine test, cardiac evaluation, EKG and, on occasion cardiac catheterization. We do psychological testing and a complete physical examination and body composition with K40. We correct reversible problems. We ask the anesthesiologist for his kind evaluation. They are interested in seeing them aside from just the night before when we are ready to go.
OPERATIVE TECHNIQUE
Kenneth J. Printen, M.D., John F. Alden, M.D., James E. Seay, M.D., Cesar A. Gomez, M.D.

Narration of Film by Cesar Gomez

Gastric bypass surgery has proven to be an effective approach to the problem of massive obesity. In 1965, Dr. Edward Mason introduced the gastric bypass operation. The film you are about to see is a modification of Dr. Mason's operation. The modifications are as follows: 1) The use of surgical staple devices throughout the entire operation. These staples are manufactured by the U.S. Surgical Instrument Company. 2) The formation of a somewhat smaller gastric pouch without division of the stomach. 3) The formation of an antecolic as opposed to a retrocolic anastomosis. 4) The addition of a pyloroplasty to insure proper drainage to the bypassed stomach. A specially devised retractor plus surgical staples have been instrumental in shortening the operation from a four to five hour procedure to a 90 minute operation. The stapled suture line divides the small proximal pouch and the larger bypassed segment. Once the antecolic anastomosis has been completed, the pouch looks as if the esophagus has been extended.

I would like to share with you some of the statistical data that I have accumulated in the study of the first 173 patients. The age and sex distribution in 173 patients indicates that most patients fall into the 20-40 age group. Females outnumber males by 6:1. Complications have been minimal with one death secondary to massive pulmonary embolus. There have been five cases of atelectasis and no metabolic problems. Most patients vomit if they overeat. There has been one marginal ulcer and one severe pneumonia. The weight loss curve indicates that during the first three months there is rapid weight loss, gradually the curve tapers, and at 24 months the weight loss average is 122 lbs. Most of these patients were 125 to 150 lbs over their ideal weight preoperatively. They have come to ideal weight and have remained at that level for over 24 months. One patient came down to slightly below her ideal weight. However, she is quite healthy and quite happy. Her mother is also a patient and is one of the few in the older age group who has done extremely well as far as reaching her ideal weight. Another patient had
severe knee joint problems and after about 130 lbs weight loss her symptoms have definitely improved.

The incision is usually a midline incision, most of the time extending from the xiphoid process to the umbilicus. In regard to the particular patient in the film the distance was too short, and I did not hesitate to extend it to the right and below the umbilicus. The electrocautery is used freely, here dividing the linea alba in the midline. The thick pad of fat is removed by dividing the avascular planes on both sides of the incision, leaving the peritoneum quite free and bare, which facilitates closure at the conclusion of surgery. The falciform ligament of the liver is properly clamped and divided and ligated. The excess pad of fat is removed. The specially designed retractor is now inserted, four bars are fixed to the operating table to which four additional bars are attached. This forms a rectangle which is quite sturdy and to which two Deaver type retractors are inserted to elevate both subcostal areas. A Hyerson-type retractor is then applied at the midline to elevate the sternum anywhere from 4 to 6 inches, leaving excellent room for proper exposure and proper division of the triangular ligament of the left lobe of the liver, allowing the left lobe to be retracted to the right which will be secured by another self-retaining retractor.

The pyloric area is held between the index finger and thumb of the left hand of the surgeon. An incision is made longitudinally, transecting the pyloric muscle. This incision is closed transversely after stay sutures are inserted, facilitating the application of the TA-55 staple gun. The TA-55 is now applied, properly calibrated, the staples are advanced, and the excess tissue is excised. Before the TA-55 is removed the small bleeding points of the cut surfaces are coagulated with electrocautery.

The splenic flexure of the colon is properly mobilized in order to place the anastomosis in the trough between the spleen and the colon at the conclusion of surgery. The lesser peritoneal sac is now entered and the gastrocolic vessels are properly clamped, divided, and ligated using the LDS staple gun. This instrument facilitates ligation and division of these vessels and also it expedites the procedure. The mobilization of
the greater curvature starts at the pars media and is carried all the way to the esophagus including the vasa brevia to the spleen. The fundus of the stomach is now properly mobilized and peripherally free and will accept the TA-90 instrument without any difficulty. Now the site of the pouch is outlined. The right hand of the surgeon is inserted behind the fundus and by blunt dissection the esophagogastric junction is exposed. With electrocautery the avascular planes are divided and one can see the operator's finger coming through the opening. The TA-90 instrument is then inserted through this opening and after it has been properly calibrated the staples are advanced, outlining the small proximal pouch and the distal bypassed segment. The pouch measures approximately 10 cm in length by 3-4 cm in the greatest and smallest diameters.

The ligament of Treitz is identified and with electrocautery is divided as far as the fourth part of the duodenum. This allows for a short loop of jejunum for antecolic anastomosis. The inferior mesenteric vein can be identified. One stay suture is inserted at the antimesenteric segment of the short loop of jejunum. A very small opening is made with the knife. The opening is enlarged with a hemostat and one blade of the GIA instrument is inserted. A small opening is then made at the gastric pouch and the other blade of the GIA instrument inserted, this one containing the cartridge with the staples. The two blades are approximated in such a manner as to fashion the anastomosis at the antimesenteric segment of the jejunum and at the greater curvature of the gastric pouch. The instrument has been properly calibrated at the 2 cm level. The staples are advanced and the instrument is removed. This incision will be closed transversely using the TA-30 instrument. Two seromuscular sutures of 4-0 Polydek material are inserted at the apex of the suture line to prevent tension at the staple line. Stay sutures are now inserted at each corner of the end of the suture line from the GIA instrument. This is done to facilitate the insertion of the TA-30 instrument. The pouch now is irrigated with saline solution. This pouch syringe contains approximately 100 cc of saline which is just about the maximum amount of saline which the pouch can tolerate. The last stay suture being inserted in preparation for the TA-30 instrument.
At this point the nasogastric tube is advanced through the anastomosis. A #18 French Salem tube is used. The TA-30 instrument now is inserted, placing tension on the stay sutures in order to apply the instrument against the #18 French nasogastric tube which then allows for a small 1.2-1.5 cm in diameter anastomosis. The staples are advanced and the excess tissue is excised. Before the instrument is removed the small bleeding points are coagulated with electrocautery. The nasogastric tube now is pulled back to the gastric pouch and secured in place approximately 3-4 cm proximal to the anastomosis. The pouch lies with a smooth angle between the afferent and efferent loops of jejunum. The splenic flexure of the colon now is pulled to the right, placing the anastomosis in the trough between the hilum of the spleen and the splenic flexure of the colon. The left lobe of the liver now is placed in its proper anatomic position and the appendix is removed as it is usually done.

The abdomen is closed with #1 Prolene figure-of-8 sutures. No subcutaneous sutures are inserted except for several interrupted subcutaneous stay sutures using #1 Prolene. The skin is closed with interrupted stainless steel staples. During the past four years my experience with this operation has been most gratifying. Most patients have achieved their desired goal while maintaining excellent health.

DR. ALDEN: In order for this operation to be successful it has to fulfill several criteria. 1) It has to be safe, 2) it has to be simple, and 3) you must have success if the operation is going to bring patients back into energetic good health. This is where it differs considerably from the intestinal bypass. Those patients lose weight but they never really find the energy, good health and the good life that is enjoyed after the gastric bypass. In searching for a simpler way to do the gastric bypass I also came across the stapling instruments. I would say that without them there is no way that I would even consider doing a gastric bypass. It is much too difficult and time-consuming. With the stapling instrument, our average operating time runs at about 68 minutes for a gastric bypass and when I used to do the intestinal bypass it was about a 60 minute skin-to-skin operation. These figures I don't give as impressive for time but simply to indicate that the operation is simple and by using the advantages of mechanical devices one can do the operation within a reasonable amount of time and not have it be too exhausting.
for the surgeon. Obviously it is going to be safer for the patient with less anesthetic time.

All of our operations have been done through a midline incision extending from the xiphoid to the umbilicus and never below the umbilicus and never splitting the sternum. We haven’t used any special drapes and we have only used the standard operating table, never widening it because of the obesity of the patient. I would be very opposed to doing that because it is so difficult on your back. I had one opportunity to come into an operating room to help a new resident. He put my patient on two tables and it was just terribly difficult to reach that far and to try to operate over a second table. That is absolutely wrong and I think one should avoid any of those unnecessary troubles. I like to keep the table at about a 10 degree elevation for the head of the table. I think this facilitates the breathing of the patient and it also lets the viscera slide down so you get a little better exposure in the upper abdomen.

After the patient is intubated with the endotracheal tube I ask the anesthesiologist to insert immediately the nasogastric tube. The first step I do, then, is to encircle the esophagus and place a Penrose drain there as a guide for the rest of the operation. The Penrose drain is what keeps me honest in this operation. When I get to this part of the stomach, if I do not use this drain as a marker I am prone to think this will be a small pouch. People would agree with me, but I would have a pouch that was probably too big. With the stomach freed entirely up to the esophagus so that the Penrose drain will slide all the way down, I know that I have freed the stomach up adequately so that there is no question in my mind that I will have a small pouch.

At that point I usually stop this part of the operation and go on to whatever ancillary procedure might be needed. During the time I am doing something else, such as tying the tubes, removing the gallbladder or biopsying the liver, what little bleeding that has been caused by the blunt dissection will subside and I can come back to this at a time when it is a little bit dryer. Incidentally, if you like to tie the tubes on
these patients, the LDS is a marvelous instrument to reach down and snap off the tubes.

I free the greater curvature in its proximal third, and I have been using Weck clips for this. I like to skeletonize these vessels and keep them very close to the serosal surface of the stomach. For the lower vessels the small Weck clip is fine. However, when you get up to the splenic area, I think you should have the large Weck clips. They are about a half inch long, and grasp a little more tissue. This is important because these vessels are quite elusive and they can't be skeletonized well.

I make a window alongside the lesser curvature, and this window is made right on the serosal surface of the stomach. It is greatly facilitated by placing my hand around the stomach with my forefinger and thumb rubbing the tissue until I have just the gastric wall. Then I simply strip the serosa off the stomach here and don't take any vessels down at all. I make a window, put a Carmalt through it and spread it, pick up a Penrose drain and then put my index finger through it.

The next step is to bring up the jejunal loop anticolically and tack it onto the proposed site of the gastric pouch. I tack the jejunum high up on the stomach with three stitches of silk and then I perforate the stomach wall anteriorly and laterally and the jejunum with an electrocautery, spread the opening with a curved hemostat and insert my index finger as a guide to the size of the anastomosis. Next I fire the GIA which I insert to 2 cm. When the GIA is removed it is very wise to open the anastomosis and take a look at the back of it because there occasionally will be a little artery that bleeds and that is best controlled not with cautery but with stick-ties of silk. I used to close the anastomosis with the TA-30, but I think I am going a little higher than I was before and I feel I can make a better closure of that anastomosis by using silk. Therefore, I complete the perimeter of the anastomosis with interrupted 3-0 silk Lembert sutures and at that time roll in a fair amount of stomach and jejunum, trying to make that anastomosis just a little bit smaller in diameter all the time.
Before I staple across the stomach, I like to get the nasogastric tube down in the efferent limb of the jejunum, and one of the reasons for that is I am afraid that going as high as we do, I might someday staple off the esophagus and trim it off a little too closely. Therefore, I like to have that nasal tube in as a guide so I know where I am. I try to work the stapling instrument upward and pull the stomach downward. I use Babcock forceps a great deal for this. There are two or three vessels at this point which are easy to avoid and it is essential that this is absolutely freed by getting these vessels loose. This allows you to bring the stomach down a great deal more and you will get a pouch which I am sure never measures 100 cc. We don't measure them, it has just been too difficult and I don't believe it is terribly essential anymore. The staples that I use here are the 4.8 mm staples. They are the heavier of the two staples that are available. I insert that row of staples, remove the stapling device, and then tack the stomach and the jejunum together with some interrupted 3-0 silks because I am afraid of a twist or a kink occurring in the efferent limb. With this, then, the operation is completed. I close my patients with wire using 1-0 monofilament wire interrupted sutures. I don't close the fat; and close the skin with staples. So far the operation has been safe because we haven't had a death in 249 patients. We have had one evisceration; we have had no leaks. The operating time is small and our patients average about 36% weight loss at the end of a year.

DR. SEAY: Following Dr. Alden and Dr. Gomez reminds me of a cartoon I once saw of a couple of jugglers getting ready to go on the burlesque stage. Coming off the stage was a very attractive young lady in a pajama top and a man in a bottom pushing an iron bed. One juggler turned to the other and said "Talk about hard acts to follow!" I feel a little bit in that position, but at the risk of boring you we will run through it one more time, and then maybe we'll get the technique down.

There are a number of ways of doing this procedure and the technique that I want to describe today is one that we have evolved in the process of doing 278 operations. These were done primarily in smaller hospitals where help is often a problem and because of this we have tried to devise a technique that two people can do. Our group of three surgeons
employs a full-time nurse practitioner who acts as an assistant on the procedures, but even with good help, exposure can be a problem.

I would like to tell you about the country cousin of Dr. Gomez's retractor. This is the upper hand retractor with which a number of you are familiar. We use one that is equipped with a double blade on top. I think the important thing about this retractor is that it has to be placed high enough. The arms have to be up. We got a little static from the anesthesiologists early in the game, but we have not had any shoulder problems or arm problems. I use an upper midline incision extending from the xiphoid to the umbilicus. When we get in, we do an abdominal examination. If there are gallstones present, I usually do the cholecystectomy first. We place a retractor with a curved Balfour blade under the left costal margin. If the liver is not too fat the flat blade works fine. However, if it has extensive fatty infiltration, we find sometimes we crack the liver and I think it works better to hand-hold it. This is where it helps to have a left-handed assistant if you are right-handed and we are fortunate enough to have one. We use a large Balfour on the retractor also.

Next the fundus of the stomach is completely taken down, as described by the other people. We use the LDS lower down, but we have had some slippage higher up. I feel much more secure using hemoclips high up on the short gastric vessels. I do not usually clamp the stomach side because there always seems to be a clip where I want to put my anastomosis and we usually control that with electrocautery. We don't dissect too far down on the greater curvature of the stomach, I am concerned about devascularizing it, and usually we get good enough exposure by just dividing the short gastric vessels. I think most surgeons agree now that in order to get a small pouch you have to completely take the fundus down and make a tube of stomach with the greater curvature being one side of the tube. Occasionally we see small vessels enter the stomach posteriorly. I don't think it adds anything to take those. We leave those intact, again to get a little more blood supply to our pouch.
Early in our experience we had an 8% incidence of splenectomy, but we're getting a little better at doing the operation and also with the recent work in the literature about saving the spleen we have used more Surgicel and we use Avotene if we get splenic tears. In the last 100 patients we have only had to remove three spleens. We have not had any postoperative splenic bleeding, even though we anticoagulate all our patients.

After the greater curvature is freed we go to the left of the esophagus and clear off the vessels. Then I put two fingers up behind the stomach, where Dr. Alden puts his Penrose drain, in order to make sure this is open and that the opening is large enough to pass the TA-90 stapling device. We don't disturb any vessels that might be coming down the left side of the esophagus. Since I usually do the anastomosis retrocolically, I make a hole in the transverse mesocolon and bring up a short loop of jejunum. The fundus of the stomach is then retracted caudally with a Babcock, straightening out the angle of His, and stay sutures are placed to set up the anastomosis in an isoperistaltic fashion. I usually try to place the top stay suture about 2 cm from the esophagogastric junction.

I believe good results are basically dependent on the small pouch and over the years our pouches have gotten progressively smaller. I would estimate that we leave about 5% of the stomach functioning. We have not had any cases of stomal ulceration, and I suspect this is related to the small pouch.

Stab wounds are then made in the stomach and in the jejunum. I have not concerned myself too much with the small stomas, I may be changing my mind after this meeting. We insert the GIA to about 3-3½ cm mark and then fire it. After you do that it is imperative that you check the anterior and posterior lines, primarily the posterior line. I would estimate in almost 50% of our cases we have had some trouble from bleeding here and it is best controlled with an interrupted silk suture. After this the opening is grasped with Allis forceps and the TA-30 is fired to complete the anastomosis. I generally put a couple of Lembert sutures across the TA-30 line because I pull the anastomosis beneath the mesocolon and I am concerned about whether or not I might distract it and pull the staples apart. This hasn't happened yet.
After that the two fingers are again passed behind the stomach and I insert the TA-90 from the lesser curvature side down to just past the anastomosis. We use the larger of the cartridges, the 4.8, because I would like to get a little more blood supply across the staple line if I can. Some people make an effort to leave vessels above the TA-90 staple line, between that and the esophagus on the lesser curvature. We have not found that necessary. After about the first four or five cases I quit dividing the stomach and we have not had any sloughs at all since we began doing this. We don't routinely calibrate the pouch. In this regard I agree with Dr. Gomez and doubt that I could get it much smaller.

After that the anastomosis is sewn beneath the mesocolon. Next we irrigate the abdomen with sterile saline solution and leave a suction drain in the peritoneal cavity. We have been very happy with the Jackson-Pratt drain, which is a soft silastic drain. We have had three deaths early from peritonitis, and I think it would have been possible probably to save these people if we could have made the diagnosis early enough before they arrested. Therefore, we now routinely leave a suction drain in the abdomen. We close the peritoneum with running chromic catgut. The fascial closure is set up with interrupted Neurilon sutures and we use the new wide fascia staples in between the sutures. This is the FM-20-W, which is the new cartridge that has just come out. We leave a Penrose drain in the subcutaneous fat and we close the skin with wide skin staples. As with the other men, a number of patients who are only about 100 lbs overweight can be done in about 55 minutes to an hour. Our average operating time is about 1 hour and 15 minutes.

QUESTION: What about the blood supply around the staples?

DR. GOMEZ: Do you mean the blood supply to the pouch? Well, since this movie was made, I have had two pouch perforations, both proximal to the suture line in the anterior wall. Fortunately, we were able to make the diagnosis and reoperated the two patients without mortality. One of the two patients, certainly was drinking excessive amounts of water, which we found out later. Whether this distention on a perhaps borderline blood supply pouch may have induced this I don't know. The other patient had the same thing and there was no explanation for it because the rest
of the pouch was perfectly normal with a perfectly good blood supply. As a matter of fact, I just closed the defect and it was fine.

QUESTION: Are there blood vessels coming in above the staple line on the lesser curvature?

DR. GOMEZ: There are small blood vessels on the back and I believe it is important not to disturb those. I think there are branches of the left gastric vessels that go up and if you stay very close to the stomach on the lesser curvature and make a very small opening and do not disturb anything else I think you can leave those intact. In 300 cases now I think that I feel pretty secure that the blood supply to the pouch should be quite adequate.

DR. ALDEN: I don't think there's any problem with the blood supply to the small pouch as long as you don't divide any vessels on the lesser curvature. You can go between the vessels. I usually try to leave one vessel just above where I insert the second Penrose drain to receive the TA-90. Therefore, I have had no problem with blood supply.

DR. SEAY: One thing I didn't mention is that in patients who have complicated peptic ulcer problems I do a resection of the distal stomach. I think if this happens it is imperative that you leave vessels into your pouch area. Otherwise you will get into trouble. But if you don't divide the stomach, we have not had a single problem to our knowledge.

QUESTION: What you are saying, then, is the blood supply comes through the staple line.

DR. SEAY: I think some of it does, but I can't prove it. That is a hunch, and then posteriorly. We've been concerned about the incidence of postoperative leak, and we've tried to identify this problem. As you all know, the incidence in gastric bypass has been higher than what you would expect from a routine gastrectomy and a gastrojejunostomy. What happens is, if you divide the stomach way up on the lesser curvature, then, having divided the short gastrics, you are leaving nothing but the ascending esophageal artery which is the first branch of the left gastric
artery. It is imperative to leave the first two or three branches that come off the left gastric artery in order to insure viability of the gastric pouch. There may be, in very rare instances, a couple of arteries coming from the inferior phrenic artery which supplies the proximal stomach. However, having divided the left gastric artery here, coupled with the Hofmeister type closure on this part of the gastric pouch, the edema that is resultant from the suture line plus the edema at this suture line compounds the issue and the blood supply in the anastomotic area is marginal. I am not suggesting that all leaks that occur do occur when the gastric pouch is necrotic and obviously grossly sloughed. The microscopic blood supply to the anastomotic line is critical and perhaps also important in the formation of strictures on a long term basis.

DR. PRINTEN: I think that's why most of the folks who have talked today really don't make any great effort to ligate everything and come through right next to the esophagus as a general rule.

DR. PORTER: I just wondered if anybody had noted any problems from bleeding from the TA-90 staple line.

DR. GOMEZ: Interestingly, yes, the bleeding is at the moment, and after I irrigate the pouch with cold saline then it stops. It is true, I think the staples cut through a few blood vessels in between and bleed inside, and probably they bleed some in the bypassed segment as well, but I haven't had any postoperative hemorrhage, no.

DR. ALDEN: I have never seen the inside of that staple line because I always complete the anastomosis before I make the staple line, but I have had no hemorrhages so I guess it's not a serious problem.

QUESTION: Dr. Gomez, how did you diagnose those two patients with leaks?

DR. GOMEZ: Well, both we thought were pulmonary emboli, as Dr. Mason has emphasized. I go with the idea that they have something catastrophic
inside until I have proven otherwise. I did peritoneal tapping and sure enough I could see the bile.

COMMENT: For the gentleman that asked about the stapler, if you have had more than one case of bleeding, I would suggest to inspect your stapler because they do become defective, they spring, and the staples don't close properly.

DR. PRINTEN: Another thing is, from a technical standpoint, every staple gun that is made is made with all those parts that work only for that staple gun, so if you have convinced somebody to buy you two or three, you've got to make sure that when they sterilize them and put them back together again they put all the pieces from staple gun one with staple gun one, and all the pieces from staple gun two with staple gun two. Otherwise, they sometimes don't even work.

QUESTION: I notice that Dr. Gomez does a pyloroplasty. Is that necessary? I just wondered also if anybody has followed these with postoperative and preoperative gastric acid studies to see what is really going on?

DR. ALDEN: No, I have never done a pyloroplasty and don't think I'll start doing them now. I don't really think I need them. However, about the gastrin levels, we have one man that developed an ulcer in his pouch, not at the suture line but well into the pouch, and bled quite a bit from it about six or eight months after surgery. His serum gastrin was 30 picograms, which is in the very low range. It was a 10% pouch in which the ulcer developed.

DR. PRINTEN: We have measured gastrins in pre- and postoperative patients and the gastrins are lower in the postoperative patients. In addition, we have put gastrostomies into the distal pouch when we used to divide the stomach completely (I put gastrostomies in there because I was nervous) and then afterwards in the postoperative period we did Hollander tests on the patients and used the collection of the material from the gastrostomy tube. That bypassed stomach reacts to food put into the upper pouch as well as to insulin the same way it did when it was a whole stomach. You don't do a vagotomy on that distal pouch at all.
DR. GOMEZ: In the beginning when I was dividing the stomach I wasn't doing pyloroplasty. Some patients had similar symptoms that I could not explain. It was not vomiting, but it was a peculiar feeling of fullness. They used to tell me, "Doctor, when I eat, you know, my belly gets bigger." I regarded that as probably nonsense, until this patient developed a hernia. When I opened the patient this bypassed segment was like a football filled with inspissated mucous material, and the patient had told me that she used to vomit large amounts of this thick material which wasn't food and it wasn't bile. I cured that patient with a pyloroplasty and I thought maybe I've been doing some vagotomies when I divided the stomach, so I started to do pyloroplasty on everyone. Now that I do not divide the stomach, I wonder whether I need it, but the patients have done so well I am going to stick with it.

QUESTION: Do any of you do a panniculectomy in conjunction with gastric bypass?

DR. SEAY: No.

DR. ALDEN: No.

DR. GOMEZ: No. I've done only one panniculectomy.

DR. PRINTEN: It's hard to do a panniculectomy through an up-and-down incision like that.
LEAKS AND DEATHS
Edward E. Mason, M.D.

We have had a 3% operative or early mortality, and we have had about a 3% incidence of leaks over the years. I think the improvement that we have made recently is that we can recognize the leaks now and do something about it. Consequently, the patients don't die even though we still have the problem of leaks in some patients. I only have one slide for this talk which portrays a maximum postoperative survivorship curve against which the deaths have been plotted. The first patient that was operated upon in 1966 would now have had an opportunity to survive several thousand days and the last patient would only have had an opportunity to survive a few days. There is quite a cluster of nonoperative deaths in the early experience. Most of these patients were operated upon by me. Three of them died of cancer, one from cancer of the lung, one with cancer of the larynx and one with cancer of the uterus. Most of the rest of them died of chronic heart failure, progressive angina, and so on. None of these deaths are related to the gastric bypass. There are a couple of deaths that occurred after revisions. The risk of revising one of these patients is greater than the risk of the first operation, and I think we need to keep that in mind. Pulmonary embolism was a concern, and we have had a few. One of our first patients died of a pulmonary embolus about the 15th day. This was the patient I mentioned this morning who died 24 hours after her operation. She was up walking around in the room and keeled over with a massive embolus. We have had some sudden unexplained deaths that I mentioned this morning. One of these patients was about 100 days postoperative. She had been losing weight at a fairly good rate, perhaps an excessive rate, and we understand that she was at an amusement park riding on a roller coaster and died shortly thereafter. As I mentioned this morning, sudden death is a problem of obesity. These people are still not normal weight and perhaps during the period when they are losing weight they still have the risk.

So that leaves us with the problem of intraperitoneal infection or, in other words, leaks. We really haven't improved on this very much. In our first 100 patients the 60 day mortality was 4%. In the second 100 it dropped to 1%. This is probably due to the fact that during that
period we were experimenting with gastroplasty which was an easier operation. This was an attempt to simplify the procedure. It is extremely important to find a simple way to do this surgery. Gastroplasty was such an attempt. It had a low operative mortality. It consisted of cutting across the stomach but leaving a channel along the greater curvature. There was no anastomosis. It was great, but they didn't lose weight very well. So after 1971 and 56 of those patients, we went back to the gastric bypass and our mortality went back up to 4%. Recently, our mortality had dropped to less than 1%. I hope that is true, but I think you have to take the attitude that complications can occur. If anything can happen it will, and you have got to look for trouble. Leaks are tolerable, death is not.

When somebody dies of a leak, there are two mistakes. One is a technical mistake, and the other is a mistake in patient care, that is, failure to recognize the leak. In regard to technical problems, I think one of the main cause for leaks is making a pouch that is too big. If the upper pouch is too big, it has a great diameter. One of the things that was stressed by Dr. Wangensteen in his bowel obstruction book was the formula of surface tension equal to \( P \pi D \). The surface tension is the disruptive force on the surface of the stomach, and that is related to the pressure inside the stomach. You can't change that too much. But the other factor is the diameter. If you double the diameter of the pouch, the same pressure will give you twice the disruptive force. So if you have a big pouch it is more likely to rip, and blow open. Of course, there is a bit of a problem if you make a small pouch up above, which you need to do to get the weight loss that is required, then you've got a bigger pouch distally which can also blow out. In short, you've got two pouches here that can blow out. You have two potential closed segments. You've got a staple line that closes both of them, and a cardia which, if competent, can close the upper pouch. The lower pouch can be closed by a kink in the gastroenterostomy. This operation was designed to simulate a Billroth II gastric resection; an operation in which blowout of the duodenal stump defeated many good surgeons. I remember Dr. Wangensteen worrying about the duodenal stump and trying different ways of closing it so it wouldn't blow out. I suspect that when those distal segments blow out it's not so much the way the stump was closed but
rather, it has something to do with the kinking of that gastroenterostomy in the afferent loop, or sometimes even the efferent loop.

We had one patient who had a bowel obstruction from an old tubo-ovarian abscess and adhesions. Postoperatively her bowel obstruction produced distention, markedly so in the biggest segment. It is just like the cecum in a colon obstruction. You must not leave any mechanism for distal small bowel obstruction. You must examine the abdomen and run the bowel and make sure there are no potentially obstructing areas. The simplicity of the operation is extremely important in avoiding kink of the gastroenterostomy. I remember coming on one of these patients in the recovery room the day after the operation. The team had been around, the patient had been seen. I don't know what went through their minds, but when I saw the patient I was concerned about some things I will speak of later with regard to recognition, but particularly a very high pulse rate. At any rate, the patient was taken back to the operating room and we found that the loop of small bowel had been brought up behind the colon but then it had been brought between the omentum and the greater curvature of the distal segment so that the anastomosis was under considerable pressure and had undergone necrosis. There was ischemic necrosis of the gastroenterostomy. There was a gastrostomy made with a Foley catheter in the distal segment which had been put in from the right side of the abdomen and had been intended, I am sure, to rest pointing toward the esophagus. Unfortunately, it had turned on itself so that the bag on the Foley catheter was obturating the pyloric ring. That meant that the opening in the catheter was in the duodenum and we had a closed loop produced by the very gastrostomy tube that was intended to keep that distal segment empty. At other times there has been failure of getting the mesocolon up on the stomach above the gastroenterostomy. An old problem as those of you who have done Billroth II's know, is to have the opening in the mesocolon slide down and pull those two limbs together and kink one or the other or both of them. About half of our leaks have been in the upper pouch, about half of them have been in the lower pouch, and a few of them have been in both pouches.

The majority of time when a leak occurs the first diagnosis that is made is pulmonary embolism or some cardiopulmonary catastrophe, and it just
isn't the case. These people develop tachycardia. They become short of breath and they have to sit up or get up and walk because they are in distress. Our gastric bypass nurse, made a very astute observation and described something that Hippocrates described. She says you can tell by the way they look. They look anxious. They have a hypocratic facies. Now, we are guilty of wishful thinking, you know. We don't see things, especially if we are the responsible person. If you have done the operation, you always sort of wish it were not so, and you don't even see things that actually are so. Another very important thing is the pulse rate. The mean + 1 standard deviation on the first postoperative day is 120/min. If you see a pulse rate above 120/min, you better start thinking about a leak. You can't make a diagnosis on one piece of information, but that is a good piece of information nevertheless.

After people stop thinking about cardiopulmonary complications, the next thing they do, which isn't necessarily a mistake but does cause delay, is order a hypaque swallow. Unfortunately, a hypaque swallow doesn't show the leak in many instances. It won't ever show a leak in the distal segment, because there is no way the hypaque can get to the distal segment, so 50% of the time you are not going to learn anything from the hypaque. Sometimes it won't show anything with a leak in the upper segment either. I haven't used peritoneal lavage. Perhaps this would be a much better technique than a hypaque swallow, in some instances. I am intrigued with the idea that was presented of doing peritoneal lavage. I am not quite sure that I want to leave a drain in the abdomen for the specific purpose of picking up a leak, although this is an interesting idea.

DR. PRINTEN: The one additional comment that might be made is that we feel so strongly about the business of unexplained tachycardia that we have reoperated patients on the basis of nothing but unexplained tachycardia that was unacceptable to us, and we have never yet been wrong. We didn't always find leaks, but we found a closed loop bowel obstruction once and we found a large subfascial abscess. We have always found something when we used the criteria of unexplainable tachycardia as a criteria for reoperation on these patients.
Pulmonary embolism is certainly prevalent. It may lead to immediate death, for which we have no answer, although there is current interest in attempting to prevent such deaths by prophylactic measures. In the vast majority a diagnosis is not made. Many of those patients will survive but there are more deaths from undiagnosed pulmonary emboli than those that have been diagnosed, so we have errors of omission as well as commission. These statistics have led to a variety of recommendations for prophylactic measures. As we have become more and more concerned about the risk of fatal pulmonary emboli a variety of measures to combat it, both mechanical and medical, have become available. Currently perhaps the most popular method is the use of low-dose heparin. You can also use other agents of your choice, but the question we have to fall back on is, "How often are we attempting to prevent something that may not indeed be occurring?" This is really the subject of today.

We are involved with a patient population that has many of the risk factors that are traditionally felt to be responsible for deep vein thrombosis or pulmonary embolism. We are dealing with obesity and patients who require some bed rest. We are dealing with injury which, in them, may be major. At the very least the operative factor must be considered. Many of these patients may also have preexisting venous disease. I think we have to remember that the traditional signs of deep vein thrombosis, which are the basis for most of the clinical reports about the incidence of venous thrombolic disease, are quite fallible. There are any number of objective studies, in which routine venograms have been done on patients who have either classic or suspected signs of deep vein thrombosis. These studies show that your clinical diagnosis is correct no greater than half the time and very often you would be better off flipping a coin. This fact has prompted us here, and I think more and more centers elsewhere are being prompted, to look at objective screening techniques. If one does not have screening techniques that are accurate, one still has a diagnostic standard, phlebography or venography, that one should consider. We have had a number of techniques available to us in the past five years. We have primarily relied upon
Doppler ultrasound, although plethysmography is being used in a number of centers. It is surprising how easy Doppler ultrasonic examination is in these patients. I will say something later about the fibrinogen uptake test only to indicate its fallibility, particularly in the obese patient, but more importantly its extreme sensitivity, which I think is responsible for a lot of the scare about the incidence of venous thrombosis following operation. Hematologic studies are not germane here, although they may pick up patients who have blood coagulant or platelet abnormalities predisposing them to thrombosis.

We use the pocket Doppler. This has a 5 megahertz transmission frequency which is mandatory for the obese patient. We have relied upon technician evaluation using the audio output of the instrument. All one is doing is listening to what also can be recorded. However, it is just as accurate to have the technician listen to the cyclic venous velocity wave forms, which are simply the velocity shifts with waxing and waning venous flow that are picked up by the instrument. The basic principle in these patients is to assess their common femoral venous velocity. Normally it should be cyclic and wax and wane with respiration. Although one may have to have the patient or some other scout lift up the panniculus, it is surprising how easily one can hear the common femoral artery and the adjacent venous signal with this particular instrument. One should expect a normal augmentation at this level, or at the knee if one is listening behind the knee, when one squeezes the calf. In the study I am about to report we did not assess the pedal venous signals.

In the presence of deep vein thrombosis there may be a more continuous flow or one may not hear any flow at all in the common femoral vein. Furthermore, diminished augmentation can be expected upon squeezing distal to where one is listening with the probe. With experience this technique can give you an accuracy that is as good as any other technique, plethysmograph or otherwise. Most of the errors are in isolated calf venous disease, although with experience even the majority of major calf vein thrombi can be readily detected. One should, again with experience, exceed 90% accuracy when compared to phlebography. The importance of using noninvasive objective techniques became evident in our first two year experience, in patients with a clinical diagnosis of deep vein
thrombosis, in which we found the diagnosis confirmed by Doppler in only about one-third of the patients. This is very similar to the statistics of Haeger from Sweden. Patients with pulmonary emboli have an even lesser incidence of source of clot identifiable in the leg. The important thing to realize is that pain or swelling or inflammatory conditions may be due to a variety of other factors - there are many more tissues in the leg than just veins.

On the basis of this experience, we elected to prospectively study a consecutive group of patients, who consented to screening of the lower extremities both before and after gastric bypass. These were patients that were not placed on prophylactic anticoagulants. The routine at that time was to use elastic leg bandages since commercially available antiembolism stockings did not fit these patients. Of course, the other mechanical factor that has been routine in these patients is early postoperative ambulation. We performed a screening objective Doppler exam before operation and on alternate days or every third day throughout the postoperative period until the patient was discharged. We used the portable Doppler unit so that the technician could examine the patient at the bedside, and we primarily assessed the common femoral venous flow before and after compression of the calf. We studied 57 consecutive patients who underwent gastric bypass. It is of interest, if one studies patients for any major elective surgical program, that there is a surprising incidence of preoperative venous abnormality. Four of these patients had varicose veins, four had a history of prior leg vein thrombosis, three had leg edema, one had obvious stasis disease, but only one of the patients had an abnormal Doppler exam suggesting prior deep venous obstruction. Overall there was about a 14% incidence of venous disease prior to operation. There have been some recent studies that suggest that if one is attempting to define the postoperative incidence of venous disease, a preoperative screen should be made in order to ensure that one is not dealing with prior disease.

Postoperatively, patients were helped out of bed the evening of operation and progressive ambulation was instituted during the ensuing days. The mean hospital stay for the patients was 10 days. Clinical evidence of leg vein thrombosis occurred in four of these 57 patients, but this was
confirmed in only one patient who had an abnormal Doppler exam. The three other patients with leg complaints had an entirely normal Doppler examination. One patient had leg edema, and again the deep veins were patent in that patient. Two patients gave a history that might have been interpreted as pulmonary embolus, but only one patient had a proven pulmonary embolus. That patient subsequently died but not of the pulmonary embolus. It was noted at autopsy and involved less than a lobe. This was the same patient who had the abnormal Doppler examination.

This low incidence of venous thromboembolism (< 2%) is at variance with what one reads repeatedly in the literature in a variety of surgical series. Most of what you are reading about in the literature are groups of patients who have been prospectively surveyed with the radioactive fibrinogen uptake test. That is an extremely sensitive test and it may pick up thrombosis that may not even be detectable by contrast venogram. It is important to realize that not all of those patients are at risk of significant thromboemboli. In fact, probably 80% of patients with positive fibrinogen uptake in the leg have spontaneous resolution of their problem. In the vast majority it remains confined to small muscle veins in the calf. When we use Doppler or plethysmograph techniques that are sensitive to more major leg venous disease, we find that the incidence of leg vein thrombosis in supposedly high risk groups of patients is actually relatively low.

The first prospective study we did using the Doppler, which antedated the study I have just reported, involved total hip replacement in patients who were not receiving prophylactic anticoagulants. Two of the four patients with postoperative evidence by Doppler of thrombosis developed it after they had been discharged from the hospital. Only one patient developed a pulmonary embolus and no source for that embolus was ever found by Doppler or venogram. In another study we have reported, patients undergoing major lower leg extremity amputation again had a low incidence of leg vein thrombosis. In a study initiated by a nurse to assess the efficacy of antiembolism stockings, in general surgical patients there were no major leg vein thromboses in the first 63 patients studied. If one is attempting to assess such prophylaxis as stockings, one would have to have a very, very large series of patients. Segal in Philadelphia
similarly reported a low incidence of deep vein thrombosis (< 4%) following operation in patients who did not have prior preoperative venous abnormalities. This is in a series of about 2800 patients.

The most definitive study about the use of low-dose heparin comes from the multicenter trial in the British Isles and on the continent. The study tried to determine the efficacy of low-dose heparin in preventing fatal pulmonary embolism proven by autopsy. Indeed there is a benefit in the patients who receive low-dose heparin. There is a marked reduction in the incidence of fatal pulmonary embolism. But if one looks at the incidence in the control group in these general surgical patients over the age of 40 it is no greater than the incidence in the entire series of the gastric bypasses at this institution.

What about the efficacy of other measures such as stockings? We have only one objective study defining the efficacy of antiembolism stockings in preventing major leg vein thrombosis, and we recently actually aborted this study on total hip replacements at this hospital when there was a significant benefit shown in patients wearing the stockings. This was a surprise to me. I don't know what the efficacy of the elastic wraps was on the patients I have just reported. So in conclusion, I think we can say from this study that the incidence of venous thromboembolism is low. The clinical diagnosis, though, is fallible. It was four times the incidence proven by Doppler in this study. I think one can use objective screening techniques to define this incidence. I don't know the true value of the elastic wraps. Perhaps early ambulation is an important factor in these patients. Dr. Mason has described the importance of preventing fatty acid mobilization as a predisposing factor to thrombosis. I don't know for sure from this study about the value of that, but we certainly are continuing that procedure. Lastly, I think that, as an alternative to routine anticoagulant prophylaxis which may carry a risk of increased bleeding in patients undergoing major surgical procedures, one can consider prospective diagnostic surveillance of patients and treat those very few patients who actually develop the disease. Thank you.

QUESTION: If low dose heparin is used what doses do you recommend?
DR. BARNES: Traditionally, we have employed 5,000 units subcutaneously every eight hours, which is the most efficacious dose that has been shown in the literature, although some regimens have used 5,000 units every 12. I cannot really honestly say, if one is going to consider using low-dose heparin in these patients, what the ideal dose would be. It has been shown, by Hirsh in Toronto, that many patients of similar weight react differently to a standard 5,000 unit dose given subcutaneously. Some patients are anticoagulated for a period of time after such a dose, while other patients require a much greater amount. One isn't attempting to anticoagulate the patients. One is attempting to interfere early in the cascade of developing coagulation. It is my personal bias based on this study that we are, in general, not dealing with a risk that warrants routine anticoagulant prophylaxis of all these patients, especially considering the magnitude of the wounds and the potential catastrophe of hematoma. It has certainly been shown in many total hip replacement series that standard low-dose heparin may give a significantly increased incidence of bleeding. In Hirsh's own studies an average of one additional unit of blood transfusion has been required in each patient receiving low-dose heparin.
I wouldn't begin to try to tell you how to prevent all wound infections in any surgical patient, and certainly not in gastric bypass patients, but there are some things that I think apply to all surgical patients and are simply made worse when you are operating on the obese patient. In the series compiled at the University of Iowa, the infection rate has been somewhere between 8% and 10%. When I first started out trying to be a surgeon I said that wounds healed side to side and that's why I needed that big hole. The fellow sitting over there used to tell me that they dehisce end to end and they didn't always have to be so big, and wound infections are the same problem. Wound infection in obese patients, as any of you that have had that occur know, are a real problem. I am not sure what explains all of it. Certainly the fat with its possibly poor blood supply may be an important factor, although, as you all know, some people have all their fat inside and don't really have a thick abdominal wall while other people do have a very thick abdominal wall.

These people may be malnourished, even though they are very obese. In cancer and burn patients malnutrition has a very deleterious effect on a patient's ability to fight off infection. The trauma of the operation certainly has some role. The time involved in the operation is a factor according to the studies done at the Foothills Hospital in Calgary and others. The longer the operation lasts the higher will be the incidence of wound infection. The thing that was of interest to us was to discover what organisms were causing infection and from where they were coming. Previously, when I was in Iowa City, Dr. Printen and I looked at some patients and collected cultures on them from a variety of sources. In 40 patients we had 8 infections during one bad run, that is a 20% incidence of wound infection. Some of them weren't total wound, it may have been a smaller infection, but nevertheless they had cultured organisms. We cultured the stomach as well as the nasopharynx. In six of the eight infections the organisms were from the mouth or the stomach. Therefore, the infecting organisms are endogenous and are spilled at the time of surgery to the point that many of these low virulent organisms can cause
an infection. There was one that was practically a pure culture of Vionella, which is a mouth organism. We thought that if that was the case, maybe we could decrease the incidence of wound infection if we could control the contamination. Surgical textbooks often talk about gastric cancer patients that are achlorhydric. It is often said that these people should have an antibiotic preoperatively because they have a lot more organisms, and because they don't have any acid in their stomachs.

We chose for lack of any better method to start with the bowel prep that has been popularized by Condon for colon surgery. As yet we don't have enough data to draw any firm conclusions. However, out of 20 patients treated with antibiotics we only had one wound infection and the infecting organism was not one that is found normally in the stomach or the oropharynx. Although this has little statistical meaning, I think that if we know the source of the organisms that are causing the infection, then we should try and control them. It may well be that a preoperative antibiotic regimen in the stomach would be of value.

In respect to preventing wound infections beyond that, I can only tell you the way that I do the operation as far as trying to control the wound and the way that I think most of us were doing it at the University of Iowa. It is important to have the patients clean. I like to have them bathe and have somebody come in and help wash that huge abdomen several times before the operation while they are in the hospital. As is the case with any general surgical patient, the length of preoperative hospital stay is correlated to the wound infection rate and therefore should be as short as possible. The general principle of not having the patient shaved until just prior to the operation is also important. I am not sure that it is even important to shave them at all unless there is a lot of body hair. I think one of the really good things that developed, and maybe everyone is using it now, is to use the scalpel only to make the skin incision and then with hopefully a very strong assistant, tear the fat apart down to the linea alba. The first time I saw that done I thought we had regressed terribly. It is not very esthetic but it works great. I would like to always think that I can make a nice bold slash and go right to the midline. However, particularly
on the fat people I sometimes have trouble finding where it is. The tearing method obviates this problem. This accomplishes several things: 1) it avoids stair-stepping of the fat, 2) there is less bleeding, and 3) when you close the incision you have less chance for dead space because you haven't stair-stepped it and beat it up. Therefore, I strongly feel that to tear it down to the linea alba is important.

To obtain hemostasis I use electrocautery. I also routinely use a wound protector on all abdominal cases and particularly the bypasses. This is a metal ring with a plastic drape attached. It sits underneath the fascia and protects the wound edges. It keeps them moist and keeps dust and things that are falling down from getting in the wound. The closure is important and I think any good monofilament suture material that you choose for the fascia is adequate. I use wire. I think you should try to suture just the fascia. I don't think you should risk necrosing a lot of fat with complicated sutures. It is not important to put in subcutaneous sutures. I believe in the general principle that the less foreign material you have in a wound the less likely you are going to get infection. The skin may be closed however you choose, with clips or with sutures. I think the one thing that is going to help more than anything else is the fashioning of the anterior gastroenterostomy with the stapling device. It cuts the operating time in at least half, and again, for multiple reasons, the length of operating time is related to the increased incidence of wound infection.

QUESTION: Can I assume that you don't start the antibiotics preoperatively?

DR. BOYD: I don't use parenteral antibiotics, nor am I using the oral antibiotics either, because I'm not collecting data for the study.

QUESTION: Do you irrigate the stomach or the subcutaneous tissue or anything?

DR. BOYD: No, I don't. To irrigate the stomach makes me nervous. What I try to do is to prevent any spill that I can and I try not to irrigate out the pouch. I may irrigate the upper abdomen at the completion of
the procedure to make sure everything is tight, but I don't like to spill the stomach organisms because I know that is what causes the wound infections, at least in the ones we studied.

QUESTION: I wish somebody would speak about no longer closing the subcutaneous tissue. I agree with the point about a lot of foreign material in the wound, etc., but I was trained that we had to approximate the subcutaneous tissue so we didn't have a lot of "dead space." I notice that practically everybody has given up closing the subcutaneous tissue, and I wonder why.

DR. BOYD: Well, I can only say that I was told that when we close the wound, we should close it in layers with fine sutures and bring it all together. I used to get seromas, infections and hematomas. We do a lot of things because somebody said that's the way to do it, and I'm not sure that any of it is right. I know there are a lot of different ways to do any operation, but I get tired by that time in the operation. If I don't put the subcutaneous stitches in, that's one less thing to do, and I do believe that the more you contaminate, the more you traumatize tissue, the more likely you are to have infection.

DR. PRINTEN: From a pragmatic standpoint, we tried doing everything with gastric bypass incisions except hanging the patient by her heels for three days postoperatively to let everything run off that way. The wound infection rate stayed fairly constant. It didn't matter whether we put in subcutaneous sutures or whether we closed the skin with wire.
Cholesterol gallstones are found in about 10% of the North American population, but in the obese patients we are dealing with today gallstones are two to three times more frequent. As many as one-third of the patients may have gallstones. In fact, in our series, 37% of patients operated on have gallstones detected either before or at the time of operation. There has been an increased incidence of gallstone formation following the treatment of obesity by jejunoileal bypass. To date the frequent occurrence of cholelithiasis in these obese patients has been accepted with complacency. The lipid composition of the bile has not been studied heretofore. The purpose of this study was to measure the chemical composition of the gallbladder bile in the morbidly obese patient.

We studied seven men and four women who did not have gallstones, aged 17 to 50 years, with a mean weight of 137 kg. Each patient was more than 200% of ideal weight. At the time of gastric bypass, we inserted a 20 gauge needle into the fundus of the gallbladder, being careful to avoid contamination by blood, and we completely aspirated the gallbladder. In four of these patients we were able with a small scalp vein needle to obtain hepatic bile. The gallbladder or hepatic bile or both were immediately cultured and then centrifuged in our laboratory and examined under polarized light for cholesterol crystals. We then determined cholesterol, phospholipid and bile acids. The bile from 11 patients who were of normal body weight and who also did not have gallstones was used for the control. These patients were undergoing normal elective upper gastrointestinal surgery. We measured the molar percentages by plotting on triangular coordinates the bile analysis of all these patients; that is, cholesterol, bile salt and phospholipids, which are the three components of bile. The bile of all the obese patients fell outside of the micellar zone. This curve is the standard area under which the bile of normal patients should fall, and all controls, with one exception, were well below this line. Cholesterol crystals were observed in more than one-third of the obese patients but in none of the controls.
The cholesterol concentration in millimoles per liter was nearly twice as high in the obese as it was in the 11 control patients. The phospholipids and bile salt analyses were roughly comparable. When we looked at the ratio of the phospholipids and bile salts to cholesterol, which is termed the lithogenic index, the more cholesterol there is, the greater the denominator of the equation will be. Therefore, the lower this lithogenic index the greater the chance of a patient having lithogenic bile and that is exactly what the case was in the obese patients. They were actually 100% more lithogenic than their non-obese controls.

The unique thing about cholesterol in the bile is that it is completely insoluble in water, and it is maintained in solution only by virtue of the detergent actions of these bile salts along with phospholipids. Bile is termed lithogenic when it contains more cholesterol than can be held in solution. There are two possible explanations for the lithogenic bile which I have discussed. Either there is too much cholesterol or an insufficient amount of bile salts. Admirand and Small, who devised the triangular coordinates, were able to separate the bile from patients with and without gallstones. Some recent studies have failed to confirm this clear distinction, but I am going to avoid that controversy today since it is generally accepted that patients who are at risk of forming gallstones have lithogenic bile or bile that is supersaturated with respect to cholesterol. This has been well shown in the Chippewa and Pima Indians in whom the incidence of gallstones is three to four times that of the normal population. Their bile analyses are similar to what we measured in these obese patients. Interestingly enough, the Chippewa and Pima Indian patients who do not have gallstones have bile which is nevertheless supersaturated. This condition is analogous to the supersaturated bile that we found in the obese patients who do not have gallstones. One of the important implications of this study is that the bile is abnormal at the time the operation is performed and therefore, by inference, gastric bypass cannot be implicated as an etiologic factor in the development of gallstones.

We also know clinically that the incidence of postoperative cholecystitis after gastric bypass is simply not a problem and this again is in marked contradistinction to the rather high incidence of cholecystitis after
jejunoileal bypass. In summary, 37% of these grossly obese patients selected for gastric bypass normally have gallstones. Even in the patients who do not have gallstones, there is extreme supersaturation of the bile and if you plot the composition of the three biliary components you find that chemically and graphically this is upheld. The obvious question is, what happens with weight loss. Unfortunately, we have not as yet studied this. The final point I want to make is that supersaturated bile can be caused either by too much cholesterol or not enough bile acids and the current study established only that there was too much cholesterol but not which of the two mechanisms was taking place. The next paper, by Dr. DenBesten, will deal with this.
I would like to develop a thesis with you, and that thesis essentially states that the problem of the increased incidence of cholelithiasis in the obese is secondary to 1) increased cholesterol secretion by liver, which goes back to normal after weight loss, and 2) that stagnation in the gallbladder is the mechanism by which agglomeration of cholesterol and stone formation takes place.

Renewed interest in the etiology of gallstones is only about ten years old. In the mid-1960's Zollinger and Ingelfinger in the surgical and medical journals pointed out the lack of knowledge concerning the mechanism of gallstone formation and that this disease costs about $10 billion a year in operative procedures and lost time. Concurrently new techniques for analysis of bile became available. The result has been a great burst of new knowledge on the physiology of bile secretion. Many investigators took various approaches in trying to find out why patients developed gallstones. Small worked with the physical chemical basis of gallstone formation. The ethnic differences in bile secretion was also pursued to try to get at the problem. The enzymatic failure to convert cholesterol to bile acid has been studied by Schoenfield and Salen. Finally, the metabolic basis for the development of gallstones has been of interest to Tompkins, Grundy, and myself.

The main solids in bile are the bile acids, phospholipids and cholesterol. Cholesterol, the principal component of gallstones, represents about 5% of this total. Cholesterol is a fat. It is totally insoluble in a water medium. The body economy gets by this by carrying it as lipoproteins in blood and carrying it as micelles in bile. Bile acids are polar compounds with detergent properties much like soap, which are conjugated either to taurine or glycine to make them even more strongly detergent. Oddly enough, cholesterol is almost insoluble in a bile salt solution. It is not the bile salts that solubilize cholesterol, rather phospholipids mixed with cholesterol result in a substance of the consistency of jello, a thick viscid substance that will not pour. Then if one adds bile salts the suspension is broken up into smaller units
called micelles which then are soluble in a water medium like bile. To put it graphically, you might think of a micelle as two slices of bread with butter between them. Cholesterol, the butter, is between two slices composed of phospholipids and bile acids so that the insoluble cholesterol is kept out of contact with the water and thereby made soluble. Now if there is too much fat it will squeeze out from the side and one may have cholesterol crystals. If there is an inadequate amount of bile salts or phospholipid, the same thing may occur. The quantitative relationships of the three components may be represented in three-dimensional form using triangular coordinates as mentioned by Dr. Freeman, the previous speaker. If one looks at the problem of the etiology of gallstones, the thesis that excess cholesterol excretion is the central problem in the obese is gaining increased acceptance. Another alternative, of course, is that the etiology is inadequate bile salts. This does happen in ileal bypass or ileal resections where bile salts normally absorbed in the ileum are lost in the colon and do not reenter the enterohepatic circulation. With inadequate bile salts, the micelle becomes abnormal because there isn't enough soap to chop up these units into small molecules. In other instances the micelle may be unstable and this occurs during stagnation or starvation when the gallbladder doesn't empty. Without emptying the micelle becomes unstable and then cholesterol may precipitate out under conditions where it normally is soluble.

With this background we designed experiments to test the hypothesis that excess cholesterol excretion in bile may be a significant cause of cholelithiasis. The first hypothesis tested was that dietary cholesterol ingestion influences biliary lipid excretion in normal humans. To get at that problem, we took a group of patients and we fed them a zero cholesterol diet and a 750 mg cholesterol diet in random order and discovered that cholesterol in diet influenced the amount of cholesterol in bile. In a second series of studies we measured the amount of these various components coming out in bile during a 24 hour period in postoperative patients with a balloon-occludable catheter in the common duct. On a zero cholesterol diet, as compared to 750 mg cholesterol diet, there was a 35% increase in the cholesterol coming out in bile, a much larger increase than the increase in phospholipid and bile acid.
To go a little further in trying to establish this hypothesis, we took a single patient and, in random order, fed increasing amounts of cholesterol. At zero cholesterol intake she could easily solubilize all the cholesterol being synthesized by the liver. Up to 100 mg and even with 1000 mg for two weeks her bile failed to become supersaturated with cholesterol. However, when cholesterol was given at 2000 mg a day for three weeks, or 1000 mg a day for three months, this normal patient now began to have abnormal bile and the study was terminated because she had cholesterol crystals in great quantities in the bile. We concluded from these initial studies that dietary cholesterol does influence biliary lipid secretion and that normal subjects can be stressed to secrete lithogenic bile. The individual subject's response to cholesterol feeding is constant but between subjects there is great variance. At that point we decided that we needed a more consistent human model to study this problem further. We chose the obese patient and this is about the time Dr. Freeman joined us in 1972 and did the preliminary studies that he has just reported.

We proposed the hypothesis that obese subjects secrete bile which is more nearly saturated for cholesterol than humans of normal weight. The initial study just reported by Dr. Freeman showed that, compared to control subjects, bile from the gallbladder of obese subjects was much more saturated with cholesterol. We were concerned because bile was being sampled under anesthesia, after a fast, and was only a single point measurement from the 24 hour period. To find out whether this was a fluke of the initial observation or whether it was a true index to the problem, other studies were designed. Obese subjects were placed in the Clinical Research Center and then studied by feeding them a liquid diet at a constant rate and aspirating bile while the diet was being fed. From these data we were able to derive the amount of cholesterol, phospholipid and bile acid that these subjects secreted in a 24 hour period. I'll not bother you with the kinetics of these studies except to point out that this is a method by which biliary secretion can be studied without a tube in the common duct or any other invasion of the biliary tree. In briefest summary, we found that if one takes the output of cholesterol per hour in a group of normal patients and then compares a similar group of obese patients there is an increase in the amount of
cholesterol coming into the duodenum per hour over a 24 hour basis. However, and this is the point I would like to have you focus on, the obese subjects secrete at least three times more cholesterol per hour on a zero cholesterol diet than normal patients and two times more than even patients with gallstones. In contradistinction, their bile acid and phospholipid output do not change appreciably.

Looking at this in a different way, we took these patients with various weights and plotted their cholesterol output with their weight. In males there is a linear relationship between the amount of exogenous obesity and the increase in cholesterol elaborated into the bile. If one looks, then, at the bile acids to find out if this is the problem, one sees that the gallstone patients had somewhat smaller bile acid output than normal subjects. However, the obese patients have a bile acid output which is slightly greater than normals. I am trying to make the point that the error is not in bile acid metabolism as may be partially the problem in the patient with gallstones. Finally if one looks at these obese patients when a zero cholesterol and a 750 mg cholesterol diet are fed, the obese individual secretes more cholesterol with a zero cholesterol diet than do normals fed a 750 mg cholesterol diet. Adding 750 mg of cholesterol to the diet of the obese results in an even greater excretion of cholesterol in bile, and this increase is greater than the increase observed in normal volunteers.

Dr. Scott Grundy of San Diego has studied obese patients after weight reduction. In his studies the obese patients, before weight reduction, secreted bile which was invariably supersaturated with cholesterol. After weight reduction down to 138% of ideal weight, these same abnormal patients now have a cholesterol concentration in bile which is well below the lithogenic range. In other words, they are perfectly capable of maintaining all the cholesterol in solution and are essentially at no risk for cholelithiasis. So we have concluded from this series of studies that biliary cholesterol secretion is influenced by metabolic factors, namely cholesterol ingestion and a hypercaloric state. Weight reduction by gastric bypass or diet restriction will decrease the risk of gallstone formation in these obese subjects.
QUESTION: Do the postoperative gastric bypass patients have less cholelithiasis than normal obese patients who have not had any surgery?

DR. DENBESTEN: I have not done the clinical studies on this. Mine are laboratory studies. Dr. Mason, I think, told you this morning that in the postop period during weight reduction, patients are at increased risk to develop gallstones. I have not studied this aspect of the problem. Perhaps we will one day use chenodeoxycholic acid to protect these patients during the period of acute weight loss.

QUESTION: I thought bile was lithogenic during the period of weight reduction following this type of surgery.

DR. DENBESTEN: You are correct. When any human is 'starved' his bile becomes more lithogenic; there will be more chance of stone formation during the period that he is not eating. The mechanism explaining this observation is well known. Cholesterol is secreted by the hepatocyte by two mechanisms: a constant (bile salt independent) and an augmented (bile salt dependent). If they are not eating, bile salt isn't coming back. Therefore, all the cholesterol is being excreted at a fixed rate with little or no bile acid secretion with the result that bile will be more saturated. The importance of the studies I have tried to present is that in a 24 hour period there is a marked difference in the total time lithogenic bile is secreted. Everyone of us have lithogenic bile for at least one to two hours every day. That's not the problem. The fact is that the fat patient has lithogenic bile 24 hours a day and that's where they seem to get in trouble. The momentary lithogenic bile is not a problem to humans.

DR. GOMEZ: I was beginning to think that there may be some incidence of increased cholelithiasis because I have had five patients that required cholecystectomy within the first year after gastric bypass whose gallbladders were perfectly normal prior to gastric bypass. Two of these were a sister and brother, and of the other three two had acute cholecystitis with obstruction of the cystic artery.
DR. DENBESTEN: As you know, all of us vary in the kind of bile we secrete. Some of you are secreting bile which is right on the border of supersaturation most of the day, some are lower. Some of these obese patients, even at ideal weight, will be very close to being saturated, but the incidence after the period of increased risk during weight loss should be that of the normal population.

DR. DRENNICK: Why do obese individuals increase production of cholesterol when they have so much cholesterol? What happens to the feedback controls?

DR. DENBESTEN: I can't explain it, but I think if you come back next year perhaps we will be able to. Studies are being done using tissue cultured hepatocytes from prairie dogs. There is suggestive evidence that the hepatocytes, from the prairie dog who was fat, continue to elaborate or synthesize a far greater amount of cholesterol than those hepatocytes from animals who were thin when hepatocytes were harvested. These are preliminary data and much more work remains to be done.
BODY PROTEIN PRESERVATION

George L. Blackburn, M.D.
Joel B. Freeman, M.D.

DR. FREEMAN: We have a series of what we think are eight salient points dealing with body composition in the obese patient. Any semblance between an orderly conduct of these eight questions is purely coincidental! I am going to act as a protagonist and present our visiting professor with each of these points. So without further ado, here's the first question for Dr. Blackburn. George, a 300 lb, 5' 6" female, 28 years of age, comes to your office and states that she has been overweight since the age of 18. She has been in TOPS, Weight Watchers, and has lost 50 lbs. four different times in her life and gained it back each time. She is 28 years old, can't get married, wants to know what the proper treatment is, and she hears you are a great surgeon.

DR. BLACKBURN: First of all, I congratulate her on her great choice. Most of the patients that come to us have been sent by another patient. Not until they have entered our office has any referee interacted. This is a free enterprise system and the patient is age 28, wants to get married, and weighs 300 lbs. Our first responsibility to this type of patient is to give them information about exactly what it is they are coming to us for, because we cannot assume, and you should not, that the patient is in a position to make the right decision. Right away we have to point out to them that it is not up to them to decide the surgery, that they cannot know the risks and benefits of such surgery, and therefore a variety of information will be necessary beyond what has already been given. I would start off by pointing out that we do not know what benefit can come to the patient because even though at 28, she does weigh 300 lbs, we don't know what medical harm, if any, will result from this degree of overweight. I haven't been told whether the patient is hypertensive, but I doubt that she would be, or whether she is diabetic to the extent of being symptomatic. She may have an abnormal glucose tolerance test, but I seriously doubt that she has angiopathy or nephropathy or any other sequelae of renal disease and the like. Therefore, I suggest that we do not know the benefit or the risk of being that obese. We have already heard of some males who have some problems when
they are over 40. Nevertheless, this woman is only 28. Therefore, to schedule her for operation immediately is out of the question. We have obviously been very interested in comprehensive approaches. My last point here is that the patient has only tried TOPS and Weight Watchers which have a single philosophy. She may also have been on drugs; again a single entity therapy. We now are learning that some comprehensive approach that involves nutritional education, behavior modification, exercise and relaxation therapy combined with certain dietary approaches are effective. Therefore, we need a lot of workup to find the answers.

DR. FREEMAN: If there were one answer to the first two minutes of this session, it would have to be that even though the primary indication for us to operate on patients is their inability to lose weight, this inability per se, is not an indication to go ahead with operation.

DR. BLACKBURN: I think that's clearly correct. To get started I would emphasize that it's a partnership. Our hypothetical patient has lost 50 lbs before and I would get her started losing again while we get to know each other and learn the risks.

DR. FREEMAN: The second question is: "Should we tell an overweight patient to lose weight before bypass surgery or, for that matter, before any sort of an operation?"

DR. BLACKBURN: At this point, I would draw her some pictures that explain her body. I would point out that the survival part is the body cell mass containing skeletal muscle and organs, and that her obesity is like a tumor covering that survival part. The key tissue to hold on to is the protein containing muscle mass, and that is the only reason we talk about nitrogen balance. I would explain to her a little bit about metabolism as Dr. Francis Moore might have done. What you want to save is the body cell mass; the living, functioning, energy-saving and mitotically-active cells of the body. If you are going to submit her to surgery or any therapy that is going to cause loss of that, then you've got a lot of problems. This, in effect, is why we are here today; because one surgical technique clearly has been demonstrated and reported by its investigators to have over half of the weight loss as body cell mass.
the first five months. Then again potassium and phosphate losses are problems. Body cell mass consumes oxygen and produces CO₂. Whether you do surgery or not, the only way you are going to get rid of that fat 'tumor' is to burn it. The name of the game is to burn it while not losing this muscle tissue. The loss of muscle tissue is caused, of course, by protein malnutrition which carries lots of complications. As you lose muscle tissue the organs start to fail and morbidity and mortality begin to rise. We have been very interested in fasting as a therapy since fat can be burned, particularly to ketones, which can substitute for glucose and thus spare body cell mass.

DR. FREEMAN: Therefore, weight loss is not a function of nutritional status. In point of fact, paradoxical as it may sound, the patient who is forced to lose 25 quick pounds before having his gallbladder removed may, in fact, have starved himself. The early phases of starvation, according to Cahill's studies, actually have shown that what we burn first, since glucose supplies are used up in a day, is protein. The patient who forces himself to lose weight rapidly may actually be burning up the very substances that we want him to use to heal his wounds. If any kind of a diet is to be inflicted, it should be a diet that emphasizes burning of fat, namely, a protein-sparing type of diet.

Let's assume gastric bypass has been performed. On the fourth postoperative day the nasogastric tube is removed. The patient begins to vomit and continues to do so four times a day. On endoscopy we see a large area of edema and erythema, there are no sutures or staples joining the anterior to the posterior wall and this looks like edematous postoperative obstruction. What is the management of this problem?

DR. BLACKBURN: I assume that in gastrectomies we have all had this delayed outlet obstruction problem with ileus, and no obvious anatomical or mechanical defect in the anastomosis. Before continuing I would like to emphasize that even though they won't stop this obviously abnormal behavior of overeating even with the help of medical means, on the data we have now, you are not justified to diagnose this patient as intractable to medical therapy and therefore operate. The procedures are still
under investigation. I do not think enough evidence is in at this time to justify operating on everyone who wants surgery.

Returning to the question, it is not so much edema as it is a low carbohydrate diet that is causing the ileus. Starvation such as Dr. Drennick has worked with does produce starvation ketosis and a diuresis with minimal ileus. Ketosis is not diabetic keto-acidosis. A study in the New England Journal of Medicine shows that within a week of treating a Pickwickian patient with fasting to depress CO$_2$ production, the patient recovered. Indeed, these patients will come out of CO$_2$ retention.

We do add protein to supplement the fast to reduce the loss of lean body mass. I would probably treat these people who have pulmonary dysfunction and who are Pickwickian in this way. We have also been impressed that on a low carbohydrate diet glycogen stores and the water in the liver will decrease. Therefore it is easier to fold over the left lobe of the liver which makes it easier to get up to this deep, and very high hole, for a subtotal gastrectomy. The ketosis afterwards will create a natriuresis so that we can minimize water retention and ileus. Again pulmonary function will improve because the hypoxic index will change, and also EFA deficiency will be minimized. In regard to the question of fistula the key thing is its detection. Loss of ketosis is an early warning symptom on stress that would come from a gastric fistula.

DR. FREEMAN: So Dr. Blackburn is saying that protein sparing, both preoperatively and for the relatively minor postoperative complications such as stomal edema, is a potential method of treatment. To assure that everybody knows what protein sparing means, I would cite the example of total parenteral nutrition in which 25% dextrose and 5% amino acids are infused simultaneously. The purpose of this is to meet all the energy requirements with glucose so that the amino acids are used to make new tissue. During 25% dextrose infusion, insulin levels are high, and insulin inhibits fat mobilization. The antithesis of that situation would be the infusion of amino acids only. This is in complete disharmony with all nutritional theory which states that protein must be accompanied by calories, but in protein sparing we have no source of calories. There is no glucose, insulin levels are depressed, and this permits
mobilization of endogenous body fat. The point is that infusion of amino acids either into the stomach or into a vein promotes the burning of fat tissue which can be used as a source of endogenous calories, thereby allowing the administered amino acids to be used, at least in part, for protein, hence the word "protein sparing."

We are saying that this patient whose stoma does not function properly may need some form of parenteral nutrition. Perhaps the physician is reluctant to go immediately to the expense and potential dangers of total parenteral nutrition, so that for a short period of time amino acids are being infused. Such therapy was used in a similar group of patients studied here who had postoperative stomal dysfunction. During amino acid infusion there was a three to five fold increase in the free fatty acid levels. Nevertheless, we have yet to notice deleterious effects of these elevated free fatty acids. Note parenthetically the tremendous salutary effect on nitrogen balance. It goes from -9 to -5 gm/day, during glucose infusion as opposed to 3% amino acids. The question is, do the obese patients react differently to protein sparing? The answer is, they don't, even despite their very large fat stores. In a similar experiment the investigators are running free fatty acid levels around 0.8 to 0.9 millimolar and they have documented the same nitrogen improvement. In the presence of stomal edema, these patients can tolerate protein sparing if you want to use it.

DR. BLACKBURN: Just so we don't think the only way to treat these miserable children is by surgery, I would like to point out that in one of the April issues of the New England Journal we also have an article about Prader-Willi in which I think the results are fairly comparable to the ones that Bob Soper showed us this morning. We used just the protein sparing effect orally together with a behavior and exercise program. We were able to get long term two-year weight loss on these children. The point here is that we are not yet up against the wall on the approach to treatment of obesity. Therefore, we still can do some prospective randomized studies, continue to investigate this area and develop better treatments. At the same time, because we did not create the obesity we need not feel obligated to operate.
DR. FREEMAN: In summary, early postoperative stomal edema is treated in one of three ways: we can use protein sparing, we can simply put in a subclavian catheter and give total parenteral nutrition, or we could have placed a tube either in the distal gastric pouch or the jejunum at the time of every operation, which is a method Dr. Printen favors. Of course, if you do that, you are putting a tube in every patient for the occasional patient who will have this problem, but I think that summarizes the management.

Fourth question: This next patient hypothetically does very well and, in fact, on the fourth postop day she is not vomiting and you see her having her first breakfast. The breakfast consists of a large doughnut like those lethal ones we had this morning. She is putting jam on her toast and she also is having Sugar Pops with cream. What would be your comments if you witnessed this in a patient, George?

DR. BLACKBURN: I would say that I didn't do the intake evaluation properly. If going through all this effort, the patient is not further advanced in the proper modality for eating, I would conclude that I didn't have a good dietitian on my team and I quite frankly think that that speaks very poorly of the required multidisciplinary comprehensive approach to this therapy. We see gastric bypass as an adjunct to a total program. Despite the parallelism between smoking and alcohol, obesity is not a metabolic addiction, it is a behavior pattern. I think for all the effort and the risk of doing surgery that I would be chagrined and I would communicate my grief and sorrow to the patient. I am serious in stating that we are missing the boat and that although the patient is doing fine now by loss of weight, in a year or two this is all going to be for naught without a new life style and behavior change.

DR. FREEMAN: We are poor surgeons, then, if we do this operation and allow a patient to consume calories in an improper fashion. Not only is this meal a very poor one nutritionally speaking, that is, protein deficient, but things such as putting jam on toast are a perfect way to maintain your weight. The patient's behavior modification has not been properly managed. If we want to do this operation it behooves us, then, to have proper counselling with the patient beginning before the operation.
You are in your office seeing patients and a gastric bypass patient who is one year after surgery says the following: "At first I lost 2½ kg a month. I am now down 30 kg and I am beginning to taper off. I am not losing as much, which (a) I'm not happy about and (b) I have a next door neighbor who went elsewhere and he had a jejunoileal bypass and he is losing much more weight than I am and I think you did the wrong operation."

DR. BLACKBURN: Well, this is clearly the controversy now. Those of you who are up against the jejunoileal bypass must be hearing this all the time because this is certainly one of the big points. I would again get out my pencil and paper and show the body composition saying that the weight loss that one loses can consist of fat or body cell mass. There are two ways to lose weight. In uncomplicated surgery about 10% of the weight loss is protein whereas in big stress situations as much as 50% of weight loss can be from body cell mass. Again, if it is protein you are losing, you will also be losing liver protein, albumin, immune defense, ability to repair tissue, and of course they sap away their reserves, fitness and well being. What I would then point out is that with this operation, weight loss is mostly fat. The key point here is that in gastric bypass less than or about 10% of the early weight loss is protein whereas with the jejunoileal it is about 50%. With this explained the patient can take the choice. They can talk about weight loss or they can worry about fat loss, and if you have a skin caliper you can demonstrate that. We had one patient who changed her behavior including giving up candy bars. She had 5 kg of fat loss per month for 15 months, and she vigorously exercised. She gets her fitness tested on a treadmill which takes 5 minutes. Putting these people on a 300 or 600 Kp/m exercise test on an exercise bicycle or treadmill, while measuring their pulse, will tell you in what kind of fitness they are. A comprehensive approach includes nutritional education, exercise, and consideration of surgery, because without the surgery morbid obesity is difficult to treat. Again I am encouraged on an investigational basis to use gastric bypass as an adjunct but I have serious reservation about not using a multidisciplinary comprehensive therapy. I'm against surgery as the total treatment.
DR. FREEMAN: And when we figure out a way to get these large patients in the total body counter, we can do total body potassiums and prove the whole problem.

DR. BLACKBURN: Those lean body mass measurements were made by a whole body K-40 counting.

DR. FREEMAN: She fit in?

DR. BLACKBURN: She fit in. It is an old World War II turret made out of iron that is not radioactive that ....

DR. FREEMAN: A tank.

DR. BLACKBURN: A tank. The counter will take them quite nicely. The clinician can test his own patients. Every six months, a 24-hour urine creatinine clearance will allow the calculation of creatinine height index for your patient. This will allow you to determine exactly how much muscle mass they have lost. The collection of a 24-hour urine will tell you the status of the lean body mass just as assuredly as a K-40 counter will.

DR. FREEMAN: And that was in the American Journal of Clinical Nutrition in December, 1976, "Creatinine Height Index." I hope you will take away from this that there is more to gastric bypass surgery than doing the operation.
Before I present the statistics that we have recently gathered, I would like to give you a brief description of how we collect our information. We gather our data preoperatively, operatively, and postoperatively at six weeks, six months, one year, two, three, five and ten years, etc. We have special forms that become part of the patient's record especially devised for this purpose. These forms are put into the patient's chart when the patient is evaluated in the clinic. This facilitates abstraction of data later and also helps to insure that the same data are retrieved from each patient. The operative data, such as measurement and calibration of stoma size, length of operation, length of time on respirator or intubation, and so forth, is taken directly from the chart. We also have used questionnaires, as Dr. Mason has pointed out earlier. We are trying to get a little more reliable on this. It is difficult to write a good questionnaire that eliminates bias. One cannot completely eliminate bias unless you track down the patients that did not answer to find out the reason for this and how they would have answered. The difficulty of obtaining complete information is always a constant problem with research of this type.

During review of patient charts, the information is stored on abstracts which are designed to go directly to keypunching. The data are then put on computer punch cards. After completion of a few practice runs to identify and eliminate the punching errors, we store the information permanently on computer tape.

After we have our information stored we periodically do the statistical analysis. The method of analysis has been with a computer program called "Statistical Package for the Social Sciences" or SPSS. This is an integrated system of computer programs designed for the analysis of social science data. In addition to the usual descriptive statistics such as simple frequency distributions and cross tabulations, SPSS also contains procedures for more complicated statistics such as analysis of variance, multiple regression, discriminate analysis, canonical correlations, etc. We have recently been using regression and discriminate
analysis to try to determine an equation that would predict success or failure for a prospective gastric bypass candidate. As yet, it is much too early to speculate on which variables will be the significant ones. I am not sure whether we will ever devise an equation which will be predictive a significant number of times, but if we do we will certainly let you know about it.

Before going on to the presentation of our most recent laboratory values, blood pressures and information on diabetes, I do want to emphasize the importance of obtaining complete sets of values for each case or patient. Whenever there are missing values the more sophisticated types of statistical analyses are compromised. The possibility of spurious, misleading and useless statistical results increases proportionately to the amount of missing data. The more missing information there is the more problems you have with your research. Anyone considering a prospective study on any subject should try to get as much data as possible. I realize it is not always possible in medical research since we are often dependent on the patient for followup. I noticed some people mentioned the word 'contract' when they were talking about their patients. Perhaps this might be a valid approach for setting up a prospective study. One could make a pact or deal with the patient in which the patient guarantees a certain number of return visits for which the surgeon guarantees a flat rate. I am not sure how one would work out that type of arrangement but it would assure the proper followup data to yield good statistics. That ends my little pitch for the statistics. I am sure you are more interested in some of our results.

Out of 566 patients who have their data currently stored on the computer, we had 46 (8%) who were diagnosed diabetic preoperatively. Thirty of these patients answered a recent questionnaire. Preoperatively four claimed that they were diagnosed but had no treatment, 8 were controlled by diet, 13 had to take pills, and 5 were on insulin. Postoperatively, after weight loss it is interesting to note that 17 of them claimed they were no longer diabetics at all. Two of those who previously took insulin no longer need it. What is even more interesting is the fact that many of the less severe diabetics needed no control at all postoperatively.
At one year followup we have a drop from 146/90 to 138/86 as a mean blood pressure. As the length of followup increases the drop in blood pressure remains fairly constant. Unfortunately we have lost many patients to late followup. This is a perfect example of the problem of trying to present statistics like this when you have a lot of missing information. Naturally, since some of our patients were just operated upon this year, they couldn't as yet have a five-year followup. However, there is a significant number of patients that we could have five-year followup on if we could find them. One nice thing about this operation is that they do lose weight, and, therefore, they don't see any reason to return. Conversely, if they don't lose weight they sometimes get upset with us and never want to come back. The problem, then, is that the sick ones are the ones we often see coming back at three and five years and this may be one of the reasons blood pressure goes back up somewhat with extended followup at five years.

None of this is significant statistically. Perhaps the most important thing is that basically most of these values do not change. Sometimes when you don't have a change this can also be good, particularly in regard to the laboratory values. Hemoglobin drops at six weeks, which is a constant and to be expected with major abdominal surgery. After the six week followup there really are no abnormal values. As for total protein, albumin, calcium and $P_{O_4}$, the values stay basically the same. There is no real change, whereas with some other types of surgery or some of the fad diets, you might expect to see some changes. There is somewhat of a drop in glucose, but basically everything is staying within a fairly normal value. One thing that is interesting to note is the bilirubin at six weeks. It is still within normal limits, but there have been a couple of patients that have received certain anesthetic agents, namely Halothane, and had some problems in the immediate postoperative period. There is even one patient that expired suddenly about six weeks postoperatively, and on postmortem the only thing they could say was that she had fatty metamorphosis of the liver. It is suspected that her Halothane anesthesia was perhaps a contributing factor. Triglyceride and cholesterol levels come down a little, but basically they stay just as they were. In conclusion, I would say the most important finding as far as laboratory results are concerned is that they stay
within normal limits and there are not any significant changes that you need to worry about. Unfortunately, we have not completed many of our studies as yet. However, preliminary data suggests that patients with preoperatively abnormally high laboratory values or blood pressure benefit from the gastric bypass produced weight loss in that their values return towards normal levels.
DR. FREEMAN: It's one thing to say that some of these patients do have problems and do need intravenous access but it is another thing, quite often a different problem, to find the vein. I am presuming that peripheral veins are used up and we do have a problem such as stomal obstruction or gastric fistula. Although it may seem paradoxical these patients have an excess of fat but their protein may be abnormal and they may very well need total parenteral nutrition despite being two or three times normal weight. The needle of a standard 14 gauge intracath is used throughout this country for placement of subclavian catheters. The length of this catheter however, is inadequate to reach from the skin to the subclavian vein of a massively obese patient. A #12 Argyle Medicut works by the opposite principle. Instead of being an intracath it is an angiocath, that is, you puncture the vein with this needle and the catheter slips over it. Its purpose in our hospital is in placing subclavian catheters because this needle is much longer than the conventional intracath. I puncture into the subclavian vein and slip the catheter antegrade into the vein; it is really almost like a Seldinger technique without a guidewire. Next, I withdraw the needle so that the catheter is inside the vein and then through this catheter I slip the 14 gauge intracath. You end up with the catheter in the vein and the needle, then, is just pulled out and sits on the skin of the chest wall. At this point it fits perfectly over the hub of the intracath. There are two points about placing subclavian catheters in these patients. Although we conventionally shoot for the suprasternal notch, as you all know the anatomy is so distorted in these patients that technically your needle really has to be pointing much more posteriorly than normally in order to get the vein or you will just keep hitting the clavicle or the costoclavicular ligament. The second point is, don't tape it over the breast in a female because the breast is so pendulous it will frequently make the catheter go in and out to the point that you may find yourself infusing into the subcutaneous tissues and that can go on for a long time undetected. Tape the catheter well down between the two breasts so
that movement of the fat does not affect it. There is a recent modifica-
tion of the intracath not yet available. Although we have some I don't 
think it is on the market. This again is the standard Basiray #14 gauge 
8-inch intracather with a 1½ inch 14 gauge needle. The needle goes in 
the vein, the catheter with its guidewire goes through the needle, and 
then you must put the protector so that the tip of the needle does not 
cut the catheter. This is reasonably cumbersome. The new catheter's 
needle is about the length of the Argyle that I mentioned earlier. It 
is almost 2 inches, and believe me it does reach the subclavian vein in 
a 311 lb patient. This new catheter, which only costs 30¢ more than the 
old one, is going to be available soon and I strongly recommend it to 
you, not only because of the length of the needle but, like the former 
one was, has built into it a blunt endless needle. The catheter goes 
through this needle and once the puncture is successfully completed this 
needle comes off and you just clip on the hub and therefore the patient 
does not have the cumbersome guard and protector of the conventional 
method. I think it is going to be an extremely helpful catheter for 
safe subclavian venipuncture in these patients.

COMMENT: I used that catheter for a while. The problem that develops 
with that catheter is that everybody has to be alerted that the metal 
bar in there is not a marker for the x-ray to see where it is once it's 
inside the chest. I've seen that happen. Another problem is that when 
you put the catheter hub on the little needle that's in there it has 
ocasionally come apart resulting in a lot of yelling and screaming and 
blood and fluid running around. The instructions say to screw it on. 
Not very many people read the instructions when they use these things, 
they just automatically use them. There's a little thread inside there 
that helps it from coming off, but it still does it. I found that if 
you put in a subclavian catheter, the ones that work the best are the 
one's that go in with the greatest ease. It is impossible to put that 
catheter in with great ease by yourself. Therefore, it is going to take 
at least two people to do it with ease.

QUESTION: Can you suture your catheter to the skin?
DR. FREEMAN: Not only that, but this catheter makes it much easier because the metal that is in the tip gives you something to tie around without obstructing your catheter. I should have mentioned that as a relative advantage. The former one is not easy to suture. Why somebody hasn't made a catheter with an eyelet on it I'll never understand, but there isn't one. The new catheter is much easier to suture because you put a suture in the skin and then tie very firmly around it. I am not working for this company, by the way.

DR. BLACKBURN: Mr. Blommers didn't mention anything about those hypertensives that you've had in your series. Are there any statistics on what has happened to them? What has happened to their hypertension after they have lost weight?

DR. MASON: Twenty-eight patients with preoperative diastolic pressure of 110 mmHg or higher were studied at one year. There appeared to be a correlation between decrease in blood pressure and decrease in weight but I believe this relationship is spurious. When the change in blood pressure is compared with change in percent excess weight it appears that there is no correlation and that all patients (with 2 exceptions) had at least a 10 mmHg decrease in blood pressure. The median decrease in diastolic BP was almost 30 mmHg.

Fourteen of these patients provided 2 year followup data which indicated a median drop in diastolic BP of at least 20 mmHg. These data suggest one way in which weight reduction by gastric bypass will probably improve survivorship. (This information has been added in the editing and was not available during the workshop.)

DR. BLACKBURN: I have a question for Dr. Mason in regard to the case that Dr. Freeman presented to me, a 28-year-old 300-pounder. In looking at the data that was shown us, you evidently in your population are not seeing hyperlipidemia except for a small group of the 500 because it didn't come out in the mean, or hypertension, so we've really not seen it. Thirty of the 500 patients evidently, or some small group, have diabetes. Since there aren't the risk factors, can you tell me what are the guidelines, if such a patient presented to you, how you handle them?
Do all patients get gastric bypass as long as they are not psychotic?
How do you handle the asymptomatic 28-year-old 300-pounder who wants to
get married and wants to lose weight?

DR. MASON: They must have tried all other means of weight loss. I must
confess that we go ahead with the gastric bypass. However, I think your
thesis that they ought to be tried on some protein sparing fast or
protein augmented fasting, is a good one. One of the problems with this
is that we don't have the help to handle it. I understand that if you
want to do this you need to have a program where you can actually teach
these people body composition, physiology, exercise, motivational changes,
etc., and I think this is excellent. This is what we ought to do, and
maybe what I ought to do now is to cut back on my operating and start
doing more psychiatric counselling and more instruction.

DR. GRIFFEN: Dr. Blackburn, how many patients do you really get who
follow the ideal program? They sure are addicted to food. We've had a
great thing with behavior therapy and they've even sent me the patients
because of failure of their treatment. I think it's a great idea and I
think some of them will do it, but very few of them.

DR. BLACKBURN: I think that your questions are good, and I'm not here
to push protein sparing. I am a user and a firm believer in the role of
gastric bypass, but I think we are going to be under the same criticism
that the jejunooileal people are, in using gastric bypass with no studies.
I am concerned that any of you, private practice or academic will say we
can't follow the patients because they leave us and we can't get to
them. I think you must build in, front-end if you will, in your surgical
fees, what have you, the capacity to fund researchers to find these
patients, because it is investigational.

I'm certainly convinced that the chances of most people who are 300 lbs
to get a cure medically are not very good. What I'm concerned about is
that in 1977 if a patient can't lose weight any other way they should
get a gastric bypass. I have reservations about that at this time.
QUESTION: We all have reservations. I think what you are really asking is what is the bottom line. At what level does it become optional, at what level is it going to become a strong recommendation?

DR. BLACKBURN: I think you need to get a partner that will followup and keep records and treat it comprehensively. I think there are a few such people around and they are going to give you your best feedback and be the most productive.

DR. FREEMAN: George, are you saying you are using the operation as part of your behavior modification? Are you going to use the operation as a reward much as a mother might say "Lose 20 lbs and I'll buy you a new suit or a new dress." Are you trying to pick the patient by those that lose a certain percentage but not enough by protein sparing or some other means?

DR. BLACKBURN: I'm trying to pick the people who are in partnership and are not trying to use me. It's more a partnership rather than reward. You do your part, and I'll do my part, and we'll get something accomplished.

QUESTION: Why should there be any question about use of a procedure that can be applied with a low mortality and morbidity to the treatment of people who are more than twice their estimated ideal weight? Morbid obesity is a disease, isn't it?

DR. BLACKBURN: I have to disagree with you, and I would ask you to produce that result. We just saw 500 entries here with normal blood pressures, with mean normal triglycerides and cholesterols, and if it's true, if it's a disease as you say it, that's counter to editorials written in the New England Journal. It's not recognized as a disease by any insurance company for primary treatment. I think the burden is on you to prove that point.

DR. PRINTEN: Try and buy insurance, George. If you're 20% overweight you can't do it.
DR. BLACKBURN: I'm talking about medical insurance paying for operations for obesity. You can get life insurance at increased premiums. That's already been pointed out by Dr. Mason as one of the ambivalences in the industry. But nevertheless, to say in 1977 that every disease you diagnose you have to treat, I wonder if you can really say that. Almost all of us come up with diseases that need treatment that we can't provide. All I'm saying is that in 1977, there must be prospective studies to get this properly in focus. You can't just say that you've got a patient and operating room time and that's all it takes.

DR. KRIDELBAUGH: I'd like to clarify some of the statistics that are presented. You've talked about diabetics who claimed they are no longer diabetic after weight loss. If you take only the hypertensives out of your total group, what happens to their hypertension? On an average, what happens to their hypertension in the course of weight loss? In my experience they are improved and this should translate into prolonged life and decreased morbidity. When I interview these people I'm in a critical legal situation as a private practitioner rather than an institutional investigator. Therefore I can demand of these people that they demonstrate some of the signs which I consider to be potentially catastrophic in morbidly obese conditions. They must be morbidly obese. I don't think that the weight of 300 lbs. means a thing. It depends upon whether that 300 lbs. is on a 4' 9" woman or a 6' 7" basketball player or football player. I want to know what their body weight ought to be.

I want to know if they have hypertension or if they are showing some physical defect of an orthopedic nature or something that needs weight reduction. I disagree with you that this is a medically treatable condition. These people are foodaholics. If they don't touch a bite of food they can stay off food for a long time. It may be their acidosis contributes to their continued anorexia. Nobody has mentioned the reports in the medical literature showing in the last year that the patients with jejunoleal bypasses were losing weight strictly on the basis of decrease in caloric intake, probably due to the acidosis and cramps and aversion to diarrhea.
DR. BLACKBURN: I said there was no metabolic cause of obesity. These people have a behavior that is abnormal and they are consuming food in an uncontrolled, involuntary manner, in excess of need. It is not a metabolic addiction, it is behavioral. Maybe we can get Dr. Drennick, who probably really knows more about this, to give us some guidelines in this area. I agree that these people have abnormal behavior and they are eating themselves to death. But, if the patient says he wants to get married, where does that fit in your categorization? What are the indications?

DR. MASON: Let me ask George, on his data about blood pressures, how does he know that a blood pressure taken in a 300 lb. person with a particular sized cuff and particular sized arm, has true meaning in relationship to the blood pressure taken after weight loss? Are these determined with arterial punctures or are they just a cuff, what size cuff?

DR. BLACKBURN: Well, of course, the blood pressures have to be determined with a large cuff for the large arm. We know there is a difference between the obese cuff pressure and the true arterial pressure, but the variables I am talking about, Ed, are way beyond that. They are people who start off with diastolic pressures of 110 to 120 that drop to 80 or 90. I think it is beyond the technic area. Is it out of place to ask Dr. Drennick to input as to what our obligation is to treatment for these massively obese people? He has probably done as much as anyone in the field.

DR. DRENNICK: Thank you very much. It is a very big question and I am afraid I have no good or valid or all-inclusive answer. Everyone of you will have to individualize for any one particular patient. The reason I am here, as an internist, I think, is a testimonial to the fact that I don't know how to permanently relieve these obese people of their obesity. George, you say it is not a disease, and I think in the strict sense you are correct. Obesity in itself, perhaps, is not a disease, but I am quite convinced that anyone who carries 100% excess weight over a period of years is going to damage his body in a variety of ways. The figures that I mentioned to you this morning about reduced survival in the very
obese individual are simply a result of the wear and tear to which the superobese individual is subjected. If you expect that so many years of obesity are doing harm, you should do something to prevent that harm.

How are we going to do it? I will admit to you that in those 200 or so patients that we were able to treat and reduce to normal weight, after a 10 year period only 7% remained reduced even though they all had been reduced to normal weight. I know that George is doing a better job with keeping people reduced, but I think he should continue that and he should follow his patients very carefully for ten or more years and see how many of his patients will still be reduced at that time. Our patients who have no followup care to speak of or very little, all stayed reduced for two or three years, reasonably well, but after that there was a very rapid lapse and they regained. George says he only accepts a certain number of potential behavior modification patients, but what to do with the others. I think we still have to offer them something, even though they may not be suited for behavior modification or protein sparing fasts. I think that with some exceptions, which most of us are able to spot, anyone who is 100% excess in weight, not an absolute number but excess weight 100%, and who has been in that weight range for 10 years is almost certain to be a hopeless medical treatment candidate. I just say that from experience. You may point out five or ten years from now that I am wrong, but I don't think that you or I should wait and put every obese individual to the risk of the next ten years with the chance of developing all those diseases they eventually may succumb to just because there may be a better treatment ahead that we can recommend ten years from now.

DR. BLACKBURN: Would you randomize now? How do we decide who we do or do we do them all now? I did not say I didn't think it was a disease, being 100% overweight. I feel the opposite. I'm concerned about getting the studies so that it will be established by criteria rather than just gaining 500 cases with some weight loss. We haven't heard the five-year followup of those cases. What is your recommendation on getting started today? We certainly want to treat some of them. What are the guidelines at 100% overweight?
DR. DRENNICK: I think that we have to separate very, very clearly what our desires are and what the patient's desires are. They are two totally different perspectives. Your desire is to furnish objective scientific information and criteria. The patient is not interested in that. He knows that he can't get a job, that he has been unemployable for the last ten years, and he wants to finally live. Even though it is very desirable that we should accumulate objective data that would be guidelines for all of us, I think I would still treat almost any patient who, by what we have discussed this morning, indicates that he is sincerely motivated and has a purpose in losing weight. I would not send him away, I would not defer beyond maybe two or three months to see if this man is really determined to have the surgery and accept the risks and possible ill effects. I don't think I would turn anybody away any longer. I think that morbid obesity is a serious enough threat to the patient's normal health and life span that we have to help him.

DR. BLACKBURN: Ed, you must be the only people who have a five-year followup. What is the mean change in this 100% overweight patient?

DR. MASON: (added in editing) We have some data for 10 years and 42 patients have been followed for 5 years. We have observed a 35 kg weight loss at 5 years, 24% of initial weight, 43% of excess weight and 27.7% standard deviation of percent of excess weight. Others are observing better weight loss and our data is better now for the patient operated upon with a small measured pouch volume and a small stoma. The earlier data indicate that the one year followup data are a reliable index of the five year figure. On this basis it appears that our 5 year results with a small pouch should produce a 39 kg weight loss, 31% of initial weight, 55% of excess weight and a standard deviation of 17.9% of excess weight.

DR. DRENNICK: With an average individual, and I think this is probably in your experience as it is with most of us, the average obese individual male will be between 300 and 350 lbs. If you can get 80 to 100 lbs off an individual like that, I don't know if he is going to be much healthier but certainly he is going to have a much better quality of life and I think this is an aim and a goal that is worth our pursuing.
DR. BLACKBURN: I think we can't have it from a better source, so the goal of 80 to 100 lbs looks like the criteria of major importance.

DR. RANK: I do feel like I have to just say that we have to be careful to ask the right question, and we almost got off on the wrong question. I believe the question we are trying to get at is, will this work and can we do it safely? Now if we get off into equitable considerations we are going to lose a little bit of ground. I have never been morbidly obese, but I dare say that there are probably some of you here that have gone into this because you were, and I would hate to be confronted as a 350 pounder when someone tells me I had a good life. So we must decide: will it work and can we do it safely, and are there any harmful long term ill effects? The plastic surgeons don't ask you if you have a disease when they do a mammary augmentation, and they don't ask you if you have a disease when they fix a face or a crooked leg, or a rhinoplasty if you're a salesman. Let's not forget all those things. There's one last little thing. I've had a chance to talk about motivation, that's my business, and I talked about it this morning. There are ways. But right now everyone think about one question. Do you know that no one has looked at what are food sensors. For example, you come in after a hot day and you're warm and you start to drink some water. You drink a glass, and you drink another glass, and that's enough and you quit. Have you ever noticed that? You have some sort of sensor that tells you when you have enough water. We don't know anything about food sensors, and we're forgetting something. When I sit down to satisfy myself, I'm feeding 165 lbs. But that person who weighs 265 lbs is feeding 265 lbs and we don't really know about what the sensors are. Do the thin people have something that says "that's enough for your weight", and are the fat people feeding a 300 pound weight. That would be a good question to look at.
I'm one of the rabbits on the gastric bypass tour. If I have anything to contribute to this workshop it may be to tell some of my experiences to the people who have not done gastric bypasses and are wondering whether they should start or to those who have done a few and are wondering whether to continue, particularly if they are doing them in relatively small hospitals. Also, after hearing yesterday's heroes it occurred to me that perhaps the workshop needs a villain and I might be able to supply that. I am a general and thoracic surgeon at Thayer Hospital in Waterville, Maine, where we have 175 beds and no interns and no residents. This is not going to be a scientific talk in any way; it is going to be personal and anecdotal. I will try to tell you how, in my experience, gastric bypass fits into private practice in the community hospital.

Since 1970 I have done 80 gastric bypasses, 52 of them in the last two years. My assistant on all but seven of these patients has been a board-certified general and vascular surgeon who is perfectly capable of doing a gastric bypass but he has never done one. He has had considerable exposure to my postoperative patients when I am away. When I ask why he doesn't do gastric bypasses, his invariable answer is "I just can't stand these people." My secretary is a highly intelligent and well disciplined college graduate. She does not approve of what she calls my hobby. She says bypass patients are a nuisance, have no phone numbers, move around a lot and never keep appointments. I'll enlarge upon the obvious implications of these attitudes later, but first I'll describe how I got into the bypass business.

Back in 1970 a gaggle of internist, psychologist, rehabilitationist, social service varieties and what not had been struggling for three years with a 350 lb 30-year-old mother of three who was divorced from a husband who also needed psychiatric help. Finally the internist came to me and said "Do something." I knew nothing of surgery for obesity and had no interest in it, and jejunoileal bypass was the only procedure I was aware of. I read up on how to do it and did one on this patient.
She lost 100 lbs in the next year but spent most of the time in the hospital with electrolyte and other problems. Again our internist came to me and said "Do something." I had become aware of gastric bypass, so I put her small bowel back together and did a gastric bypass. She got down to 180 lbs, where she has stayed since then, and she has also stayed in the hospital, working part time, when she doesn't have phlebitis, and every day she says "Hello, Dr. Hornberger." For the next three to four years I did a bypass about every six weeks, and my fourth patient, a 350+ lb 5' female died suddenly on the third postoperative day, very likely due to a leak. I decided to terminate my career in obesity.

A couple of months later, though, I was called to a coastal town by a general practitioner who virtually commanded me to do a bypass on a lady whom he was very fond of and very tired of. She was a 52-year-old 350 lb widow with a 14-year-old son. She had no money, no hope, a total cripple physically, economically and emotionally. She eventually got down to the 170 to 180 lb range and stayed there. She has taken an LPN course and has become a useful, self-supporting citizen and sort of a mother to many of my subsequent patients. Her son has also developed. He goes 280 and is attending a chef's school in Providence. Always reluctantly, I did a few more and in the fall of 1974 I operated on my first male patient. He died three days later, also of a leak. He was number 23 in my series. This gave me two deaths in 23 cases, a mortality rate, according to my secretary, of 11.1%. This was certainly not an acceptable statistic for an elective procedure, particularly in the microcosm of a small hospital where everybody counts. The operation had achieved a bad reputation. I reevaluated my own position and attitude toward gastric bypass. Despite a bad record, patient referral and occasional doctor referral caused someone very heavy to show up in my office every two to three weeks. I discussed the problem with our Medical Director and a few others who felt that the need for this kind of surgery was minimal but since I had started, why didn't I learn how to do it.

In late 1974 I boarded a series of airplanes and taxicabs which brought me to a motel on the outskirts of Iowa City. In the dining room of the
motel that night I had catfish for supper, figuring you only live once. The next day I met Drs. Mason and Printen, talked, and watched Dr. Printen do a bypass. It turned out that with minor variations we were doing about the same thing. I got useful hints about the postoperative care and danger signals but left with a great degree of skepticism. These folks had residents who knew what to do and knew what was going on and could reinsert subclavian lines and ride herd postoperatively. If I started doing bypasses again I was still going to be very much alone. I went home undecided as to what to do, and the real question was, was I willing to stand the grief. Somewhat fortified by my Iowa exposure and modestly in demand, I did a few more with good results, although not without trouble.

The turning point in my own attitude and confidence came in May, 1975, when Big Joe came to see me. I am telling the story to make a simple point that obese people are absolutely desperate. By this time I had developed a basic sales talk which went about like this: "I have done 27 of these operations, I have had two deaths for reasons possibly but not certainly preventable. I have had two marginal ulcers, two or three pulmonary emboli, five wound infections plus other complications. It is a major surgical procedure with a possibility of a variety of major complications." I didn't want the patients to be as scared as I was, but I wanted them to be wary. Getting back to Big Joe, he was 32, weighed 465 lbs and filled the hallway between the waiting room and my office. I have made a point of watching each prospective patient as he or she takes this walk. I deny the allegation of my surgical assistant that if they can make it I'll operate on them. Big Joe, a construction worker, walked the 20 yards with a springy, almost jaunty step, despite his weight. In the course of history taking, I asked him about his parents. He told me that his father had died while being operated on for a hernia. I thought to myself, this sort of thing used to happen around here with all the GP surgeons we had in the town in those days, and I said, "When was that and which klutz was operating on him?" "You were," Joe said, "about 15 years ago." That's a story in itself. I said "Yes, I remember ... now." "Look, Joe," I said, "I've only done one as big as you and she's had a lot of trouble. I've only done one other man, and he died, and I operated on your father and he died. How
do you like them odds?" Joe said "Doctor, the way I'm going I'll be
dead in five years anyway." I really wanted to run away from Joe, but I
figured that he was a needy client. I offered to ship him to Iowa City,
but he couldn't or wouldn't. He had the operation, he broke his incision
open four days later, and I got it back together. Three weeks from the
time of his bypass he was at work in his company office. I have since
repaired his incisional hernia, and 18 months postoperatively he had
lost 250 lbs and has been readmitted to the Marine Corps Reserves. He
weighs less than I do and is generally considered better looking. As a
result of determination, weight lifting and other exercises he has very
little flab on his belly or anywhere else. A result like this feeds a
surgeon's ego and it also feeds his practice. Joe is now back building
roads all over the State of Maine, I have done a bypass on his sister
and probably a dozen overweight females and one male he has encountered
in various hash houses or just along the roadside. Joe will stop when
he sees someone fat walking along the road and talk to them, and, likely
as not, give them my name.

Now I'd like to discuss to some extent three problems which seem signifi­
cant to me. One is the individual physician's attitude and philosophy
toward gastric bypass, the second is selection of patients, and the
third is the medical profession's attitude towards weight-reducing
operations. The first, the individual doctor's attitude, I was pushed
although not forced into bypass surgery. I will admit to having become
at least mildly fascinated by it. This may simply be because my results
are so much better than my results in surgery of lung cancer or other
cancer. I think the chief factor is that a high percentage of these
people are so delighted with the results that they make me feel good, or
maybe they make me feel important when it works out well.

Secondly, the selection of patients. With few exceptions, I have adhered
to the basic principles that they should be twice normal weight or at
least 100 lbs overweight with a five year history of futility and not
over 50 years of age. Basically what I have done is bypass virtually
all of them who have met the qualifications with the attitude that I am
purely a mechanic and can probably make them thinner, at least get them
through the average door. I see no major role for psychiatry in this
business and am particularly wary of patients referred to me by psychiatrists. I have rejected only two patients who have met the standards. One was a pickwickian 40-year-old man who had already had a bypass. It was a 75% bypass instead of 90% or more. His wife said that all he did was sit in the kitchen and drink beer and eat hamburgers. He sat before me like a giant bullfrog and burped every 15 seconds. There was nothing I could do. This might have been an interesting surgical exercise, but the patient's attitude was negative and I demand positivity. The other rejectee was a 23-year-old male, a member of the uninformed, uneducated and combative proletariat. He asked innumerable meaningless unanswerable questions and he paid no attention to anything I said, and took an hour doing it. I told him I could tell him everything I knew about the operation in five minutes, that there was no way I wanted to sell the operation, and that the only people who get it are those who qualify and beg for it or come close to that after being told clearly and repeatedly of the possible problems and risks. The patient complained to his welfare worker that I had been impolite to him. That may be, but I won't operate on him.

I think I have reached the point where I can make a fairly shrewd guess about how a given patient will do. The ideal patient is a reasonably intelligent, basically sensible, still married housewife and mother who is not divorced, likes to go swimming or bowling, has a few outside interests and just happens to be overweight. These are the people who come in, ask a few basic questions, and listen to what I have to say. I give them a list of earlier patients who are willing to talk to new patients, and this kind usually simply says "Put me on the schedule, Doctor, and yes, I would like to talk to the former patients." The other extreme is the patient frequently referred by a psychiatrist or someone in some clinic who will do anything to get off the hook. This patient sprawls before you with a face devoid of expression, understands little or nothing of what is said, but declares that she wants the operation. One classic statement was "The old man has got to roll me out of bed. I can't get up my owself." This 30-year-old 300 pounder arrived in a battered pickup truck with a skinny husband and four children. This kind I will do, if enough people get after me, but I do it with reservations because I know that even though they lose weight they
may still be a burden to themselves and their family and even society. Between these extremes is a fascinating variety. As far as I can determine, the most profound statement one can make about them is that they are better off thin or approaching it than obese.

In reviewing my series of cases, I can pick out at least 10, or roughly 15%, that I wish I had never heard of, even though the surgery may have achieved the desired result. I can pick out another 10 whom I classified as bad news preoperatively but have become good results in every way. I would like to reject the patients whom I instinctively don't like or whom I instinctively feel are not good candidates, but I don't really think one should be that whimsical in the selection of patients. This brings us to the subject of the medical profession's attitude toward weight-reducing. Although alcoholism and drug addiction are now accepted as diseases, many people in and out of our profession look upon obesity as something undesirable and something vaguely sinful and unpleasant, and something which is obviously the patient's own fault. Although there are crusades against obesity, we have no modern Carrie Nations who fire bomb these stores that sell Pepsi Cola and potato chips. We do have Weight Watchers and various organizations which are helpful to many. Perhaps I see only their failures, but my impression is that they are not as effective as Alcoholics Anonymous. As doctors, whatever our specialty, we avoid obese people. Their medical, surgical and psychiatric problems are magnified, obviously. A lot of doctors are repelled by them and have no sympathy for them. Two friends who for over 15 years have sent me all the surgery they could find or conjure up, have never referred a gastric bypass patient. One of them has said, "I don't see why you are doing this." Other doctors in my community who frequently refer surgery have sent a few bypasses, but usually it is a weak referral. They may see a patient and suggest that he or she be sent to me, but they don't call me directly. Perhaps one reason for this is that about 20% of my patients have been such a nuisance to the whole medical community in one way or another that no one wants to get anywhere near the action.

I spoke on gastric bypass to the New England Surgical Society in the fall of 1975 and the paper appeared in the American Journal of Surgery.
As a result I've had referrals from areas I don't usually serve, as well as a few from out of state. With rare exceptions this is still an oblique referral. The patient is given my name. If someone wants me to see a patient with a lump in the lung, he usually calls or writes and gives me a recital of the blood counts and the x-rays, etc., and asks me how I am hitting the golfball. The same doctor referring a bypass patient says "See Hornberger. Here's his phone number." At least 50% of my patients have been patient referrals. I have already spoken of Big Joe, the construction worker. He has become a gastric bypass missionary, and there are others like him. Last fall I operated on two early 30's mothers who worked in the same office. Last summer they were on a beach in bathing suits exhibiting their total of 600 lbs, and a total stranger approached them, said she had had the same problem and why didn't they go see Hornberger, and catch the thins. They did, and they have done very well. They have a chart in their office showing weekly weight loss and they discuss it with every customer, and I think most of the fat people in the whole county rotate through there and talk to them. I think patient referral has this significance. The majority are glad to have the operation and indeed are leading dramatically improved lives. Even those who have had problems have lost weight and they recommend it to their large friends. This leads to a fairly steady stream of candidates.

I wrote or scratched out most of this talk during the Christmas holidays. Since that time I have done 16 bypasses in eight weeks with no significant complications and no hospital stays beyond eight or nine days. Having done two cases a week for eight weeks and having another ten or so scheduled for January and February, I began to question where I was going with this business. As I said, it started involuntarily and went along for four or five years, but now it suddenly exploded. At that time I didn't really know what to do about it, and I still don't. My long-suffering surgical assistant has declared that he can't stand more than one a week even though his involvement in the case is seldom more than an hour. My secretary has requested that I refrain from operating on 300 lb females who come to the office wearing shorts. Back around Christmas time, I had done between 40 and 50 consecutive cases with no mortality, no anastomotic leaks, only one major complication, and only
one which threatened to be fatal. If I pick the right 50, I've got a
great record. At that point I figured that I had the touch and decided
the operation was easy. I would like to leave out the next paragraph!

In my last ten cases I have had: 1) a major pulmonary embolus, 2) a
sudden death three hours postoperatively for reasons completely unknown
and not discovered at autopsy, 3) a wound infection, and 4) an anastomotic
leak.

When Dr. Mason asked me to speak on gastric bypass in private practice,
I think he was really asking two questions: 1) should an individual
surgeon not affiliated with a large teaching hospital undertake this
kind of surgery, and 2) is the need for gastric bypass great enough to
justify doing it away from the big league? I think the answer to both
questions is yes with qualifications. There is no reason why a trained
surgeon in private practice away from a medical center shouldn't do this
operation, particularly if he has had experience working in this corner
of the belly, and second, the need is great. Obviously there are vast
hordes of these people and I think many of them will get well only
through surgery. They will try beads, acupuncture, or any other absurdity,
so if you can make this operation safe it can well be done in the boon­
docks.

The real question is, from the surgeon's point of view, is one willing
to put up with the grief. Not just the surgical complications, but the
handling or dodging of fringe problems, medical, psychiatric and fami­
lial, with which one becomes involved. Gastric bypass is not for the
occasional operator, as my early and late series indicate. However,
with the majority of good results, the surgeon who starts will certainly
have an increasing bypass practice and the other surgery that it gener­
ates, and it does generate a lot of surgery. As he becomes more familiar
with the technics the problems will diminish, but as the numbers of
patients increase he is likely to find the bypass business becoming a
relatively high percentage of his total work. I would figure that if I
keep on with this there is too good a chance that in another couple of
years I will be doing nothing else, and I don't really want that.
In conclusion, I would like to present one case which combines many of the problems common in this business. Dolly came from 75 miles upcountry, standing 5' 9" tall and weighing 330 lbs. She had three children. She could never get to sleep until 2:00 a.m. and had to get up at 4:30 to get her husband off to work. She had some kind of inadequate hospital insurance and her husband had a job. This made her relatively deprived because she didn't qualify for Aid to Dependent Children or any other taxpayer's subsidies which would pay me, the hospital or anyone else. She did very well and left the hospital in six days, mostly because she knew some of her own money was being spent. Her surgery was in September. Just before Christmas she appeared in my office 60 lbs lighter, trying to sell me a line of kitchenware for $100. She offered an unbelievable bargain which would spare my wife hours of labor but not actually serve the meals. I was able to resist this and prescribed sleeping pills. A year later, weighing 190 lbs, she got gallstones. Her bypass, when she weighed 330, was uneventful. After cholecystectomy at 190 she had a subphrenic abscess and a prolonged hospitalization. I finally sent her home on an oral antibiotic with instructions to call me if her temperature went over 100. Two weeks later she called and said "Doctor, three nights ago I made love to my husband for the first time in seven weeks, and ever since then my temperature hasn't gone over 97. Doctor, can you tell me what that means?" "Dolly," I explained to her, "that means that you are frigid."
GASTRIC VS. INTESTINAL BYPASS, SEQUENTIAL STUDY
William R. Jewell, M.D.

As an introduction for this talk, I think I can simply say that I share all of the concerns and all of the observations I have heard up to this point in this total symposium. One thing did strike me yesterday as I was sitting here listening and sharing thoughts with the speakers. It was the fact that we didn't know much about morbid obesity and it seemed to be bothering everyone. It bothered us that we didn't know much about the etiology. It bothered us that we didn't know much about the risk of morbid obesity. It will always bother us until we answer those problems. With respect to etiology it upset me a little bit, too. I started to think and I couldn't figure out too many things that I operate on for which I really do understand the etiology. I started with cancer, which is the other two-thirds of what I do. I don't understand the etiology of cancer, and yet we operate for it. It seems to me that most of the time when we figure out what is the matter and what the etiology of these things are that we can stop operating on them. I think probably that is going to happen with obesity, at least I would hope that's what would happen. I am sure that surgery is not the ultimate answer.

For the usual reasons, all given here by Dr. Hornberger and other speakers, we got interested in the concept of surgery for the morbidly obese patient. We have a very active Metabolic Service, headed by Dr. Robert Bollinger. He has been taking care of morbidly obese people for a long time with various sorts of diets which we call, since we are in Kansas, the Jayhawk I, the Jayhawk II, and the Jayhawk III diets. He observed that any patient who weighs more than 100 lbs over their ideal weight simply cannot diet adequately. In the initial part of our program we asked a question that turned out to be the wrong question: "Which of the two contemporary operations for morbid obesity is best, the Scott operation, or the Payne procedure?" We selected our patients in the usual sort of way, rejecting those that seemed to us or others to not be good candidates. Most of our initial patients came from the Metabolic Service. They had been on Jayhawk I, II and III, had failed miserably, and were sent to us. I agree with Dr. Hornberger when he states that patients
not only referred from psychiatrists but those who are referred from doctors are sometimes not the right ones to operate on. Many doctors are wanting to get rid of the obese patient. They are tired of trying to deal with these people and trying to get them to lose weight.

We selected our patients 125 lbs over ideal weight. We didn't find anything endocrinologically wrong. As far as Cushing's disease is concerned, if the patient weighs more than 225 lbs, that's not Cushing's disease. Consequently we are no longer checking for Cushing's disease unless there is hirsutism or something else in the history or physical examination that would indicate there might be something metabolically wrong. Initially, we did pulmonary function studies on everyone, but lately we have stopped doing that. We are still getting serum lipids and cholesterol and doing all the things that are required to operate on these patients.

We were comparing the jejunoileal shunt end-to-end as advocated by Scott and the end-to-side operation advocated by Howard Payne. We set up a randomized study in which patients were evaluated and randomized for one of the two operations. We pulled a card at the time of surgery to decide which operation would be done. We all favored the Payne operation because it was technically easier to do. About midway through the study, as we set about the work of getting our data together for our first presentation of this information, we totalled up all our complications including inadequate weight loss as one of them and we came up with 81 complications. This was fairly striking in view of the fact that we only had 52 patients. We obviously had more than enough complications to go around for all patients.

There were the usual sort of surgical complications. I'm not going to dwell on these this morning because I think you have all read the literature and you have had this hashed about before and are familiar with the things I am talking about. The surgical complications, except for one, I can understand. That one was an acute dilatation of the colon. It was in a man who just dilated his colon, became sick, and we ended up doing a colectomy and almost lost him. I don't understand what happened to him, but the other surgical complications I do understand.
All of us who have done jejunoileal bypass have observed gastrointestinal tract complaints and complications. I think most of these are fairly acceptable. All of the patients had all of these things to some degree. We did not count them as complications unless they became bad enough to require hospitalization. For instance, we didn't count diarrhea unless the patient had to be hospitalized and intensively treated with intravenous fluids. The metabolic complications, however, were, to me at least, terrifying. There were so many things here that were bad, but the really bad part was I didn't understand them. Now I think we have come to understand some of them. The business about the arthritis has been worked out. We had a patient get systemic lupus with a butterfly rash. I don't know if that is a genetic thing, but I don't think so. I think somehow or other it is related to the physiology or pathophysiology of the jejunoileal bypass. It seems to me that when you take patients that you do not understand who have diseases that you do not understand, and then impose upon them an operation that you also do not understand, you are taking a great risk. Three wrongs certainly don't make a right. At this point we became very concerned. Our inadequate weight loss in this group of 52 patients was a total of 15, which I thought was excessive. Maybe we were doing the operation wrong, and we had been told that, and it is possible. We'd be the first to admit that.

We scored our results into excellent, good or poor, just as a very crude way of looking at our information. Essentially we counted an excellent result in a patient who had had adequate sustained weight loss greater than 6 lbs per month. A good result was that same weight loss but with one significant complication, not minor diarrhea or a minor complication but only a major complication. A poor result was a patient who had inadequate weight loss or more than one significant complication. It is a fairly crude scoring system, but it is a way of looking at the data. We looked at our different groups, the end-to-side and the end-to-end, to see what had happened. Although prospectively we didn't plan it this way, what was happening was that individual surgeons (there were three of us doing these; at The University of Kansas: Craig Hardin, Arlo Hermrick and myself) were seeing patients who weren't losing weight adequately and we were shortening the limbs that we were using so that we ended up with different groups. For instance, with the end-to-side
procedure we had groups of 14 and 4, and 10 and 4 inches. Nevertheless, if you look at the total distribution of all operations we had 11 excellent, 11 good and 29 poor results. If you look at the distribution with any of the combinations of how they were done or what the lengths were the curve or the bias in the data seems to be always over toward the poor side. We were very chagrined at this and in a way very surprised that it had worked out this way. We were expecting it to be better. We observed that older patients did worse than the rest. The younger patients didn't do very well either, but the older ones seemed to do very poorly. The best group, certainly, were those that were 30 years of age or younger. In this age group we had 15 good or excellent results and only 6 poor results. If you have to continue to do the operation, it should be in very obese patients where it is technically difficult to do the gastric bypass procedure and it should be done on the very young patients. Older patients just don't hold up well with this operation.

At this point we felt we had to change something. We were very dissatisfied with how the study was going. Perhaps unwisely we decided, rather than to start into the gastric bypass work, (since we had our minds set against the jejunoileal shunt) to just set it aside. Now I realize there are others who disagree with that, and that is fine, but that was our feeling and that is why I am here today, to relate to you how we felt at the time and what we did.

We did start to do the gastric bypass, and since then we have done several .... I am going to tell you about a couple of different series. The data gets a little confusing at this point, but I think you will be able to understand what I am talking about. In total now we have done about 250 among the three surgeons that are involved. We have essentially used the old-fashioned technique with a Billroth II type of gastrojejunostomy. We go into the lesser curve about the same way but we take down the greater curvature to the gastroesophageal junction. I can feel the left gastric artery in only about 75% of the cases, and in the other 25% I just can't seem to be really confident about where it is, so I stay down low enough. We then transect the stomach using the TA-90 stapling device. Our original work was based on using what we estimated to be a 10% pouch. We try to be very careful about saving the upper
branches of the left gastric. We do that by passing a Robinson catheter through the lesser curve side and guiding the TA-90 through, dividing the stomach, and then bringing it down and doing an anastomosis below the colon. We don't do it above. We try to keep the anastomosis site small, and I think that is also important. I want to share with you some data and why I think that is important.

There were 75 patients in our initial group. We have had two B complex vitamin deficiencies. One, a patient who refused to take vitamins even though we hospitalized and force fed them, and another, a patient I don't understand, who vomited quite a bit initially, very quickly got a vitamin deficiency and who got sick and almost died before she recovered. She had a peripheral neuropathy which has now improved and she has done quite well. We had 7 patients in that original group that we felt had an inadequate weight loss. We scored our results in the same way that we had the jejunoileal shunt into excellent, good and poor. We looked at the weight loss in the patients, comparing our gastric bypass series with the jejunoileal shunt series. We feel our data demonstrate that there is about equal weight loss in both groups. Therefore, we felt that, in our hands at least, the gastric bypass would produce equal weight loss to the jejunoileal shunt. We had 34 excellent results, 11 good results and only 9 poor results, which was quite a bit better than the jejunoileal shunt series. When we looked at this versus age it was remarkable, to me at least, that the older patients as well as the younger patients seemed to do quite well with this operation.

We then did one other thing that I want to briefly allude to because it is going to be discussed later as well. We have now taken down 14 of our jejunoileal bypasses for a variety of reasons. Some of these have had a gastric bypass. The reasons for taking the patients down are the usual sorts of things. We felt that these problems were a trial for these patients and intolerable. The patients were tired of them; we were tired of having the patients complaining about these things and also worried about all of these patients so we took them down. In the asynchronous group we took down the jejunoileal bypass. They then gained back weight so we did the gastric bypass and these patients didn't lose weight very well. I think the reason for that was very
simple, we were conservative in doing the gastric bypass because we were concerned that in a patient who had already lost weight the weight loss might be excessive but I don't think it would be. In those patients where we synchronously took down the jejunileal bypass and then did the gastric bypass at the same time, they in fact lost a little bit more weight, although not a lot. The conclusion of this study was that the synchronous procedure is best.

I want to tell you about my personal experience. There were 88 patients when I collected these data about the first of the year. I now have over 100 that I have done personally. The average weight was 277 lbs and the age range was between 22 and 51. There were no deaths in this group of 88 patients. I've been lucky so far and if we keep going we're certainly going to see deaths. As a matter of fact, you'll notice also that there were no leaks in these 88 patients. Just last week we had our first leak. Thanks to Dr. Mason's warning about rapid pulse, we did a gastrografin injection of the stomach, demonstrated a leak, went back, repaired it and he's home now. We did have, unfortunately, a laceration of the diaphragm.

Nine patients had inadequate weight loss out of the 88. They were all females. All male patients lost weight well. I don't know why except that our male patients have been working or want to. Of concern are the patients who have had inadequate weight loss. I count adequate weight loss as averaging 40% of the body weight. That means if someone weighed 250 lbs they have to lose 100 lbs, if they weigh 300 they would have to lose 120, etc. That really is the minimum that we should expect from any operation that we are doing for this problem. All of these operations of which I am speaking now were done prior to June, 1975.

We have revised four because of inadequate weight loss and we did that for cosmetic reasons. We were pressured by the patients and I'm not sure we should have done them. The weight loss in all these patients was 80 or 90 lbs. These first 88 patients have taught us that it is no longer necessary to be overly concerned with respect to the endocrine system. We only are doing a T4 and we still aren't finding anything. I'm not sure we should even do that. We're doing a cholecystogram on
all the patients because we've got the experience of having to do cholecystectomy within 1½ years after surgery. I think that what happened was that we just missed at the time of surgery identifying a bad gallbladder. A lot of these patients have a lot of fat around the gallbladder and I've had trouble evaluating it. I'm not absolutely certain that I haven't missed stones in some of them.

We're also doing psychological tests. We have a clinical psychologist who is very interested in this problem. We use the Minnesota Multiphasic Personality Inventory and other intelligence and personality evaluation tests. We are going to recheck the patients afterwards to see what effect the bypass has had. There have been such studies in the past. I hasten to point out that if you want to look at an area where improvement has been shown in regard to operations performed for morbid obesity it is in this area. The psychologists are convinced that there is benefit. It gets back to the basic problem or idea, that obesity is a behavior disorder. The psychologists will listen to me, and they also talk a language that I understand. Our psychologist will call up and say "that patient's not a suitable candidate and I don't think you ought to operate," and usually I'll agree. If I don't agree, then I do operate. But that seldom happens. Most of the time we agree. I give the patients, the first time I see them, a very detailed description similar to the booklet you have here. It's all about the operation as I see it, and I make the patient and family read it and I go over it with them. After that I'll answer questions for them.

We get an arterial oxygen determination preoperatively. I think that's probably the best single parameter to determine pulmonary function. We use preoperative antibiotics and we've had very few wound infections. We start Keflin the day before surgery, give two doses of 500 mg each, continue it only 24 hours postoperatively, and then stop. We don't do a formal bowel prep using the kind of prep you would for colon carcinoma. We extubate all our patients in the immediate postoperative period. At Kansas we have an extubation protocol which we follow. When patients meet the requirements for extubation we extubate.
It's critical to have the pouch size so we measure it, as Dr. Boyd suggested, by putting a nasogastric tube in place and inserting 60 ml of saline at the time that the TA-90 is in place. If it seems that the 60 ml isn't filling the pouch we set it up a little bit higher. Another thing we insist upon is a stoma size of 12 mm. I don't believe that adequate weight loss will result if the stoma size is very much larger. There is considerable disagreement, however, concerning this point. I know many surgeons prefer a larger anastomosis. It is noteworthy that the gastrojejunostomy done with this small anastomosis almost invariably opens up by the fourth postoperative day.

In conclusion, it appears that the gastric type bypass will produce at least as good a weight loss as the jejunoileal shunt. Complications of gastric bypass are considerably less than jejunoileal shunt. We have demonstrated that jejunoileal shunts can be taken down and a synchronous gastric bypass can be performed safely.
INTESTINAL VS. GASTRIC BYPASS, RANDOMIZED STUDY
Joseph A. Buckwalter, M.D.

This morning I would like to share with you some of the findings that have emerged from our clinical trial, a randomized study of jejunoileal and gastric bypass. We define morbid obesity as existing when patients have been at least two times their normal weight for five years or more. In the two years this study has been in progress we have done 19 jejunoileal and 19 gastric bypasses. All the patients were examined preoperatively by the author, the operations were performed either by the author or by residents with his assistance and have been seen postoperatively by the author.

I was in Iowa City when Dr. Mason began to do gastric bypass about ten years ago. My reaction was quite similar to many of his colleagues. That is, that this was of interest and possibly of some importance but was a kind of surgery none of us wanted anything to do with. About 3 years ago it became clear at Memorial Hospital in Chapel Hill that somebody needed to start doing these operations. More and more requests for this surgery were being received. When approached by Colin Thomas my initial reaction to this was consistent with the one I had ten years ago. Within one year my attitude toward this surgery and these patients completely changed. These are the most interesting and gratifying patients I have ever had the opportunity to operate upon and care for.

All patients had evidence that the obesity was morbid. That is, a patient who has begun to have difficulty in ambulation because of painful ankle joints or knees, who has increasing shortness of breath and other symptoms secondary to obesity and is beginning to decompensate because of overweight.

Diet failure means that the patient has been under adequate medical dietary supervision and usually has attended TOPS or Weight Watchers but has only achieved temporary weight loss. Some of our patients have joined rigidly controlled diet programs such as the Kempner Rice Diet at Duke. I don't know whether or not any of our patients have been to Boston to see George Blackburn. The usual sad story is that even under
these rigidly controlled conditions, they lose the weight but when they leave this expensive and structured environment, they quickly regain it. The morbidly obese patient has usually been rejected by society, by their families, by physicians, and by the time they reach the surgeon they are desperate people.

The third requirement in selection for morbid obesity surgery, is the attitude the patient. This operation is never "sold" at North Carolina Memorial Hospital. The expected morbidity and mortality of the operation and the fact that there is no way to guarantee the loss of the desired amount of weight, is indicated. If the patient responds by saying, "Yes, Doctor, I understand that I have three to four chances in a hundred of dying from the operation, and I understand the risks of wound infection and all the other things which you have explained to me, I want the operation," this is the primary requirement for going ahead with the surgery. The positive attitude is the reason for these patients being so appreciative, cooperative and "fun" to take care of and follow.

In the beginning I thought in terms of excluding patients with serious heart disease and other serious organic problems which would increase the morbidity and mortality of the operation. The lowest operative mortality and best weight loss occurs in younger healthy patients. My thinking has changed. I suggest that the patients who may need the operation most are older patients who may have serious organic disease, heart, renal and other. Which patients do we exclude? If the patient has a positive attitude, meets the other requirements and has an 80% chance of surviving the operation, it is done.

About half of our patients are white, half black. It is my impression our black patients have done as well as the whites. The important point is their understanding of the operation risks and expectations. During the initial interview with the patient I show them diagrams of jejunoileal and gastric bypass and explain the weight loss mechanisms in words they can understand. I point out that diarrhea must occur with jejunoileal bypass if they are to lose weight. I tell them they will have difficulty eating after gastric bypass. They must understand that with gastric bypass, if they compulsively try to eat after the operation as they did
before, they will have nausea and vomiting. The patient is admitted to
the Clinical Research Unit four days before the operation for appropriate
biochemical, x-ray and other studies.

I will tell you about a case unusual in our experience. The patient was
a 28 year old, 275 lb, 5'1" woman beginning to have symptoms secondary
to her obesity. These symptoms took the form of painful ankles which
limited her walking, and shortness of breath. She had a history of
menstrual irregularity. It is not our custom to routinely obtain skull
films. However, they were taken and showed a double-floored pituitary
sella, suggesting the possibility of a pituitary tumor.

Prior to admission to the Clinical Research Unit, patients are randomized
by computer for jejunoileal or gastric bypass. Patients are randomized
into 8 groups by sex, age (above and below 35 years) and weight (women
above and below 285 lbs, men 325 lbs). We perform the original gastric
bypass described by Dr. Mason. The jejunoileal bypass is that described
by Dr. Scott.

In the first 39 patients operated upon, there was 1 death. The patient
was discharged after an uncomplicated postoperative course on the 8th
postoperative day following gastric bypass. She was readmitted 2 weeks
later with a popliteal embolus. Following removal, she developed a
second one in the brachial artery. While this was being removed she had
a massive pulmonary embolus. We do not heparinize patients postopera­
tively. Perhaps low-dose heparin may be in order.

We have had, in the 39 patients, 3 significant wound infections, 2 with
gastric and 1 with jejunoileal bypass. Patients are placed on tetracy­
cline and neomycin 48 hour bowel preparation. One patient has had
severe alkaline gastritis after gastric bypass. She was placed on
Maalox and cholestyramine. Two weeks before we were planning to reop­
erate and convert her to a Roux-en-Y, she improved. During the 2 years
we have been doing bypasses, we have seen none of the really serious
complications of the jejunoileal bypass. Some of our patients have lost
some hair. One patient had a classic picture of bypass enteritis which
responded to ampicillin. None of the patients have had evidence of liver failure. One patient has had urinary tract stones.

Some have reported inadequate weight loss in up to 30% of patients following jejunoileal bypass. One patient not included in the clinical trial and operated upon 2 years before coming to Chapel Hill, lost only 40 lbs. We reoperated upon her. She had 12 inches of jejunum and 15 inches of ileum. We removed an additional 3 inches of both. Again the patient did not have significant diarrhea and lost only 20 lbs during the 6 months following operation. One of our 19 patients has had minimal diarrhea with disappointing weight loss after jejunoileal bypass. No satisfactory explanation for this unpredictability of response to jejunoileal bypass has been provided.

As of March 1977, we had 18 patients, 9 that have been followed for 4-6 months and 9 followed for a year. All that can be said is that postoperative weight loss is comparable with jejunoileal and gastric bypass.

More impressive is our anecdotal experience. Individual patients have had spectacular results with both operations. A 35 year old, 325 lb truck driver lost 150 lbs in the first year after gastric bypass, enough to convert her into such an attractive female that her husband no longer would permit her to drive a truck! She regards this as a miracle. She is convinced that something magical happened in Memorial Hospital. Another 350 lb woman, 38 years old, 5'3" tall, had very severe gout. She was on allopurinol, took colchicine for acute exacerbations, was hypertensive and diabetic. This patient in 1 year after jejunoileal bypass lost 140 lbs. She has been gout-free with no exacerbations to everyone's surprise! We were concerned about the impact of this iatrogenic kwashiorkor on her gout. Her blood pressure has returned to normal and her diabetes has improved. She also had a sudden weight loss of 20 lbs at 1 year; a panniculectomy.

The most important criterion is the attitude of the patient. This applies equally to the usual patient with an expected mortality rate of 2 to 3% as well as the poorer risk patient. An example of a high risk patient was a 520 lb, 28 year old woman who had had 3 myocardial infarcts,
and terrible blood gas studies. She was admitted and scheduled for operation. She left the hospital the night before the scheduled operation because she was aware that there was a good possibility she might die on the operating table. I had told her there was 1 chance in 5. Three months later she had a jejunoileal bypass. She left the hospital 7 days later. She has lost 200 lbs in a year. Her life has been transformed. Unfortunately such a happy story does not always unfold in this type of patient. A heavier patient - no way to weigh her - was admitted to the Respiratory Intensive Care Unit. The medical people felt the only chance was an obesity operation. We scheduled her for panniculectomy. If the wound healed and she survived, a bypass would be done later. She died before the panniculectomy could be done.

Which is the better operation? Although we have not as yet observed serious morbidity with jejunoileal bypass, the variable results in terms of weight loss must be considered an argument for gastric bypass. It is obvious we all need to consider revising the Mason gastric bypass technique in view of the experience of Alden and others using the nontransection staple procedure.

What about the edentulous patient? Is this patient a candidate for jejunoileal bypass instead of gastric bypass? Are there other types of patients who still may be candidates for jejunoileal rather than gastric bypass?

It has been for me a privilege and valuable experience to attend this meeting. I take this opportunity to express a tribute to my long-time friend and esteemed colleague, Ed Mason. I am aware of the many hours and days he spent in the laboratory and operating room during the inception of the concept of gastric bypass. It was his commitment, imagination and expertise which resulted in this meeting. As we all proceed to do gastric bypasses we have reason to be grateful for his effort. Thank you, Ed, for what you have done for all of us and countless numbers of patients.
INTESTINAL VS. GASTRIC BYPASS, RANDOMIZED STUDY
Ward O. Griffen, M.D.

Before I get started in presenting my material on the randomized study, I would like to make some basic remarks. I, too, agree with much of what has been said over the past day and a half, and I am sure I will agree with much of what will follow. I would like to say, first of all, that this series I am going to present is a personal series so I will be using the word "I." I really object to the word "we." Often in the residents' conferences, when they start talking about "we did this" and "we did that," I remind them that Mark Twain said that "we" is a word which should be reserved for newspaper editors, crowned heads of state and people with tapeworm. So I will be using the first person very often because this is a personal series. Although fully 90% of the patients that I am going to present were operated on by the residents, that is, the residents were wielding the knife, I was there throughout the procedure and I feel obligated to be there.

Also I was impressed with the simplicity with which gastric bypass can now be done. Nevertheless, I have some ambivalence about the fact that it has been made so simple. Obesity is an area where there are many charlatans and I think we all need to recognize that. There is a man down in Georgia who has done 1,000 jejunoileal bypasses in 18 months. He must be taking all comers. I know that at least three patients he operated on for middle age spread were only about 20 lbs overweight. Now I think that the jejunoileal bypass is an unsafe operation, as you will see as my story unfolds, but the gastric bypass is not an unsafe operation if it is done technically correctly. It worries me that we are going to see a lot of patients undergoing gastric bypass who are 10, 15 or 20 lbs overweight, and that makes me a bit nervous. Nevertheless, I think that it is good to simplify operations. I am a great believer in having simple operations. I personally can't stand much longer than about six hours. I get tired. Therefore, if any operation takes longer than six hours it makes me very nervous. We still do the standard type of operations, as you'll see.
The other point I would like to emphasize is that once you start, once you embark on this course, you will get flooded with patients that by and large are referrals from other patients. I, too, like to avoid the ones the psychiatrists send me. I think the psychiatrists have little role to play in this entire endeavor. Perhaps the approach that Bill Jewell uses with the clinical psychologists is a good one, but for the most part psychiatrists are sending you patients who have been total failures. I also feel very strongly that patients can help each other. We have formed a Fatty Club just the way that we have Ostomy Clubs, The Lost Chord, the Reach for Recovery and all the other things that are done along those lines. This is helpful because these patients can tell each other much better than I what the various complications are and how the new patients are going to feel. They also can help you, by the way, in getting your randomized studies going. The minute you tell a patient you are going to do an operation based on the flip of a coin or the fact that their hospital number is even or odd (which is the way we randomize our series) or any other kind of randomization procedure, at least the intelligent ones seem to shy away from joining with you in that particular effort. The clubs can be helpful here in adding encouragement and enthusiasm. Therefore, I think all these things are terribly important and are items that must be followed if you are going to obtain any kind of scientific information from it.

I also feel, as George Blackburn was trying to point out yesterday, that we do need to have science in this area. Doctors are now becoming the public scapegoat for everything, from social injustice all the way down the line. Unless we conduct ourselves with at least some sort of scientific evaluation we are open to criticism and should be criticized. That is why we should guard against the charlatans who get into this business. I would encourage everyone not only to read the book by Dr. Bray, which is an excellent book, but if you want to get a shorter reading, you can read the two-part article that appeared in July and August, 1974, by Gordon Mann about the whole obesity problem. Mann points out that obesity per se does not necessarily shorten the patient's life. If it is complicated, as it very often is, by diabetes, hypertension, cardiac disease, pulmonary disease, etc., it will shorten the patient's life span, but obesity per se does not. In that article he
also points out the fact that diet therapy in obesity can be stated quite succinctly, it doesn't work. The reason it doesn't work is because these people do diet and lose weight but then they put it all back on again. It becomes a recurrent cycle until the point where they come to you for some kind of help. Mann further adresses the problem of the quacks who get into this business and is very concerned about the fact that there are a lot of quacks in the business of obesity. That's why there are so many books currently available on various diets that in paperback editions have sold millions.

There is another reason. I tell the students quite often that there must be a reason why the chiropractors still stay around. The reason is because the chiropractors sit down and talk to their patients; something many doctors won't do. If we would or could spend more time talking to our patients, convincing a patient, for example, that the only thing he needs to do is stop eating so much, stop drinking so much, stop smoking so much and get a little exercise, we probably would have a healthier nation. We would probably have a public that was happier with the medical profession. Unfortunately I don't think that's going to happen. Therefore, we don't have an ideal situation. I would agree that obese people who meet the criteria, who come to you having almost invariably been overweight all their lives, having been on 10, 15 or 20 diets and having a positive attitude should be considered for this procedure.

I am sure you are all well aware of the rationale of these two procedures. Basically the gastric bypass procedure is a form of enforced and permanent starvation, that is, if the operation is done correctly. Jejunoileal bypass, on the other hand, is controlled malabsorption. That is to say, it is hopefully controlled, but sometimes it is uncontrolled and that is when you get into trouble. In 1973 I started doing patients with morbid obesity. As many other speakers ahead of me have indicated, the minute that you start doing this operation you begin to see the fat people crawling out of the woodwork. It does involve an exponential curve. You go through a learning process, get somewhat disturbed and back off from doing them. Then you take it up again because some friend of yours wants you to do one. The patient has a spectacular result so you get a little more enthusiastic and pretty soon it's exponential.
I am going to talk today about the experience that we have had since January, 1974. When I began, I had known about Dr. Mason's procedure. I had also heard of the criticisms or comments that had been made when he first presented the material, indicating they were concerned about stomal ulcer, the fact that it was a technically difficult operation, and the hundred other criticisms that were made of it. It's very easy to criticize, as I'm sure you're all aware. I did only the jejunoileal bypasses in the first 30 patients or so, but then I said, well, as long as I'm going to start doing this, I might as well do it the scientific way. Therefore, we started a randomized study in January, 1974. By January, 1977, we had 32 patients in the gastric bypass group and 27 in the jejunoileal bypass group with three years of followup. I made an arbitrary decision that I was going to get 50 in each group and I can report that as of this time I already have 50 in each group. If it is any consolation to those of you who are protagonists of the gastric bypass, I can report that from now on we are only going to do the gastric bypass in my institution.

The operation that I used for the jejunoileal bypass procedure was the operation that had been described by Scott except for the fact that I used 30 cm of jejunum, 12 inches, and 25 cm of ileum, 10 inches, with an end-to-end anastomosis. I have drained all of the bypassed segments into the sigmoid colon and I recognize all the arguments pro and con as to where you should drain. I personally don't think it makes very much difference, it just seemed easiest to put it there. I have not been terribly unhappy with the operation from the standpoint of getting pneumocystis or from a lot of gas in the bypassed segment. I really don't think that's been a major problem with the procedure.

The first seven patients that we did in the gastric bypass group were done with a retrocolic loop gastroenterostomy. I was impressed by the fact that the patients would overindulge and vomit. We finally learned how to control it. But I also had two patients who had vomiting inadigestively. One patient in particular used to wake up at two o'clock in the morning and vomit pure bile. She also had a great deal of heartburn. I finally did a fundoplication on that patient even though she had had a gastric bypass about a year earlier. Fortunately, it worked.
She got rid of her heartburn, although she hasn't gotten rid of all her other problems. Nevertheless, I decided that I was having too much problem with the bilious vomiting. Therefore, I went to the Roux-en-Y technic with a retrocolic type of anastomosis. One of the nice things about it is that you don't have to drag that gastric pouch down below the transverse mesocolon. You can just tack that loop of jejunum right up to the mesocolon where it lies. I have had no problem with this in terms of either alkaline gastritis or stomal ulcers.

I've been doing this particular procedure since the fall of 1974, and I think it is a perfectly safe operation. The reason it is safe is twofold: 1) there aren't too many parietal cells in the fundic pouch and 2) the gastric remnant or the bypassed segment does produce acid so that the gastrin mechanism is shut off effectively after the patient eats. Consequently, there isn't any hypersecretion from that little 5% pouch even if there should be enough parietal cells. I did not, until recently, start measuring the size of the pouch but I do think it is a good idea to measure the volume that the pouch will accept along with the pressure measurement, as Dr. Terry has indicated.

There are two other points that I would like to make before going on. One is the fact that we are basically in trouble because of the lay concept of instant repair. Everybody in this country believes they can over-indulge in whatever they wish and then they come to the body shop known as a hospital and we mechanics put it back together again so that they can be perfectly normal. As Harry Schwartzmann once said, "The medical profession will always be in trouble because what the American public wants is immediate cure at low cost to old age with full sexual prowess," and that's an impossibility. The other point I would like to address concerns the various things we do surgically around the operation: 1) The use of parenteral antibiotics. Previously, I had used them only in the jejunoileal bypass patient and then because we opened up the colon, but because of the high incidence of infection in the gastric bypass group, I started using the antibiotics with them also. I do use oral antibiotics on the patients undergoing jejunoileal bypass procedures but we do not use oral antibiotics on the patients undergoing the gastric bypass. I use parenteral antibiotics the night before and the day of
surgery and then stop them. 2) The use of mini-dose heparin. I do use low-dose heparin on all these patients. We have a regular protocol that takes about five working days to complete. Therefore, the patients are in the hospital for a week prior to their operation and as soon as they are admitted they are put on low-dose heparin. Although in non-obese patients, I use low-dose heparin at a rate of 5,000 units every 12 hours, in these patients, because of their size, I use 5,000 units every 8 hours. The patients do not bleed excessively. I really have not had any problems with low-dose heparin in my total experience except for two patients on whom I did a Whipple procedure. Interestingly enough when we discovered that they were bleeding intra-abdominally postoperatively their coagulogram showed obvious total heparinization even though they were on low-dose heparin. Aside from this, I have been rather pleased with low-dose heparin. I think it is something that we need to look at very critically. I am sure many of you are well aware, there are now at least three lawsuits that are on the basis of pulmonary embolus and one death postoperatively due to a pulmonary embolus where low-dose heparin was not used. 3) A third feature of it is that we do concomitant or simultaneous procedures with the bypass operation, taking out the gallbladder, tying tubes, taking down the ovaries, clipping the vena cava, etc. I have clipped the vena cava in three patients who had documented pulmonary embolus prior to having the operation for morbid obesity, and again have had no particular problem with that. Naturally, I too, stress the early ambulation and respiratory support. Extubation is carried out as soon as the patients meet the criteria of their respiratory mechanics.

The criteria for the inclusion in this study is that they be more than 50 kg over their ideal weight. We do a whole battery of endocrine studies but like Bill Jewell I suspect we are going to stop doing all that material of the patients once we stop doing the randomized series because we haven't found anything positive in them. We do a whole host of absorption tests and we have not found absorptive deficits, as you might expect we would, but we don't find anybody with supervalues either. They just overeat, it isn't that their bowel does such an excellent job.
I prefer to have patients who have concomitant conditions, diabetes, hypertension, hyperlipidemia or whatever, because then I feel as if I'm doing something more than cosmetic operations. Nevertheless, I have a number of patients who have not had anything more than their morbid obesity. I still get psychiatric evaluations. However, I just received a memorandum before I left from the Psychiatric Department saying that their Consultation Service will no longer see the patients. As a matter of fact, I feel that those of us who do this operation are really the patient's psychiatrist anyway. As many of you know the patients will call you up about everything, their alcoholic husband or their kid who is on drugs or the fact that they can't get it up or get it down or whatever.

Of course, the patient has to be willing to participate in the protocol. We have got to have informed consent. We go through the whole rigmarole with a written document, the first statement of which is in capital letters, "THIS OPERATION MAY KILL YOU," because I think the patients ought to understand that this isn't a picnic that they're going on, it's a real risk.

We have had an interesting distribution in terms of males in that we have had quite a number of males. However, when the gastric group is broken up for some reason there were many more females. This explains why the average preoperative weight and height are a little bit lower in the gastric bypass group than in the jejunoileal. Nevertheless, they are all sizable candidates. The smallest patient I've operated on weighed 223 lbs, that's a 110 kg woman. She was 4'9" tall, so I thought she fit the criteria. She was just a little butterball instead of a big butterball. I have done some patients over the age of 50. The oldest patient I've done actually was 62. She was a severe diabetic and had had two myocardial infarctions and had hypertension. I really felt as if she needed the operation as a therapeutic measure more than anything else. I was gratified to find that she did very well.

In regard to the concomitant conditions many of them had had hypertension. This has been hypertension that has been documented by arterial lines, on the operating table, and it is true arterial hypertension. Many of
them have been pickwickian and several have had cardiac disease. Many have also been diabetics. By diabetes I mean patients who either have overt diabetes, either taking pills or insulin, or patients whose glucose tolerance curve is abnormal when they come into the hospital. Several of the patients have had hyperlipidemia.

Now we can look at the complications. There have been no operative deaths. In fact, in the somewhat more than 120 procedures that I have done since 1973 I have only lost one patient. She died from a pulmonary embolus on her 15th postoperative day. That patient, by the way, had an error in the medication in that the low-dose heparin was stopped on her eighth postoperative day and none of us picked it up.

There have been two late deaths that I think are very interesting. One late death in the jejunoileal group was in a man who lost 250 lbs in ten months. First of all, he came back every six months and I kept yapping at him about the fact that he was not eating properly because he was losing more than 15 lbs a month. I think 15 is an outside limit, I prefer having them lose only about 10 lbs a month. I tried to explain to him, as I do to all of the patients, that they must keep their nutrition up and their oral intake must be adequate. This is particularly true in the jejunoileal group because if you add, on top of controlled malabsorption, an exogenous nutritional deficit the patient surely will get into liver trouble, and he did. At ten months he absolutely refused to let me put it back together again, even though it was obvious to me and everybody else that he was not faring well. At 12 months he didn't keep his appointment at the clinic and I called to find out that he had been admitted to another hospital jaundiced and in renal failure and kidney failure. He did, in fact, die just one year after he had had the operation.

The patient who died in the gastric group represents one of the vitamin deficiencies to which Dr. Jewell has already alluded. This was a patient who was three months past her gastric bypass operation. She came to the clinic and just said she wasn't feeling very well. She didn't have any specific complaints. She still had a bit of vomiting when she overate. She did not have any diarrhea. She was losing weight satisfactorily, in
fact, in three months she lost about 56 lbs. But we went ahead and admitted her and the only thing abnormal we could find was that her serum potassium on admission was 2.9. We treated her with glucose-containing materials and high amino acids and she seemed to straighten out and was eating on the third hospital day. She suddenly collapsed on the bathroom floor and when the nurses found her she was dead. We did get an autopsy, thinking that surely she had had a pulmonary embolus. However, she did not have a pulmonary embolus and I suspect that this represents a vitamin deficiency that we perhaps just kicked over the rest of the way by giving her glucose-containing fluids when she first came in.

The rehospitalization rate is interesting. Four patients have been rehospitalized from the gastric group. Two of the patients are ones who had leaks and we will come to that later. There was one other patient in addition to the one who died. The ten patients who were rehospitalized in the jejunoileal group represent a variety of things. One of the patients was rehospitalized about seven times for hypokalemia in the 18 months that she had had the jejunoileal bypass in place. Many of the other rehospitalizations in the jejunoileal group are patients who had undergone subsequent cholecystectomy. I personally believe that the jejunoileal bypass is a lithogenic operation and that almost 100% of these patients will eventually develop gallstones. One patient in the gastric group has had a reanastomosis and that is one of the patients who leaked. She did not require a second operation as the leak was not a major leak, but she developed so much fibrosis around the anastomosis that it eventually got down to a measurable 2 mm by endoscopy. Although she was happy because she went from 412 lbs down to a svelte 119 lbs, I wasn't particularly happy because she vomited about two or three times a day. Therefore, we took her down and interestingly enough she is back up to 300 lbs, so we're probably going to have to go back in and do something. There was one jejunoileal bypass that we took down in a patient who had a combination of electrolyte imbalance and beginning liver failure. I convinced her that she ought to be redone and she accepted this. We've had our complications and I think it's important to recognize that this procedure is not without complications. Nevertheless, in the last 25 patients we have only had one wound infection and
that was a jejunoileal bypass patient. Wound infection in these patients is disastrous. Ever since we put the gastric group on antibiotics, we've only had one infection. We do, by the way, transverse incisions in these patients, both the gastric and the jejunoileal group. I believe you can get perfectly adequate visualization with a transverse incision. I don't have the four corner retractor that Dr. Gomez has, but that looks like it would be interesting because then maybe I wouldn't get so tired.

We have had dehiscence in each group. We have had two anastomotic leaks. In the second anastomotic leak the patient did have to be reoperated upon. The rapid pulse business is true, it does work and it gives you an idea that something is wrong. Unfortunately, we weren't smart enough to tumble to the fact and we actually didn't get back in until five days down the pike. This was in a patient who had a Roux-Y anastomosis. One of the nice things about the Roux-Y anastomosis is that the leak doesn't probably present as much of a problem as it does when you've got a loop. This is because you don't have the bile and pancreatic juice coming out, you simply have saliva and gastric juice. In this patient we were able to close the leak and put a gastrostomy tube into the bypassed segment and actually started feeding that patient by her own GI tract on the third post closure day of the leak and she did very well.

We have had a couple of subphrenic abscesses in the gastric group and one in the jejunoileal. Urinary tract infection has been a significant problem and interestingly enough all of them have been females. We have had no pulmonary embolus in either group that we have recognized. They may have been there but we certainly haven't recognized that, that is, in the early stage. We have had some in the late stage and I'll get to that in a minute. The three other complications in the gastric group are splenectomies. That is a part of this operation and must be recognized as such.

In regards to the late surgical complications, nausea and vomiting is an integral part of the gastric bypass, and I think the patient should know that. Basically what we are doing is helping them in their behavioral
training. We are teaching them that they just can't overeat. Diarrhea, of course, is a major complication of the jejunoileal bypass. Fifteen of our patients had severe diarrhea. We do the same as Bill Jewell. We expect diarrhea in all of these patients, we only call it a complication when they have more than six bowel movements a day and require medication.

There are 20 patients on medication in the jejunoileal bypass group. This is one of the reasons why I think this operation will probably go by the wayside. Even though you can get the diarrhea under control, it often requires a very significant amount and type of medication. Also I think that that is perhaps another indication for gastric bypass. Although I don't like the gastric bypass operation necessarily in people who aren't too smart and don't understand what we are trying to do, let me tell you that the jejunoileal bypass in a dumb person is a disaster. They simply don't understand the disease process. They also don't understand the medication needs. If they really are going to end up having to take a lot of medication, lack of intelligence is really a contraindication for doing the jejunoileal bypass.

As far as kidney stones are concerned, it is interesting that we have not seen a single kidney stone in a gastric bypass patient. We have seen it in 4 of 27 in the jejunoileal bypass patients. I think that is because the jejunoileal bypass group does represent a metabolic disorder that you have created on top of a metabolic disorder that already existed. In addition they are losing weight and can go into periods of relative dehydration, depending upon how much diarrhea they have. On the other hand the gastric bypass patients, even though they may not be able to take in solids without knowing about it, seem to be able to handle liquids fairly well. Consequently they keep their water balance up, their urine flow high and therefore do not get the concentrated urine that produces kidney stones.

We have had some late pulmonary emboli. These have all occurred in patients who have gone home and languished in the bed. Therefore, we do try and get them into vigorous ambulation exercise programs as soon as possible. In fact, we start it the day after their operation or on the night of their operation, and continue running them for the rest of
their lives, which I think is terribly important. We have done 10 reoperations for various reasons such as cholecystectomy and hernia repair.

We have taken some liver specimens intraoperatively and then later at one year postoperatively. In the jejunoileal bypass group the specimens look even worse at one year. If Dr. Drennick were in the audience he would clearly point out that at one year they look terrible while at two years they begin to come around to be halfway decent. On the other hand, in the gastric bypass group the liver is really getting to be pretty normal at one year postoperatively. We haven't seen the liver problems in the gastric bypass group that are seen in the patients with jejunoileal bypass.

In regard to weight loss, there are 18 gastrics and 22 jejunoileal bypass patients in this series that have had more than a year of followup. The gastric bypass group had a mean weight loss of 51 kg at one year, while the jejunoileal group had a mean loss of 57.9 kg. This is not significantly different. These patients do lose weight at the same rate, as Dr. Jewell indicated. In summary I can say that gastric bypass is technically more difficult although with the new modifications that statement may have to be erased. I do think that their anastomotic leak will still be at a somewhat higher rate than the jejunoileal group because of the fact that you are using the stomach as part of the anastomosis. On the other hand, the list of disadvantages of the jejunoileal bypass is tremendous—malabsorption, medication and the rehospitalization rates are extremely high. I again say that I personally believe that the jejunoileal bypass operation is lithogenic and all these patients will eventually develop cholelithiasis. And, of course, liver disease is there as a specter of death continually. In conclusion I would say that all patients operated upon for morbid obesity require close supervision postoperatively. That is particularly true of the jejunoileal group, and considering all factors, the gastric bypass is a more satisfactory operation for morbid obesity.
HIATUS HERNIA, REVISIONS AND RELATED PROBLEMS
Edward E. Mason, M.D.

The problem of hiatus hernia and reflux is due to certain incompatibilities of gastric bypass and reflux operations. There are competing objectives. The antireflux operations, Nissen fundoplication, Hill, Belsey, and so forth, have to do with wrapping the esophagus and forming a valve. This requires some redundancy of the stomach. The objective of gastric bypass is to make the upper segment as small as possible, to take down the fundus, to get rid of the angle of His, to do things which seemingly would be productive of reflux. Certainly it does not leave a redundant pouch to wrap around the esophagus.

Another complication exists in that a successful antireflux operation by definition prevents vomiting and belching. A successful gastric bypass with a gastroenterostomy requires a small pouch and a small stoma which slows up emptying. The obvious potential hazard here is a closed segment. The area of the lesser curvature with the upper branches of the left gastric artery must be preserved. If the stomach is transected this is the blood supply to that upper segment. If you interfere with that blood supply the upper segment will undergo necrosis. I am very concerned about some of the pictures that were shown about the technique yesterday. I wonder if they truly depict what the surgeons are doing. I am worried about devascularizing that upper segment. It may be that if the stomach is stapled in continuity there is enough blood flow through the staples to feed the upper segment even after its complete devascularization. However, I wouldn't depend on it and I don't think in that regard that you need to go that high on the lesser curvature. I think maybe the speakers yesterday are leaving a little bit more than those illustrations suggested.

To get back to the competing objectives in gastric bypass, you are trying to leave blood supply on the upper aspect of the lesser curvature. At the same time the antireflux procedure requires pulling down on bites of tissue from the sling of Helvitius to recreate the angle of His. The fundoplication is around the esophagus. It should not be around the lesser curvature but nevertheless you are working in this area and there
cholesterol coming into the duodenum per hour over a 24 hour basis. However, and this is the point I would like to have you focus on, the obese subjects secrete at least three times more cholesterol per hour on a zero cholesterol diet than normal patients and two times more than even patients with gallstones. In contradistinction, their bile acid and phospholipid output do not change appreciably.

Looking at this in a different way, we took these patients with various weights and plotted their cholesterol output with their weight. In males there is a linear relationship between the amount of exogenous obesity and the increase in cholesterol elaborated into the bile. If one looks, then, at the bile acids to find out if this is the problem, one sees that the gallstone patients had somewhat smaller bile acid output than normal subjects. However, the obese patients have a bile acid output which is slightly greater than normals. I am trying to make the point that the error is not in bile acid metabolism as may be partially the problem in the patient with gallstones. Finally if one looks at these obese patients when a zero cholesterol and a 750 mg cholesterol diet are fed, the obese individual secretes more cholesterol with a zero cholesterol diet than do normals fed a 750 mg cholesterol diet. Adding 750 mg of cholesterol to the diet of the obese results in an even greater excretion of cholesterol in bile, and this increase is greater than the increase observed in normal volunteers.

Dr. Scott Grundy of San Diego has studied obese patients after weight reduction. In his studies the obese patients, before weight reduction, secreted bile which was invariably supersaturated with cholesterol. After weight reduction down to 138% of ideal weight, these same abnormal patients now have a cholesterol concentration in bile which is well below the lithogenic range. In other words, they are perfectly capable of maintaining all the cholesterol in solution and are essentially at no risk for cholelithiasis. So we have concluded from this series of studies that biliary cholesterol secretion is influenced by metabolic factors, namely cholesterol ingestion and a hypercaloric state. Weight reduction by gastric bypass or diet restriction will decrease the risk of gallstone formation in these obese subjects.
QUESTION: Do the postoperative gastric bypass patients have less cholelithiasis than normal obese patients who have not had any surgery?

DR. DENBESTEN: I have not done the clinical studies on this. Mine are laboratory studies. Dr. Mason, I think, told you this morning that in the postop period during weight reduction, patients are at increased risk to develop gallstones. I have not studied this aspect of the problem. Perhaps we will one day use chenodeoxycholic acid to protect these patients during the period of acute weight loss.

QUESTION: I thought bile was lithogenic during the period of weight reduction following this type of surgery.

DR. DENBESTEN: You are correct. When any human is 'starved' his bile becomes more lithogenic; there will be more chance of stone formation during the period that he is not eating. The mechanism explaining this observation is well known. Cholesterol is secreted by the hepatocyte by two mechanisms: a constant (bile salt independent) and an augmented (bile salt dependent). If they are not eating, bile salt isn't coming back. Therefore, all the cholesterol is being excreted at a fixed rate with little or no bile acid secretion with the result that bile will be more saturated. The importance of the studies I have tried to present is that in a 24 hour period there is a marked difference in the total time lithogenic bile is secreted. Everyone of us have lithogenic bile for at least one to two hours every day. That's not the problem. The fact is that the fat patient has lithogenic bile 24 hours a day and that's where they seem to get in trouble. The momentary lithogenic bile is not a problem to humans.

DR. GOMEZ: I was beginning to think that there may be some incidence of increased cholelithiasis because I have had five patients that required cholecystectomy within the first year after gastric bypass whose gallbladders were perfectly normal prior to gastric bypass. Two of these were a sister and brother, and of the other three two had acute cholecystitis with obstruction of the cystic artery.
DR. DENBESTEN: As you know, all of us vary in the kind of bile we secrete. Some of you are secreting bile which is right on the border of supersaturation most of the day, some are lower. Some of these obese patients, even at ideal weight, will be very close to being saturated, but the incidence after the period of increased risk during weight loss should be that of the normal population.

DR. DRENNICK: Why do obese individuals increase production of cholesterol when they have so much cholesterol? What happens to the feedback controls?

DR. DENBESTEN: I can't explain it, but I think if you come back next year perhaps we will be able to. Studies are being done using tissue cultured hepatocytes from prairie dogs. There is suggestive evidence that the hepatocytes, from the prairie dog who was fat, continue to elaborate or synthesize a far greater amount of cholesterol than those hepatocytes from animals who were thin when hepatocytes were harvested. These are preliminary data and much more work remains to be done.
DR. FREEMAN: We have a series of what we think are eight salient points dealing with body composition in the obese patient. Any semblance between an orderly conduct of these eight questions is purely coincidental! I am going to act as a protagonist and present our visiting professor with each of these points. So without further ado, here's the first question for Dr. Blackburn. George, a 300 lb, 5' 6" female, 28 years of age, comes to your office and states that she has been overweight since the age of 18. She has been in TOPS, Weight Watchers, and has lost 50 lbs. four different times in her life and gained it back each time. She is 28 years old, can't get married, wants to know what the proper treatment is, and she hears you are a great surgeon.

DR. BLACKBURN: First of all, I congratulate her on her great choice. Most of the patients that come to us have been sent by another patient. Not until they have entered our office has any referee interacted. This is a free enterprise system and the patient is age 28, wants to get married, and weighs 300 lbs. Our first responsibility to this type of patient is to give them information about exactly what it is they are coming to us for, because we cannot assume, and you should not, that the patient is in a position to make the right decision. Right away we have to point out to them that it is not up to them to decide the surgery, that they cannot know the risks and benefits of such surgery, and therefore a variety of information will be necessary beyond what has already been given. I would start off by pointing out that we do not know what benefit can come to the patient because even though at 28, she does weigh 300 lbs, we don't know what medical harm, if any, will result from this degree of overweight. I haven't been told whether the patient is hypertensive, but I doubt that she would be, or whether she is diabetic to the extent of being symptomatic. She may have an abnormal glucose tolerance test, but I seriously doubt that she has angiopathy or nephropathy or any other sequelae of renal disease and the like. Therefore, I suggest that we do not know the benefit or the risk of being that obese. We have already heard of some males who have some problems when
they are over 40. Nevertheless, this woman is only 28. Therefore, to schedule her for operation immediately is out of the question. We have obviously been very interested in comprehensive approaches. My last point here is that the patient has only tried TOPS and Weight Watchers which have a single philosophy. She may also have been on drugs; again a single entity therapy. We now are learning that some comprehensive approach that involves nutritional education, behavior modification, exercise and relaxation therapy combined with certain dietary approaches are effective. Therefore, we need a lot of workup to find the answers.

DR. FREEMAN: If there were one answer to the first two minutes of this session, it would have to be that even though the primary indication for us to operate on patients is their inability to lose weight, this inability per se, is not an indication to go ahead with operation.

DR. BLACKBURN: I think that's clearly correct. To get started I would emphasize that it's a partnership. Our hypothetical patient has lost 50 lbs before and I would get her started losing again while we get to know each other and learn the risks.

DR. FREEMAN: The second question is: "Should we tell an overweight patient to lose weight before bypass surgery or, for that matter, before any sort of an operation?"

DR. BLACKBURN: At this point, I would draw her some pictures that explain her body. I would point out that the survival part is the body cell mass containing skeletal muscle and organs, and that her obesity is like a tumor covering that survival part. The key tissue to hold on to is the protein containing muscle mass, and that is the only reason we talk about nitrogen balance. I would explain to her a little bit about metabolism as Dr. Francis Moore might have done. What you want to save is the body cell mass; the living, functioning, energy-saving and mitotically-active cells of the body. If you are going to submit her to surgery or any therapy that is going to cause loss of that, then you've got a lot of problems. This, in effect, is why we are here today; because one surgical technique clearly has been demonstrated and reported by its investigators to have over half of the weight loss as body cell mass :=
the first five months. Then again potassium and phosphate losses are problems. Body cell mass consumes oxygen and produces CO$_2$. Whether you do surgery or not, the only way you are going to get rid of that fat 'tumor' is to burn it. The name of the game is to burn it while not losing this muscle tissue. The loss of muscle tissue is caused, of course, by protein malnutrition which carries lots of complications. As you lose muscle tissue the organs start to fail and morbidity and mortality begin to rise. We have been very interested in fasting as a therapy since fat can be burned, particularly to ketones, which can substitute for glucose and thus spare body cell mass.

DR. FREEMAN: Therefore, weight loss is not a function of nutritional status. In point of fact, paradoxical as it may sound, the patient who is forced to lose 25 quick pounds before having his gallbladder removed may, in fact, have starved himself. The early phases of starvation, according to Cahill's studies, actually have shown that what we burn first, since glucose supplies are used up in a day, is protein. The patient who forces himself to lose weight rapidly may actually be burning up the very substances that we want him to use to heal his wounds. If any kind of a diet is to be inflicted, it should be a diet that emphasizes burning of fat, namely, a protein-sparing type of diet.

Let's assume gastric bypass has been performed. On the fourth postoperative day the nasogastric tube is removed. The patient begins to vomit and continues to do so four times a day. On endoscopy we see a large area of edema and erythema, there are no sutures or staples joining the anterior to the posterior wall and this looks like edematous postoperative obstruction. What is the management of this problem?

DR. BLACKBURN: I assume that in gastrectomies we have all had this delayed outlet obstruction problem with ileus, and no obvious anatomical or mechanical defect in the anastomosis. Before continuing I would like to emphasize that even though they won't stop this obviously abnormal behavior of overeating even with the help of medical means, on the data we have now, you are not justified to diagnose this patient as intractable to medical therapy and therefore operate. The procedures are still
under investigation. I do not think enough evidence is in at this time to justify operating on everyone who wants surgery.

Returning to the question, it is not so much edema as it is a low carbohydrate diet that is causing the ileus. Starvation such as Dr. Drennick has worked with does produce starvation ketosis and a diuresis with minimal ileus. Ketosis is not diabetic keto-acidosis. A study in the New England Journal of Medicine shows that within a week of treating a Pickwickian patient with fasting to depress CO\textsubscript{2} production, the patient recovered. Indeed, these patients will come out of CO\textsubscript{2} retention.

We do add protein to supplement the fast to reduce the loss of lean body mass. I would probably treat these people who have pulmonary dysfunction and who are Pickwickian in this way. We have also been impressed that on a low carbohydrate diet glycogen stores and the water in the liver will decrease. Therefore it is easier to fold over the left lobe of the liver which makes it easier to get up to this deep, and very high hole, for a subtotal gastrectomy. The ketosis afterwards will create a natriuresis so that we can minimize water retention and ileus. Again pulmonary function will improve because the hypoxic index will change, and also EFA deficiency will be minimized. In regard to the question of fistula the key thing is its detection. Loss of ketosis is an early warning symptom on stress that would come from a gastric fistula.

DR. FREEMAN: So Dr. Blackburn is saying that protein sparing, both preoperatively and for the relatively minor postoperative complications such as stomal edema, is a potential method of treatment. To assure that everybody knows what protein sparing means, I would cite the example of total parenteral nutrition in which 25% dextrose and 5% amino acids are infused simultaneously. The purpose of this is to meet all the energy requirements with glucose so that the amino acids are used to make new tissue. During 25% dextrose infusion, insulin levels are high, and insulin inhibits fat mobilization. The antithesis of that situation would be the infusion of amino acids only. This is in complete disharmony with all nutritional theory which states that protein must be accompanied by calories, but in protein sparing we have no source of calories. There is no glucose, insulin levels are depressed, and this permits
mobilization of endogenous body fat. The point is that infusion of amino acids either into the stomach or into a vein promotes the burning of fat tissue which can be used as a source of endogenous calories, thereby allowing the administered amino acids to be used, at least in part, for protein, hence the word "protein sparing."

We are saying that this patient whose stoma does not function properly may need some form of parenteral nutrition. Perhaps the physician is reluctant to go immediately to the expense and potential dangers of total parenteral nutrition, so that for a short period of time amino acids are being infused. Such therapy was used in a similar group of patients studied here who had postoperative stomal dysfunction. During amino acid infusion there was a three to five fold increase in the free fatty acid levels. Nevertheless, we have yet to notice deleterious effects of these elevated free fatty acids. Note parenthetically the tremendous salutary effect on nitrogen balance. It goes from -9 to -5 gm/day, during glucose infusion as opposed to 3% amino acids. The question is, do the obese patients react differently to protein sparing? The answer is, they don't, even despite their very large fat stores. In a similar experiment the investigators are running free fatty acid levels around 0.8 to 0.9 millimolar and they have documented the same nitrogen improvement. In the presence of stomal edema, these patients can tolerate protein sparing if you want to use it.

DR. BLACKBURN: Just so we don't think the only way to treat these miserable children is by surgery, I would like to point out that in one of the April issues of the New England Journal we also have an article about Prader-Willi in which I think the results are fairly comparable to the ones that Bob Soper showed us this morning. We used just the protein sparing effect orally together with a behavior and exercise program. We were able to get long term two-year weight loss on these children. The point here is that we are not yet up against the wall on the approach to treatment of obesity. Therefore, we still can do some prospective randomized studies, continue to investigate this area and develop better treatments. At the same time, because we did not create the obesity we need not feel obligated to operate.
DR. FREEMAN: In summary, early postoperative stomal edema is treated in one of three ways: we can use protein sparing, we can simply put in a subclavian catheter and give total parenteral nutrition, or we could have placed a tube either in the distal gastric pouch or the jejunum at the time of every operation, which is a method Dr. Printen favors. Of course, if you do that, you are putting a tube in every patient for the occasional patient who will have this problem, but I think that summarizes the management.

Fourth question: This next patient hypothetically does very well and, in fact, on the fourth postop day she is not vomiting and you see her having her first breakfast. The breakfast consists of a large doughnut like those lethal ones we had this morning. She is putting jam on her toast and she also is having Sugar Pops with cream. What would be your comments if you witnessed this in a patient, George?

DR. BLACKBURN: I would say that I didn't do the intake evaluation properly. If going through all this effort, the patient is not further advanced in the proper modality for eating, I would conclude that I didn't have a good dietitian on my team and I quite frankly think that that speaks very poorly of the required multidisciplinary comprehensive approach to this therapy. We see gastric bypass as an adjunct to a total program. Despite the parallelism between smoking and alcohol, obesity is not a metabolic addiction, it is a behavior pattern. I think for all the effort and the risk of doing surgery that I would be chagrined and I would communicate my grief and sorrow to the patient. I am serious in stating that we are missing the boat and that although the patient is doing fine now by loss of weight, in a year or two this is all going to be for naught without a new life style and behavior change.

DR. FREEMAN: We are poor surgeons, then, if we do this operation and allow a patient to consume calories in an improper fashion. Not only is this meal a very poor one nutritionally speaking, that is, protein deficient, but things such as putting jam on toast are a perfect way to maintain your weight. The patient's behavior modification has not been properly managed. If we want to do this operation it behooves us, then, to have proper counselling with the patient beginning before the operation.
You are in your office seeing patients and a gastric bypass patient who is one year after surgery says the following: "At first I lost 2½ kg a month. I am now down 30 kg and I am beginning to taper off. I am not losing as much, which (a) I'm not happy about and (b) I have a next door neighbor who went elsewhere and he had a jejunoileal bypass and he is losing much more weight than I am and I think you did the wrong operation."

DR. BLACKBURN: Well, this is clearly the controversy now. Those of you who are up against the jejunoileal bypass must be hearing this all the time because this is certainly one of the big points. I would again get out my pencil and paper and show the body composition saying that the weight loss that one loses can consist of fat or body cell mass. There are two ways to lose weight. In uncomplicated surgery about 10% of the weight loss is protein whereas in big stress situations as much as 50% of weight loss can be from body cell mass. Again, if it is protein you are losing, you will also be losing liver protein, albumin, immune defense, ability to repair tissue, and of course they sap away their reserves, fitness and well being. What I would then point out is that with this operation, weight loss is mostly fat. The key point here is that in gastric bypass less than or about 10% of the early weight loss is protein whereas with the jejunoileal it is about 50%. With this explained the patient can take the choice. They can talk about weight loss or they can worry about fat loss, and if you have a skin caliper you can demonstrate that. We had one patient who changed her behavior including giving up candy bars. She had 5 kg of fat loss per month for 15 months, and she vigorously exercised. She gets her fitness tested on a treadmill which takes 5 minutes. Putting these people on a 300 or 600 Kp/m exercise test on an exercise bicycle or treadmill, while measuring their pulse, will tell you in what kind of fitness they are. A comprehensive approach includes nutritional education, exercise, and consideration of surgery, because without the surgery morbid obesity is difficult to treat. Again I am encouraged on an investigational basis to use gastric bypass as an adjunct but I have serious reservation about not using a multidisciplinary comprehensive therapy. I'm against surgery as the total treatment.
DR. FREEMAN: And when we figure out a way to get these large patients in the total body counter, we can do total body potassiums and prove the whole problem.

DR. BLACKBURN: Those lean body mass measurements were made by a whole body K-40 counting.

DR. FREEMAN: She fit in?

DR. BLACKBURN: She fit in. It is an old World War II turret made out of iron that is not radioactive that ....

DR. FREEMAN: A tank.

DR. BLACKBURN: A tank. The counter will take them quite nicely. The clinician can test his own patients. Every six months, a 24-hour urine creatinine clearance will allow the calculation of creatinine height index for your patient. This will allow you to determine exactly how much muscle mass they have lost. The collection of a 24-hour urine will tell you the status of the lean body mass just as assuredly as a K-40 counter will.

DR. FREEMAN: And that was in the American Journal of Clinical Nutrition in December, 1976, "Creatinine Height Index." I hope you will take away from this that there is more to gastric bypass surgery than doing the operation.
DATA ON DIABETES, BLOOD PRESSURE, AND LABORATORY TESTS AFTER GASTRIC BYPASS
Thomas J. Blommers, Ph.D.

Before I present the statistics that we have recently gathered, I would like to give you a brief description of how we collect our information. We gather our data preoperatively, operatively, and postoperatively at six weeks, six months, one year, two, three five and ten years, etc. We have special forms that become part of the patient's record especially devised for this purpose. These forms are put into the patient's chart when the patient is evaluated in the clinic. This facilitates abstraction of data later and also helps to insure that the same data are retrieved from each patient. The operative data, such as measurement and calibration of stoma size, length of operation, length of time on respirator or intubation, and so forth, is taken directly from the chart. We also have used questionnaires, as Dr. Mason has pointed out earlier. We are trying to get a little more reliable on this. It is difficult to write a good questionnaire that eliminates bias. One cannot completely eliminate bias unless you track down the patients that did not answer to find out the reason for this and how they would have answered. The difficulty of obtaining complete information is always a constant problem with research of this type.

During review of patient charts, the information is stored on abstracts which are designed to go directly to keypunching. The data are then put on computer punch cards. After completion of a few practice runs to identify and eliminate the punching errors, we store the information permanently on computer tape.

After we have our information stored we periodically do the statistical analysis. The method of analysis has been with a computer program called "Statistical Package for the Social Sciences" or SPSS. This is an integrated system of computer programs designed for the analysis of social science data. In addition to the usual descriptive statistics such as simple frequency distributions and cross tabulations, SPSS also contains procedures for more complicated statistics such as analysis of variance, multiple regression, discriminate analysis, canonical correlations, etc. We have recently been using regression and discriminate
analysis to try to determine an equation that would predict success or failure for a prospective gastric bypass candidate. As yet, it is much too early to speculate on which variables will be the significant ones. I am not sure whether we will ever devise an equation which will be predictive a significant number of times, but if we do we will certainly let you know about it.

Before going on to the presentation of our most recent laboratory values, blood pressures and information on diabetes, I do want to emphasize the importance of obtaining complete sets of values for each case or patient. Whenever there are missing values the more sophisticated types of statistical analyses are compromised. The possibility of spurious, misleading and useless statistical results increases proportionately to the amount of missing data. The more missing information there is the more problems you have with your research. Anyone considering a prospective study on any subject should try to get as much data as possible. I realize it is not always possible in medical research since we are often dependent on the patient for followup. I noticed some people mentioned the word 'contract' when they were talking about their patients. Perhaps this might be a valid approach for setting up a prospective study. One could make a pact or deal with the patient in which the patient guarantees a certain number of return visits for which the surgeon guarantees a flat rate. I am not sure how one would work out that type of arrangement but it would assure the proper followup data to yield good statistics. That ends my little pitch for the statistics. I am sure you are more interested in some of our results.

Out of 566 patients who have their data currently stored on the computer, we had 46 (8%) who were diagnosed diabetic preoperatively. Thirty of these patients answered a recent questionnaire. Preoperatively four claimed that they were diagnosed but had no treatment, 8 were controlled by diet, 13 had to take pills, and 5 were on insulin. Postoperatively, after weight loss it is interesting to note that 17 of them claimed they were no longer diabetics at all. Two of those who previously took insulin no longer need it. What is even more interesting is the fact that many of the less severe diabetics needed no control at all postoperatively.
At one year followup we have a drop from 146/90 to 138/86 as a mean blood pressure. As the length of followup increases the drop in blood pressure remains fairly constant. Unfortunately we have lost many patients to late followup. This is a perfect example of the problem of trying to present statistics like this when you have a lot of missing information. Naturally, since some of our patients were just operated upon this year, they couldn't as yet have a five-year followup. However, there is a significant number of patients that we could have five-year followup on if we could find them. One nice thing about this operation is that they do lose weight, and, therefore, they don't see any reason to return. Conversely, if they don't lose weight they sometimes get upset with us and never want to come back. The problem, then, is that the sick ones are the ones we often see coming back at three and five years and this may be one of the reasons blood pressure goes back up somewhat with extended followup at five years.

None of this is significant statistically. Perhaps the most important thing is that basically most of these values do not change. Sometimes when you don't have a change this can also be good, particularly in regard to the laboratory values. Hemoglobin drops at six weeks, which is a constant and to be expected with major abdominal surgery. After the six week followup there really are no abnormal values. As for total protein, albumin, calcium and $P_{4}O_{4}$, the values stay basically the same. There is no real change, whereas with some other types of surgery or some of the fad diets, you might expect to see some changes. There is somewhat of a drop in glucose, but basically everything is staying within a fairly normal value. One thing that is interesting to note is the bilirubin at six weeks. It is still within normal limits, but there have been a couple of patients that have received certain anesthetic agents, namely Halothane, and had some problems in the immediate postoperative period. There is even one patient that expired suddenly about six weeks postoperatively, and on postmortem the only thing they could say was that she had fatty metamorphosis of the liver. It is suspected that her Halothane anesthesia was perhaps a contributing factor. Triglyceride and cholesterol levels come down a little, but basically they stay just as they were. In conclusion, I would say the most important finding as far as laboratory results are concerned is that they stay
within normal limits and there are not any significant changes that you need to worry about. Unfortunately, we have not completed many of our studies as yet. However, preliminary data suggests that patients with preoperatively abnormally high laboratory values or blood pressure benefit from the gastric bypass produced weight loss in that their values return towards normal levels.
DR. FREEMAN: It's one thing to say that some of these patients do have problems and do need intravenous access but it is another thing, quite often a different problem, to find the vein. I am presuming that peripheral veins are used up and we do have a problem such as stomal obstruction or gastric fistula. Although it may seem paradoxical these patients have an excess of fat but their protein may be abnormal and they may very well need total parenteral nutrition despite being two or three times normal weight. The needle of a standard 14 gauge intracath is used throughout this country for placement of subclavian catheters. The length of this catheter however, is inadequate to reach from the skin to the subclavian vein of a massively obese patient. A #12 Argyle Medicut works by the opposite principle. Instead of being an intracath it is an angiocath, that is, you puncture the vein with this needle and the catheter slips over it. Its purpose in our hospital is in placing subclavian catheters because this needle is much longer than the conventional intracath. I puncture into the subclavian vein and slip the catheter antegrade into the vein; it is really almost like a Seldinger technique without a guidewire. Next, I withdraw the needle so that the catheter is inside the vein and then through this catheter I slip the 14 gauge intracath. You end up with the catheter in the vein and the needle, then, is just pulled out and sits on the skin of the chest wall. At this point it fits perfectly over the hub of the intracath. There are two points about placing subclavian catheters in these patients. Although we conventionally shoot for the suprasternal notch, as you all know the anatomy is so distorted in these patients that technically your needle really has to be pointing much more posteriorly than normally in order to get the vein or you will just keep hitting the clavicle or the costoclavicular ligament. The second point is, don't tape it over the breast in a female because the breast is so pendulous it will frequently make the catheter go in and out to the point that you may find yourself infusing into the subcutaneous tissues and that can go on for a long time undetected. Tape the catheter well down between the two breasts so
that movement of the fat does not affect it. There is a recent modific­
ation of the intracath not yet available. Although we have some I don't think it is on the market. This again is the standard Basiray #14 gauge 8-inch intracather with a 1½ inch 14 gauge needle. The needle goes in the vein, the catheter with its guidewire goes through the needle, and then you must put the protector so that the tip of the needle does not cut the catheter. This is reasonably cumbersome. The new catheter's needle is about the length of the Argyle that I mentioned earlier. It is almost 2 inches, and believe me it does reach the subclavian vein in a 311 lb patient. This new catheter, which only costs 30¢ more than the old one, is going to be available soon and I strongly recommend it to you, not only because of the length of the needle but, like the former one was, has built into it a blunt endless needle. The catheter goes through this needle and once the puncture is successfully completed this needle comes off and you just clip on the hub and therefore the patient does not have the cumbersome guard and protector of the conventional method. I think it is going to be an extremely helpful catheter for safe subclavian venipuncture in these patients.

COMMENT: I used that catheter for a while. The problem that develops with that catheter is that everybody has to be alerted that the metal bar in there is not a marker for the x-ray to see where it is once it's inside the chest. I've seen that happen. Another problem is that when you put the catheter hub on the little needle that's in there it has occasionally come apart resulting in a lot of yelling and screaming and blood and fluid running around. The instructions say to screw it on. Not very many people read the instructions when they use these things, they just automatically use them. There's a little thread inside there that helps it from coming off, but it still does it. I found that if you put in a subclavian catheter, the ones that work the best are the ones that go in with the greatest ease. It is impossible to put that catheter in with great ease by yourself. Therefore, it is going to take at least two people to do it with ease.

QUESTION: Can you suture your catheter to the skin?
DR. FREEMAN: Not only that, but this catheter makes it much easier because the metal that is in the tip gives you something to tie around without obstructing your catheter. I should have mentioned that as a relative advantage. The former one is not easy to suture. Why somebody hasn't made a catheter with an eyelet on it I'll never understand, but there isn't one. The new catheter is much easier to suture because you put a suture in the skin and then tie very firmly around it. I am not working for this company, by the way.

DR. BLACKBURN: Mr. Blommers didn't mention anything about those hypertensives that you've had in your series. Are there any statistics on what has happened to them? What has happened to their hypertension after they have lost weight?

DR. MASON: Twenty-eight patients with preoperative diastolic pressure of 110 mmHg or higher were studied at one year. There appeared to be a correlation between decrease in blood pressure and decrease in weight but I believe this relationship is spurious. When the change in blood pressure is compared with change in percent excess weight it appears that there is no correlation and that all patients (with 2 exceptions) had at least a 10 mmHg decrease in blood pressure. The median decrease in diastolic BP was almost 30 mmHg.

Fourteen of these patients provided 2 year followup data which indicated a median drop in diastolic BP of at least 20 mmHg. These data suggest one way in which weight reduction by gastric bypass will probably improve survivorship. (This information has been added in the editing and was not available during the workshop.)

DR. BLACKBURN: I have a question for Dr. Mason in regard to the case that Dr. Freeman presented to me, a 28-year-old 300-pounder. In looking at the data that was shown us, you evidently in your population are not seeing hyperlipidemia except for a small group of the 500 because it didn't come out in the mean, or hypertension, so we've really not seen it. Thirty of the 500 patients evidently, or some small group, have diabetes. Since there aren't the risk factors, can you tell me what are the guidelines, if such a patient presented to you, how you handle them?
Do all patients get gastric bypass as long as they are not psychotic? How do you handle the asymptomatic 28-year-old 300-pounder who wants to get married and wants to lose weight?

DR. MASON: They must have tried all other means of weight loss. I must confess that we go ahead with the gastric bypass. However, I think your thesis that they ought to be tried on some protein sparing fast or protein augmented fasting, is a good one. One of the problems with this is that we don't have the help to handle it. I understand that if you want to do this you need to have a program where you can actually teach these people body composition, physiology, exercise, motivational changes, etc., and I think this is excellent. This is what we ought to do, and maybe what I ought to do now is to cut back on my operating and start doing more psychiatric counselling and more instruction.

DR. GRIFFEN: Dr. Blackburn, how many patients do you really get who follow the ideal program? They sure are addicted to food. We've had a great thing with behavior therapy and they've even sent me the patients because of failure of their treatment. I think it's a great idea and I think some of them will do it, but very few of them.

DR. BLACKBURN: I think that your questions are good, and I'm not here to push protein sparing. I am a user and a firm believer in the role of gastric bypass, but I think we are going to be under the same criticism that the jejunooileal people are, in using gastric bypass with no studies. I am concerned that any of you, private practice or academic will say we can't follow the patients because they leave us and we can't get to them. I think you must build in, front-end if you will, in your surgical fees, what have you, the capacity to fund researchers to find these patients, because it is investigational.

I'm certainly convinced that the chances of most people who are 300 lbs to get a cure medically are not very good. What I'm concerned about is that in 1977 if a patient can't lose weight any other way they should get a gastric bypass. I have reservations about that at this time.
QUESTION: We all have reservations. I think what you are really asking is what is the bottom line. At what level does it become optional, at what level is it going to become a strong recommendation?

DR. BLACKBURN: I think you need to get a partner that will follow up and keep records and treat it comprehensively. I think there are a few such people around and they are going to give you your best feedback and be the most productive.

DR. FREEMAN: George, are you saying you are using the operation as part of your behavior modification? Are you going to use the operation as a reward much as a mother might say "Lose 20 lbs and I'll buy you a new suit or a new dress." Are you trying to pick the patient by those that lose a certain percentage but not enough by protein sparing or some other means?

DR. BLACKBURN: I'm trying to pick the people who are in partnership and are not trying to use me. It's more a partnership rather than reward. You do your part, and I'll do my part, and we'll get something accomplished.

QUESTION: Why should there be any question about use of a procedure that can be applied with a low mortality and morbidity to the treatment of people who are more than twice their estimated ideal weight? Morbid obesity is a disease, isn't it?

DR. BLACKBURN: I have to disagree with you, and I would ask you to produce that result. We just saw 500 entries here with normal blood pressures, with mean normal triglycerides and cholesterols, and if it's true, if it's a disease as you say it, that's counter to editorials written in the New England Journal. It's not recognized as a disease by any insurance company for primary treatment. I think the burden is on you to prove that point.

DR. PRINTEN: Try and buy insurance, George. If you're 20% overweight you can't do it.
DR. BLACKBURN: I'm talking about medical insurance paying for operations for obesity. You can get life insurance at increased premiums. That's already been pointed out by Dr. Mason as one of the ambivalences in the industry. But nevertheless, to say in 1977 that every disease you diagnose you have to treat, I wonder if you can really say that. Almost all of us come up with diseases that need treatment that we can't provide. All I'm saying is that in 1977, there must be prospective studies to get this properly in focus. You can't just say that you've got a patient and operating room time and that's all it takes.

DR. KRIDELBAUGH: I'd like to clarify some of the statistics that are presented. You've talked about diabetics who claimed they are no longer diabetic after weight loss. If you take only the hypertensives out of your total group, what happens to their hypertension? On an average, what happens to their hypertension in the course of weight loss? In my experience they are improved and this should translate into prolonged life and decreased morbidity. When I interview these people I'm in a critical legal situation as a private practitioner rather than an institutional investigator. Therefore I can demand of these people that they demonstrate some of the signs which I consider to be potentially catastrophic in morbidly obese conditions. They must be morbidly obese. I don't think that the weight of 300 lbs. means a thing. It depends upon whether that 300 lbs. is on a 4' 9" woman or a 6' 7" basketball player or football player. I want to know what their body weight ought to be.

I want to know if they have hypertension or if they are showing some physical defect of an orthopedic nature or something that needs weight reduction. I disagree with you that this is a medically treatable condition. These people are foodaholics. If they don't touch a bite of food they can stay off food for a long time. It may be their acidosis contributes to their continued anorexia. Nobody has mentioned the reports in the medical literature showing in the last year that the patients with jejunointestinal bypasses were losing weight strictly on the basis of decrease in caloric intake, probably due to the acidosis and cramps and aversion to diarrhea.
DR. BLACKBURN: I said there was no metabolic cause of obesity. These people have a behavior that is abnormal and they are consuming food in an uncontrolled, involuntary manner, in excess of need. It is not a metabolic addiction, it is behavioral. Maybe we can get Dr. Drennick, who probably really knows more about this, to give us some guidelines in this area. I agree that these people have abnormal behavior and they are eating themselves to death. But, if the patient says he wants to get married, where does that fit in your categorization? What are the indications?

DR. MASON: Let me ask George, on his data about blood pressures, how does he know that a blood pressure taken in a 300 lb. person with a particular sized cuff and particular sized arm, has true meaning in relationship to the blood pressure taken after weight loss? Are these determined with arterial punctures or are they just a cuff, what size cuff?

DR. BLACKBURN: Well, of course, the blood pressures have to be determined with a large cuff for the large arm. We know there is a difference between the obese cuff pressure and the true arterial pressure, but the variables I am talking about, Ed, are way beyond that. They are people who start off with diastolic pressures of 110 to 120 that drop to 80 or 90. I think it is beyond the technic area. Is it out of place to ask Dr. Drennick to input as to what our obligation is to treatment for these massively obese people? He has probably done as much as anyone in the field.

DR. DRENNICK: Thank you very much. It is a very big question and I am afraid I have no good or valid or all-inclusive answer. Everyone of you will have to individualize for any one particular patient. The reason I am here, as an internist, I think, is a testimonial to the fact that I don't know how to permanently relieve these obese people of their obesity. George, you say it is not a disease, and I think in the strict sense you are correct. Obesity in itself, perhaps, is not a disease, but I am quite convinced that anyone who carries 100% excess weight over a period of years is going to damage his body in a variety of ways. The figures that I mentioned to you this morning about reduced survival in the very
obese individual are simply a result of the wear and tear to which the superobese individual is subjected. If you expect that so many years of obesity are doing harm, you should do something to prevent that harm.

How are we going to do it? I will admit to you that in those 200 or so patients that we were able to treat and reduce to normal weight, after a 10 year period only 7% remained reduced even though they all had been reduced to normal weight. I know that George is doing a better job with keeping people reduced, but I think he should continue that and he should follow his patients very carefully for ten or more years and see how many of his patients will still be reduced at that time. Our patients who have no followup care to speak of or very little, all stayed reduced for two or three years, reasonably well, but after that there was a very rapid lapse and they regained. George says he only accepts a certain number of potential behavior modification patients, but what to do with the others. I think we still have to offer them something, even though they may not be suited for behavior modification or protein sparing fasts. I think that with some exceptions, which most of us are able to spot, anyone who is 100% excess in weight, not an absolute number but excess weight 100%, and who has been in that weight range for 10 years is almost certain to be a hopeless medical treatment candidate. I just say that from experience. You may point out five or ten years from now that I am wrong, but I don't think that you or I should wait and put every obese individual to the risk of the next ten years with the chance of developing all those diseases they eventually may succumb to just because there may be a better treatment ahead that we can recommend ten years from now.

DR. BLACKBURN: Would you randomize now? How do we decide who we do or do we do them all now? I did not say I didn't think it was a disease, being 100% overweight. I feel the opposite. I'm concerned about getting the studies so that it will be established by criteria rather than just gaining 500 cases with some weight loss. We haven't heard the five-year followup of those cases. What is your recommendation on getting started today? We certainly want to treat some of them. What are the guidelines at 100% overweight?
DR. DRENNICK: I think that we have to separate very, very clearly what our desires are and what the patient's desires are. They are two totally different perspectives. Your desire is to furnish objective scientific information and criteria. The patient is not interested in that. He knows that he can't get a job, that he has been unemployable for the last ten years, and he wants to finally live. Even though it is very desirable that we should accumulate objective data that would be guidelines for all of us, I think I would still treat almost any patient who, by what we have discussed this morning, indicates that he is sincerely motivated and has a purpose in losing weight. I would not send him away, I would not defer beyond maybe two or three months to see if this man is really determined to have the surgery and accept the risks and possible ill effects. I don't think I would turn anybody away any longer. I think that morbid obesity is a serious enough threat to the patient's normal health and life span that we have to help him.

DR. BLACKBURN: Ed, you must be the only people who have a five-year followup. What is the mean change in this 100% overweight patient?

DR. MASON: (added in editing) We have some data for 10 years and 42 patients have been followed for 5 years. We have observed a 35 kg weight loss at 5 years, 24% of initial weight, 43% of excess weight and 27.7% standard deviation of percent of excess weight. Others are observing better weight loss and our data is better now for the patient operated upon with a small measured pouch volume and a small stoma. The earlier data indicate that the one year followup data are a reliable index of the five year figure. On this basis it appears that our 5 year results with a small pouch should produce a 39 kg weight loss, 31% of initial weight, 55% of excess weight and a standard deviation of 17.9% of excess weight.

DR. DRENNICK: With an average individual, and I think this is probably in your experience as it is with most of us, the average obese individual male will be between 300 and 350 lbs. If you can get 80 to 100 lbs off an individual like that, I don't know if he is going to be much healthier but certainly he is going to have a much better quality of life and I think this is an aim and a goal that is worth our pursuing.
DR. BLACKBURN: I think we can't have it from a better source, so the goal of 80 to 100 lbs looks like the criteria of major importance.

DR. RANK: I do feel like I have to just say that we have to be careful to ask the right question, and we almost got off on the wrong question. I believe the question we are trying to get at is, will this work and can we do it safely? Now if we get off into equitable considerations we are going to lose a little bit of ground. I have never been morbidly obese, but I dare say that there are probably some of you here that have gone into this because you were, and I would hate to be confronted as a 350 pounder when someone tells me I had a good life. So we must decide: will it work and can we do it safely, and are there any harmful long term ill effects? The plastic surgeons don't ask you if you have a disease when they do a mammary augmentation, and they don't ask you if you have a disease when they fix a face or a crooked leg, or a rhinoplasty if you're a salesman. Let's not forget all those things. There's one last little thing. I've had a chance to talk about motivation, that's my business, and I talked about it this morning. There are ways. But right now everyone think about one question. Do you know that no one has looked at what are food sensors. For example, you come in after a hot day and you're warm and you start to drink some water. You drink a glass, and you drink another glass, and that's enough and you quit. Have you ever noticed that? You have some sort of sensor that tells you when you have enough water. We don't know anything about food sensors, and we're forgetting something. When I sit down to satisfy myself, I'm feeding 165 lbs. But that person who weighs 265 lbs is feeding 265 lbs and we don't really know about what the sensors are. Do the thin people have something that says "that's enough for your weight", and are the fat people feeding a 300 pound weight. That would be a good question to look at.
GASTRIC BYPASS IN PRIVATE PRACTICE
H. Richard Hornberger, M.D.

I'm one of the rabbits on the gastric bypass tour. If I have anything to contribute to this workshop it may be to tell some of my experiences to the people who have not done gastric bypasses and are wondering whether they should start or to those who have done a few and are wondering whether to continue, particularly if they are doing them in relatively small hospitals. Also, after hearing yesterday's heroes it occurred to me that perhaps the workshop needs a villain and I might be able to supply that. I am a general and thoracic surgeon at Thayer Hospital in Waterville, Maine, where we have 175 beds and no interns and no residents. This is not going to be a scientific talk in any way; it is going to be personal and anecdotal. I will try to tell you how, in my experience, gastric bypass fits into private practice in the community hospital.

Since 1970 I have done 80 gastric bypasses, 52 of them in the last two years. My assistant on all but seven of these patients has been a board-certified general and vascular surgeon who is perfectly capable of doing a gastric bypass but he has never done one. He has had considerable exposure to my postoperative patients when I am away. When I ask why he doesn't do gastric bypasses, his invariable answer is "I just can't stand these people." My secretary is a highly intelligent and well disciplined college graduate. She does not approve of what she calls my hobby. She says bypass patients are a nuisance, have no phone numbers, move around a lot and never keep appointments. I'll enlarge upon the obvious implications of these attitudes later, but first I'll describe how I got into the bypass business.

Back in 1970 a gaggle of internist, psychologist, rehabilitationist, social service varieties and what not had been struggling for three years with a 350 lb 30-year-old mother of three who was divorced from a husband who also needed psychiatric help. Finally the internist came to me and said "Do something." I knew nothing of surgery for obesity and had no interest in it, and jejunoileal bypass was the only procedure I was aware of. I read up on how to do it and did one on this patient.
She lost 100 lbs in the next year but spent most of the time in the hospital with electrolyte and other problems. Again our internist came to me and said "Do something." I had become aware of gastric bypass, so I put her small bowel back together and did a gastric bypass. She got down to 180 lbs, where she has stayed since then, and she has also stayed in the hospital, working part time, when she doesn't have phlebitis, and every day she says "Hello, Dr. Hornberger." For the next three to four years I did a bypass about every six weeks, and my fourth patient, a 350+ lb 5' female died suddenly on the third postoperative day, very likely due to a leak. I decided to terminate my career in obesity.

A couple of months later, though, I was called to a coastal town by a general practitioner who virtually commanded me to do a bypass on a lady whom he was very fond of and very tired of. She was a 52-year-old 350 lb widow with a 14-year-old son. She had no money, no hope, a total cripple physically, economically and emotionally. She eventually got down to the 170 to 180 lb range and stayed there. She has taken an LPN course and has become a useful, self-supporting citizen and sort of a mother to many of my subsequent patients. Her son has also developed. He goes 280 and is attending a chef's school in Providence. Always reluctantly, I did a few more and in the fall of 1974 I operated on my first male patient. He died three days later, also of a leak. He was number 23 in my series. This gave me two deaths in 23 cases, a mortality rate, according to my secretary, of 11.1%. This was certainly not an acceptable statistic for an elective procedure, particularly in the microcosm of a small hospital where everybody counts. The operation had achieved a bad reputation. I reevaluated my own position and attitude toward gastric bypass. Despite a bad record, patient referral and occasional doctor referral caused someone very heavy to show up in my office every two to three weeks. I discussed the problem with our Medical Director and a few others who felt that the need for this kind of surgery was minimal but since I had started, why didn't I learn how to do it.

In late 1974 I boarded a series of airplanes and taxicabs which brought me to a motel on the outskirts of Iowa City. In the dining room of the
motel that night I had catfish for supper, figuring you only live once. The next day I met Drs. Mason and Printen, talked, and watched Dr. Printen do a bypass. It turned out that with minor variations we were doing about the same thing. I got useful hints about the postoperative care and danger signals but left with a great degree of skepticism. These folks had residents who knew what to do and knew what was going on and could reinsert subclavian lines and ride herd postoperatively. If I started doing bypasses again I was still going to be very much alone. I went home undecided as to what to do, and the real question was, was I willing to stand the grief. Somewhat fortified by my Iowa exposure and modestly in demand, I did a few more with good results, although not without trouble.

The turning point in my own attitude and confidence came in May, 1975, when Big Joe came to see me. I am telling the story to make a simple point that obese people are absolutely desperate. By this time I had developed a basic sales talk which went about like this: "I have done 27 of these operations, I have had two deaths for reasons possibly but not certainly preventable. I have had two marginal ulcers, two or three pulmonary emboli, five wound infections plus other complications. It is a major surgical procedure with a possibility of a variety of major complications." I didn't want the patients to be as scared as I was, but I wanted them to be wary. Getting back to Big Joe, he was 32, weighed 465 lbs and filled the hallway between the waiting room and my office. I have made a point of watching each prospective patient as he or she takes this walk. I deny the allegation of my surgical assistant that if they can make it I'll operate on them. Big Joe, a construction worker, walked the 20 yards with a springy, almost jaunty step, despite his weight. In the course of history taking, I asked him about his parents. He told me that his father had died while being operated on for a hernia. I thought to myself, this sort of thing used to happen around here with all the GP surgeons we had in the town in those days, and I said, "When was that and which klutz was operating on him?" "You were," Joe said, "about 15 years ago." That's a story in itself. I said "Yes, I remember ... now." "Look, Joe," I said, "I've only done one as big as you and she's had a lot of trouble. I've only done one other man, and he died, and I operated on your father and he died. How
do you like them odds?" Joe said "Doctor, the way I'm going I'll be
dead in five years anyway." I really wanted to run away from Joe, but I
figured that he was a needy client. I offered to ship him to Iowa City,
but he couldn't or wouldn't. He had the operation, he broke his incision
open four days later, and I got it back together. Three weeks from the
time of his bypass he was at work in his company office. I have since
repaired his incisional hernia, and 18 months postoperatively he had
lost 250 lbs and has been readmitted to the Marine Corps Reserves. He
weighs less than I do and is generally considered better looking. As a
result of determination, weight lifting and other exercises he has very
little flab on his belly or anywhere else. A result like this feeds a
surgeon's ego and it also feeds his practice. Joe is now back building
roads all over the State of Maine, I have done a bypass on his sister
and probably a dozen overweight females and one male he has encountered
in various hash houses or just along the roadside. Joe will stop when
he sees someone fat walking along the road and talk to them, and, likely
as not, give them my name.

Now I'd like to discuss to some extent three problems which seem signi­
ficant to me. One is the individual physician's attitude and philosophy
toward gastric bypass, the second is selection of patients, and the
third is the medical profession's attitude towards weight-reducing
operations. The first, the individual doctor's attitude, I was pushed
although not forced into bypass surgery. I will admit to having become
at least mildly fascinated by it. This may simply be because my results
are so much better than my results in surgery of lung cancer or other
cancer. I think the chief factor is that a high percentage of these
people are so delighted with the results that they make me feel good, or
maybe they make me feel important when it works out well.

Secondly, the selection of patients. With few exceptions, I have adhered
to the basic principles that they should be twice normal weight or at
least 100 lbs overweight with a five year history of futility and not
over 50 years of age. Basically what I have done is bypass virtually
all of them who have met the qualifications with the attitude that I am
purely a mechanic and can probably make them thinner, at least get them
through the average door. I see no major role for psychiatry in this
business and am particularly wary of patients referred to me by psychiatrists. I have rejected only two patients who have met the standards. One was a pickwickian 40-year-old man who had already had a bypass. It was a 75% bypass instead of 90% or more. His wife said that all he did was sit in the kitchen and drink beer and eat hamburgers. He sat before me like a giant bullfrog and burped every 15 seconds. There was nothing I could do. This might have been an interesting surgical exercise, but the patient's attitude was negative and I demand positivity. The other rejectee was a 23-year-old male, a member of the uninformed, uneducated and combative proletariat. He asked innumerable meaningless unanswerable questions and he paid no attention to anything I said, and took an hour doing it. I told him I could tell him everything I knew about the operation in five minutes, that there was no way I wanted to sell the operation, and that the only people who get it are those who qualify and beg for it or come close to that after being told clearly and repeatedly of the possible problems and risks. The patient complained to his welfare worker that I had been impolite to him. That may be, but I won't operate on him.

I think I have reached the point where I can make a fairly shrewd guess about how a given patient will do. The ideal patient is a reasonably intelligent, basically sensible, still married housewife and mother who is not divorced, likes to go swimming or bowling, has a few outside interests and just happens to be overweight. These are the people who come in, ask a few basic questions, and listen to what I have to say. I give them a list of earlier patients who are willing to talk to new patients, and this kind usually simply says "Put me on the schedule, Doctor, and yes, I would like to talk to the former patients." The other extreme is the patient frequently referred by a psychiatrist or someone in some clinic who will do anything to get off the hook. This patient sprawls before you with a face devoid of expression, understands little or nothing of what is said, but declares that she wants the operation. One classic statement was "The old man has got to roll me out of bed. I can't get up my ownself." This 30-year-old 300 pounder arrived in a battered pickup truck with a skinny husband and four children. This kind I will do, if enough people get after me, but I do it with reservations because I know that even though they lose weight they
may still be a burden to themselves and their family and even society. Between these extremes is a fascinating variety. As far as I can determine, the most profound statement one can make about them is that they are better off thin or approaching it than obese.

In reviewing my series of cases, I can pick out at least 10, or roughly 15%, that I wish I had never heard of, even though the surgery may have achieved the desired result. I can pick out another 10 whom I classified as bad news preoperatively but have become good results in every way. I would like to reject the patients whom I instinctively don't like or whom I instinctively feel are not good candidates, but I don't really think one should be that whimsical in the selection of patients. This brings us to the subject of the medical profession's attitude toward weight-reducing. Although alcoholism and drug addiction are now accepted as diseases, many people in and out of our profession look upon obesity as something undesirable and something vaguely sinful and unpleasant, and something which is obviously the patient's own fault. Although there are crusades against obesity, we have no modern Carrie Nations who fire bomb these stores that sell Pepsi Cola and potato chips. We do have Weight Watchers and various organizations which are helpful to many. Perhaps I see only their failures, but my impression is that they are not as effective as Alcoholics Anonymous. As doctors, whatever our specialty, we avoid obese people. Their medical, surgical and psychiatric problems are magnified, obviously. A lot of doctors are repelled by them and have no sympathy for them. Two friends who for over 15 years have sent me all the surgery they could find or conjure up, have never referred a gastric bypass patient. One of them has said, "I don't see why you are doing this." Other doctors in my community who frequently refer surgery have sent a few bypasses, but usually it is a weak referral. They may see a patient and suggest that he or she be sent to me, but they don't call me directly. Perhaps one reason for this is that about 20% of my patients have been such a nuisance to the whole medical community in one way or another that no one wants to get anywhere near the action.

I spoke on gastric bypass to the New England Surgical Society in the fall of 1975 and the paper appeared in the American Journal of Surgery.
As a result I've had referrals from areas I don't usually serve, as well as a few from out of state. With rare exceptions this is still an oblique referral. The patient is given my name. If someone wants me to see a patient with a lump in the lung, he usually calls or writes and gives me a recital of the blood counts and the x-rays, etc., and asks me how I am hitting the golfball. The same doctor referring a bypass patient says "See Hornberger. Here's his phone number." At least 50% of my patients have been patient referrals. I have already spoken of Big Joe, the construction worker. He has become a gastric bypass missionary, and there are others like him. Last fall I operated on two early 30's mothers who worked in the same office. Last summer they were on a beach in bathing suits exhibiting their total of 600 lbs, and a total stranger approached them, said she had had the same problem and why didn't they go see Hornberger, and catch the thins. They did, and they have done very well. They have a chart in their office showing weekly weight loss and they discuss it with every customer, and I think most of the fat people in the whole county rotate through there and talk to them. I think patient referral has this significance. The majority are glad to have the operation and indeed are leading dramatically improved lives. Even those who have had problems have lost weight and they recommend it to their large friends. This leads to a fairly steady stream of candidates.

I wrote or scratched out most of this talk during the Christmas holidays. Since that time I have done 16 bypasses in eight weeks with no significant complications and no hospital stays beyond eight or nine days. Having done two cases a week for eight weeks and having another ten or so scheduled for January and February, I began to question where I was going with this business. As I said, it started involuntarily and went along for four or five years, but now it suddenly exploded. At that time I didn't really know what to do about it, and I still don't. My long-suffering surgical assistant has declared that he can't stand more than one a week even though his involvement in the case is seldom more than an hour. My secretary has requested that I refrain from operating on 300 lb females who come to the office wearing shorts. Back around Christmas time, I had done between 40 and 50 consecutive cases with no mortality, no anastomotic leaks, only one major complication, and only
one which threatened to be fatal. If I pick the right 50, I've got a great record. At that point I figured that I had the touch and decided the operation was easy. I would like to leave out the next paragraph!

In my last ten cases I have had: 1) a major pulmonary embolus, 2) a sudden death three hours postoperatively for reasons completely unknown and not discovered at autopsy, 3) a wound infection, and 4) an anastomotic leak.

When Dr. Mason asked me to speak on gastric bypass in private practice, I think he was really asking two questions: 1) should an individual surgeon not affiliated with a large teaching hospital undertake this kind of surgery, and 2) is the need for gastric bypass great enough to justify doing it away from the big league? I think the answer to both questions is yes with qualifications. There is no reason why a trained surgeon in private practice away from a medical center shouldn't do this operation, particularly if he has had experience working in this corner of the belly, and second, the need is great. Obviously there are vast hordes of these people and I think many of them will get well only through surgery. They will try beads, acupuncture, or any other absurdity, so if you can make this operation safe it can well be done in the boondocks.

The real question is, from the surgeon's point of view, is one willing to put up with the grief. Not just the surgical complications, but the handling or dodging of fringe problems, medical, psychiatric and familial, with which one becomes involved. Gastric bypass is not for the occasional operator, as my early and late series indicate. However, with the majority of good results, the surgeon who starts will certainly have an increasing bypass practice and the other surgery that it generates, and it does generate a lot of surgery. As he becomes more familiar with the technics the problems will diminish, but as the numbers of patients increase he is likely to find the bypass business becoming a relatively high percentage of his total work. I would figure that if I keep on with this there is too good a chance that in another couple of years I will be doing nothing else, and I don't really want that.
In conclusion, I would like to present one case which combines many of the problems common in this business. Dolly came from 75 miles upcountry, standing 5' 9" tall and weighing 330 lbs. She had three children. She could never get to sleep until 2:00 a.m. and had to get up at 4:30 to get her husband off to work. She had some kind of inadequate hospital insurance and her husband had a job. This made her relatively deprived because she didn't qualify for Aid to Dependent Children or any other taxpayer's subsidies which would pay me, the hospital or anyone else. She did very well and left the hospital in six days, mostly because she knew some of her own money was being spent. Her surgery was in September. Just before Christmas she appeared in my office 60 lbs lighter, trying to sell me a line of kitchenware for $100. She offered an unbelievable bargain which would spare my wife hours of labor but not actually serve the meals. I was able to resist this and prescribed sleeping pills. A year later, weighing 190 lbs, she got gallstones. Her bypass, when she weighed 330, was uneventful. After cholecystectomy at 190 she had a subphrenic abscess and a prolonged hospitalization. I finally sent her home on an oral antibiotic with instructions to call me if her temperature went over 100. Two weeks later she called and said "Doctor, three nights ago I made love to my husband for the first time in seven weeks, and ever since then my temperature hasn't gone over 97. Doctor, can you tell me what that means?" "Dolly," I explained to her, "that means that you are frigid."
As an introduction for this talk, I think I can simply say that I share all of the concerns and all of the observations I have heard up to this point in this total symposium. One thing did strike me yesterday as I was sitting here listening and sharing thoughts with the speakers. It was the fact that we didn't know much about morbid obesity and it seemed to be bothering everyone. It bothered us that we didn't know much about the etiology. It bothered us that we didn't know much about the risk of morbid obesity. It will always bother us until we answer those problems. With respect to etiology it upset me a little bit, too. I started to think and I couldn't figure out too many things that I operate on for which I really do understand the etiology. I started with cancer, which is the other two-thirds of what I do. I don't understand the etiology of cancer, and yet we operate for it. It seems to me that most of the time when we figure out what is the matter and what the etiology of these things are that we can stop operating on them. I think probably that is going to happen with obesity, at least I would hope that's what would happen. I am sure that surgery is not the ultimate answer.

For the usual reasons, all given here by Dr. Hornberger and other speakers, we got interested in the concept of surgery for the morbidly obese patient. We have a very active Metabolic Service, headed by Dr. Robert Bollinger. He has been taking care of morbidly obese people for a long time with various sorts of diets which we call, since we are in Kansas, the Jayhawk I, the Jayhawk II, and the Jayhawk III diets. He observed that any patient who weighs more than 100 lbs over their ideal weight simply cannot diet adequately. In the initial part of our program we asked a question that turned out to be the wrong question: "Which of the two contemporary operations for morbid obesity is best, the Scott operation, or the Payne procedure?" We selected our patients in the usual sort of way, rejecting those that seemed to us or others to not be good candidates. Most of our initial patients came from the Metabolic Service. They had been on Jayhawk I, II and III, had failed miserably, and were sent to us. I agree with Dr. Hornberger when he states that patients
not only referred from psychiatrists but those who are referred from doctors are sometimes not the right ones to operate on. Many doctors are wanting to get rid of the obese patient. They are tired of trying to deal with these people and trying to get them to lose weight.

We selected our patients 125 lbs over ideal weight. We didn't find anything endocrinologically wrong. As far as Cushing's disease is concerned, if the patient weighs more than 225 lbs, that's not Cushing's disease. Consequently we are no longer checking for Cushing's disease unless there is hirsutism or something else in the history or physical examination that would indicate there might be something metabolically wrong. Initially, we did pulmonary function studies on everyone, but lately we have stopped doing that. We are still getting serum lipids and cholesterol and doing all the things that are required to operate on these patients.

We were comparing the jejunoileal shunt end-to-end as advocated by Scott and the end-to-side operation advocated by Howard Payne. We set up a randomized study in which patients were evaluated and randomized for one of the two operations. We pulled a card at the time of surgery to decide which operation would be done. We all favored the Payne operation because it was technically easier to do. About midway through the study, as we set about the work of getting our data together for our first presentation of this information, we totalled up all our complications including inadequate weight loss as one of them and we came up with 81 complications. This was fairly striking in view of the fact that we only had 52 patients. We obviously had more than enough complications to go around for all patients.

There were the usual sort of surgical complications. I'm not going to dwell on these this morning because I think you have all read the literature and you have had this hashed about before and are familiar with the things I am talking about. The surgical complications, except for one, I can understand. That one was an acute dilatation of the colon. It was in a man who just dilated his colon, became sick, and we ended up doing a colectomy and almost lost him. I don't understand what happened to him, but the other surgical complications I do understand.
All of us who have done jejunoileal bypass have observed gastrointestinal tract complaints and complications. I think most of these are fairly acceptable. All of the patients had all of these things to some degree. We did not count them as complications unless they became bad enough to require hospitalization. For instance, we didn't count diarrhea unless the patient had to be hospitalized and intensively treated with intravenous fluids. The metabolic complications, however, were, to me at least, terrifying. There were so many things here that were bad, but the really bad part was I didn't understand them. Now I think we have come to understand some of them. The business about the arthritis has been worked out. We had a patient get systemic lupus with a butterfly rash. I don't know if that is a genetic thing, but I don't think so. I think somehow or other it is related to the physiology or pathophysiology of the jejunoileal bypass. It seems to me that when you take patients that you do not understand who have diseases that you do not understand, and then impose upon them an operation that you also do not understand, you are taking a great risk. Three wrongs certainly don't make a right. At this point we became very concerned. Our inadequate weight loss in this group of 52 patients was a total of 15, which I thought was excessive. Maybe we were doing the operation wrong, and we had been told that, and it is possible. We'd be the first to admit that.

We scored our results into excellent, good or poor, just as a very crude way of looking at our information. Essentially we counted an excellent result in a patient who had had adequate sustained weight loss greater than 6 lbs per month. A good result was that same weight loss but with one significant complication, not minor diarrhea or a minor complication but only a major complication. A poor result was a patient who had inadequate weight loss or more than one significant complication. It is a fairly crude scoring system, but it is a way of looking at the data. We looked at our different groups, the end-to-side and the end-to-end, to see what had happened. Although prospectively we didn't plan it this way, what was happening was that individual surgeons (there were three of us doing these; at The University of Kansas: Craig Hardin, Arlo Hermrick and myself) were seeing patients who weren't losing weight adequately and we were shortening the limbs that we were using so that we ended up with different groups. For instance, with the end-to-side
procedure we had groups of 14 and 4, and 10 and 4 inches. Nevertheless, if you look at the total distribution of all operations we had 11 excellent, 11 good and 29 poor results. If you look at the distribution with any of the combinations of how they were done or what the lengths were the curve or the bias in the data seems to be always over toward the poor side. We were very chagrined at this and in a way very surprised that it had worked out this way. We were expecting it to be better. We observed that older patients did worse than the rest. The younger patients didn't do very well either, but the older ones seemed to do very poorly. The best group, certainly, were those that were 30 years of age or younger. In this age group we had 15 good or excellent results and only 6 poor results. If you have to continue to do the operation, it should be in very obese patients where it is technically difficult to do the gastric bypass procedure and it should be done on the very young patients. Older patients just don't hold up well with this operation.

At this point we felt we had to change something. We were very dissatisfied with how the study was going. Perhaps unwisely we decided, rather than to start into the gastric bypass work, (since we had our minds set against the jejunoileal shunt) to just set it aside. Now I realize there are others who disagree with that, and that is fine, but that was our feeling and that is why I am here today, to relate to you how we felt at the time and what we did.

We did start to do the gastric bypass, and since then we have done several .... I am going to tell you about a couple of different series. The data gets a little confusing at this point, but I think you will be able to understand what I am talking about. In total now we have done about 250 among the three surgeons that are involved. We have essentially used the old-fashioned technique with a Billroth II type of gastrojejunostomy. We go into the lesser curve about the same way but we take down the greater curvature to the gastrooesophageal junction. I can feel the left gastric artery in only about 75% of the cases, and in the other 25% I just can't seem to be really confident about where it is, so I stay down low enough. We then transect the stomach using the TA-90 stapling device. Our original work was based on using what we estimated to be a 10% pouch. We try to be very careful about saving the upper
branches of the left gastric. We do that by passing a Robinson catheter through the lesser curve side and guiding the TA-90 through, dividing the stomach, and then bringing it down and doing an anastomosis below the colon. We don't do it above. We try to keep the anastomosis site small, and I think that is also important. I want to share with you some data and why I think that is important.

There were 75 patients in our initial group. We have had two B complex vitamin deficiencies. One, a patient who refused to take vitamins even though we hospitalized and force fed them, and another, a patient I don't understand, who vomited quite a bit initially, very quickly got a vitamin deficiency and who got sick and almost died before she recovered. She had a peripheral neuropathy which has now improved and she has done quite well. We had 7 patients in that original group that we felt had an inadequate weight loss. We scored our results in the same way that we had the jejunoileal shunt into excellent, good and poor. We looked at the weight loss in the patients, comparing our gastric bypass series with the jejunoileal shunt series. We feel our data demonstrate that there is about equal weight loss in both groups. Therefore, we felt that, in our hands at least, the gastric bypass would produce equal weight loss to the jejunoileal shunt. We had 34 excellent results, 11 good results and only 9 poor results, which was quite a bit better than the jejunoileal shunt series. When we looked at this versus age it was remarkable, to me at least, that the older patients as well as the younger patients seemed to do quite well with this operation.

We then did one other thing that I want to briefly allude to because it is going to be discussed later as well. We have now taken down 14 of our jejunoileal bypasses for a variety of reasons. Some of these have had a gastric bypass. The reasons for taking the patients down are the usual sorts of things. We felt that these problems were a trial for these patients and intolerable. The patients were tired of them; we were tired of having the patients complaining about these things and also worried about all of these patients so we took them down. In the asynchronous group we took down the jejunoileal bypass. They then gained back weight so we did the gastric bypass and these patients didn't lose weight very well. I think the reason for that was very
simple, we were conservative in doing the gastric bypass because we were concerned that in a patient who had already lost weight the weight loss might be excessive but I don't think it would be. In those patients where we synchronously took down the jejunoleal bypass and then did the gastric bypass at the same time, they in fact lost a little bit more weight, although not a lot. The conclusion of this study was that the synchronous procedure is best.

I want to tell you about my personal experience. There were 88 patients when I collected these data about the first of the year. I now have over 100 that I have done personally. The average weight was 277 lbs and the age range was between 22 and 51. There were no deaths in this group of 88 patients. I've been lucky so far and if we keep going we're certainly going to see deaths. As a matter of fact, you'll notice also that there were no leaks in these 88 patients. Just last week we had our first leak. Thanks to Dr. Mason's warning about rapid pulse, we did a gastrografin injection of the stomach, demonstrated a leak, went back, repaired it and he's home now. We did have, unfortunately, a laceration of the diaphragm.

Nine patients had inadequate weight loss out of the 88. They were all females. All male patients lost weight well. I don't know why except that our male patients have been working or want to. Of concern are the patients who have had inadequate weight loss. I count adequate weight loss as averaging 40% of the body weight. That means if someone weighed 250 lbs they have to lose 100 lbs, if they weigh 300 they would have to lose 120, etc. That really is the minimum that we should expect from any operation that we are doing for this problem. All of these operations of which I am speaking now were done prior to June, 1975.

We have revised four because of inadequate weight loss and we did that for cosmetic reasons. We were pressured by the patients and I'm not sure we should have done them. The weight loss in all these patients was 80 or 90 lbs. These first 88 patients have taught us that it is no longer necessary to be overly concerned with respect to the endocrine system. We only are doing a T4 and we still aren't finding anything. I'm not sure we should even do that. We're doing a cholecystogram on
all the patients because we've got the experience of having to do cholecystectomy within 1½ years after surgery. I think that what happened was that we just missed at the time of surgery identifying a bad gallbladder. A lot of these patients have a lot of fat around the gallbladder and I've had trouble evaluating it. I'm not absolutely certain that I haven't missed stones in some of them.

We're also doing psychological tests. We have a clinical psychologist who is very interested in this problem. We use the Minnesota Multiphasic Personality Inventory and other intelligence and personality evaluation tests. We are going to recheck the patients afterwards to see what effect the bypass has had. There have been such studies in the past. I hasten to point out that if you want to look at an area where improvement has been shown in regard to operations performed for morbid obesity it is in this area. The psychologists are convinced that there is benefit. It gets back to the basic problem or idea, that obesity is a behavior disorder. The psychologists will listen to me, and they also talk a language that I understand. Our psychologist will call up and say "that patient's not a suitable candidate and I don't think you ought to operate," and usually I'll agree. If I don't agree, then I do operate. But that seldom happens. Most of the time we agree. I give the patients, the first time I see them, a very detailed description similar to the booklet you have here. It's all about the operation as I see it, and I make the patient and family read it and I go over it with them. After that I'll answer questions for them.

We get an arterial oxygen determination preoperatively. I think that's probably the best single parameter to determine pulmonary function. We use preoperative antibiotics and we've had very few wound infections. We start Keflin the day before surgery, give two doses of 500 mg each, continue it only 24 hours postoperatively, and then stop. We don't do a formal bowel prep using the kind of prep you would for colon carcinoma. We extubate all our patients in the immediate postoperative period. At Kansas we have an extubation protocol which we follow. When patients meet the requirements for extubation we extubate.
It's critical to have the pouch size so we measure it, as Dr. Boyd suggested, by putting a nasogastric tube in place and inserting 60 ml of saline at the time that the TA-90 is in place. If it seems that the 60 ml isn't filling the pouch we set it up a little bit higher. Another thing we insist upon is a stoma size of 12 mm. I don't believe that adequate weight loss will result if the stoma size is very much larger. There is considerable disagreement, however, concerning this point. I know many surgeons prefer a larger anastomosis. It is noteworthy that the gastrojejunostomy done with this small anastomosis almost invariably opens up by the fourth postoperative day.

In conclusion, it appears that the gastric type bypass will produce at least as good a weight loss as the jejunoileal shunt. Complications of gastric bypass are considerably less than jejunoileal shunt. We have demonstrated that jejunoileal shunts can be taken down and a synchronous gastric bypass can be performed safely.
INTESTINAL VS. GASTRIC BYPASS, RANDOMIZED STUDY
Joseph A. Buckwalter, M.D.

This morning I would like to share with you some of the findings that have emerged from our clinical trial, a randomized study of jejunoileal and gastric bypass. We define morbid obesity as existing when patients have been at least two times their normal weight for five years or more. In the two years this study has been in progress we have done 19 jejunoileal and 19 gastric bypasses. All the patients were examined preoperatively by the author, the operations were performed either by the author or by residents with his assistance and have been seen postoperatively by the author.

I was in Iowa City when Dr. Mason began to do gastric bypass about ten years ago. My reaction was quite similar to many of his colleagues. That is, that this was of interest and possibly of some importance but was a kind of surgery none of us wanted anything to do with. About 3 years ago it became clear at Memorial Hospital in Chapel Hill that somebody needed to start doing these operations. More and more requests for this surgery were being received. When approached by Colin Thomas my initial reaction to this was consistent with the one I had ten years ago. Within one year my attitude toward this surgery and these patients completely changed. These are the most interesting and gratifying patients I have ever had the opportunity to operate upon and care for.

All patients had evidence that the obesity was morbid. That is, a patient who has begun to have difficulty in ambulation because of painful ankle joints or knees, who has increasing shortness of breath and other symptoms secondary to obesity and is beginning to decompensate because of overweight.

Diet failure means that the patient has been under adequate medical dietary supervision and usually has attended TOPS or Weight Watchers but has only achieved temporary weight loss. Some of our patients have joined rigidly controlled diet programs such as the Kempner Rice Diet at Duke. I don't know whether or not any of our patients have been to Boston to see George Blackburn. The usual sad story is that even under
these rigidly controlled conditions, they lose the weight but when they leave this expensive and structured environment, they quickly regain it. The morbidly obese patient has usually been rejected by society, by their families, by physicians, and by the time they reach the surgeon they are desperate people.

The third requirement in selection for morbid obesity surgery, is the attitude the patient. This operation is never "sold" at North Carolina Memorial Hospital. The expected morbidity and mortality of the operation and the fact that there is no way to guarantee the loss of the desired amount of weight, is indicated. If the patient responds by saying, "Yes, Doctor, I understand that I have three to four chances in a hundred of dying from the operation, and I understand the risks of wound infection and all the other things which you have explained to me, I want the operation," this is the primary requirement for going ahead with the surgery. The positive attitude is the reason for these patients being so appreciative, cooperative and "fun" to take care of and follow.

In the beginning I thought in terms of excluding patients with serious heart disease and other serious organic problems which would increase the morbidity and mortality of the operation. The lowest operative mortality and best weight loss occurs in younger healthy patients. My thinking has changed. I suggest that the patients who may need the operation most are older patients who may have serious organic disease, heart, renal and other. Which patients do we exclude? If the patient has a positive attitude, meets the other requirements and has an 80% chance of surviving the operation, it is done.

About half of our patients are white, half black. It is my impression our black patients have done as well as the whites. The important point is their understanding of the operation risks and expectations. During the initial interview with the patient I show them diagrams of jejunoileal and gastric bypass and explain the weight loss mechanisms in words they can understand. I point out that diarrhea must occur with jejunoileal bypass if they are to lose weight. I tell them they will have difficulty eating after gastric bypass. They must understand that with gastric bypass, if they compulsively try to eat after the operation as they did
before, they will have nausea and vomiting. The patient is admitted to the Clinical Research Unit four days before the operation for appropriate biochemical, x-ray and other studies.

I will tell you about a case unusual in our experience. The patient was a 28 year old, 275 lb, 5'1" woman beginning to have symptoms secondary to her obesity. These symptoms took the form of painful ankles which limited her walking, and shortness of breath. She had a history of menstrual irregularity. It is not our custom to routinely obtain skull films. However, they were taken and showed a double-floored pituitary sella, suggesting the possibility of a pituitary tumor.

Prior to admission to the Clinical Research Unit, patients are randomized by computer for jejunoileal or gastric bypass. Patients are randomized into 8 groups by sex, age (above and below 35 years) and weight (women above and below 285 lbs, men 325 lbs). We perform the original gastric bypass described by Dr. Mason. The jejunoileal bypass is that described by Dr. Scott.

In the first 39 patients operated upon, there was 1 death. The patient was discharged after an uncomplicated postoperative course on the 8th postoperative day following gastric bypass. She was readmitted 2 weeks later with a popliteal embolus. Following removal, she developed a second one in the brachial artery. While this was being removed she had a massive pulmonary embolus. We do not heparinize patients postoperatively. Perhaps low-dose heparin may be in order.

We have had, in the 39 patients, 3 significant wound infections, 2 with gastric and 1 with jejunoileal bypass. Patients are placed on tetracycline and neomycin 48 hour bowel preparation. One patient has had severe alkaline gastritis after gastric bypass. She was placed on Maalox and cholestyramine. Two weeks before we were planning to reoperate and convert her to a Roux-en-Y, she improved. During the 2 years we have been doing bypasses, we have seen none of the really serious complications of the jejunoileal bypass. Some of our patients have lost some hair. One patient had a classic picture of bypass enteritis which
responded to ampicillin. None of the patients have had evidence of liver failure. One patient has had urinary tract stones.

Some have reported inadequate weight loss in up to 30% of patients following jejunoileal bypass. One patient not included in the clinical trial and operated upon 2 years before coming to Chapel Hill, lost only 40 lbs. We reoperated upon her. She had 12 inches of jejunum and 15 inches of ileum. We removed an additional 3 inches of both. Again the patient did not have significant diarrhea and lost only 20 lbs during the 6 months following operation. One of our 19 patients has had minimal diarrhea with disappointing weight loss after jejunoileal bypass. No satisfactory explanation for this unpredictability of response to jejunoileal bypass has been provided.

As of March 1977, we had 18 patients, 9 that have been followed for 4-6 months and 9 followed for a year. All that can be said is that postoperative weight loss is comparable with jejunoileal and gastric bypass.

More impressive is our anecdotal experience. Individual patients have had spectacular results with both operations. A 35 year old, 325 lb truck driver lost 150 lbs in the first year after gastric bypass, enough to convert her into such an attractive female that her husband no longer would permit her to drive a truck! She regards this as a miracle. She is convinced that something magical happened in Memorial Hospital. Another 350 lb woman, 38 years old, 5'3" tall, had very severe gout. She was on allopurinol, took colchicine for acute exacerbations, was hypertensive and diabetic. This patient in 1 year after jejunoileal bypass lost 140 lbs. She has been gout-free with no exacerbations to everyone's surprise! We were concerned about the impact of this iatrogenic kwashiorkor on her gout. Her blood pressure has returned to normal and her diabetes has improved. She also had a sudden weight loss of 20 lbs at 1 year; a panniculectomy.

The most important criterion is the attitude of the patient. This applies equally to the usual patient with an expected mortality rate of 2 to 3% as well as the poorer risk patient. An example of a high risk patient was a 520 lb, 28 year old woman who had had 3 myocardial infarcts,
and terrible blood gas studies. She was admitted and scheduled for operation. She left the hospital the night before the scheduled operation because she was aware that there was a good possibility she might die on the operating table. I had told her there was 1 chance in 5. Three months later she had a jejunoileal bypass. She left the hospital 7 days later. She has lost 200 lbs in a year. Her life has been transformed. Unfortunately such a happy story does not always unfold in this type of patient. A heavier patient - no way to weigh her - was admitted to the Respiratory Intensive Care Unit. The medical people felt the only chance was an obesity operation. We scheduled her for panniculectomy. If the wound healed and she survived, a bypass would be done later. She died before the panniculectomy could be done.

Which is the better operation? Although we have not as yet observed serious morbidity with jejunoileal bypass, the variable results in terms of weight loss must be considered an argument for gastric bypass. It is obvious we all need to consider revising the Mason gastric bypass technique in view of the experience of Alden and others using the nontransection staple procedure.

What about the edentulous patient? Is this patient a candidate for jejunoileal bypass instead of gastric bypass? Are there other types of patients who still may be candidates for jejunoileal rather than gastric bypass?

It has been for me a privilege and valuable experience to attend this meeting. I take this opportunity to express a tribute to my long-time friend and esteemed colleague, Ed Mason. I am aware of the many hours and days he spent in the laboratory and operating room during the inception of the concept of gastric bypass. It was his commitment, imagination and expertise which resulted in this meeting. As we all proceed to do gastric bypasses we have reason to be grateful for his effort. Thank you, Ed, for what you have done for all of us and countless numbers of patients.
Before I get started in presenting my material on the randomized study, I would like to make some basic remarks. I, too, agree with much of what has been said over the past day and a half, and I am sure I will agree with much of what will follow. I would like to say, first of all, that this series I am going to present is a personal series so I will be using the word "I." I really object to the word "we." Often in the residents' conferences, when they start talking about "we did this" and "we did that," I remind them that Mark Twain said that "we" is a word which should be reserved for newspaper editors, crowned heads of state and people with tapeworm. So I will be using the first person very often because this is a personal series. Although fully 90% of the patients that I am going to present were operated on by the residents, that is, the residents were wielding the knife, I was there throughout the procedure and I feel obligated to be there.

Also I was impressed with the simplicity with which gastric bypass can now be done. Nevertheless, I have some ambivalence about the fact that it has been made so simple. Obesity is an area where there are many charlatans and I think we all need to recognize that. There is a man down in Georgia who has done 1,000 jejunoileal bypasses in 18 months. He must be taking all comers. I know that at least three patients he operated on for middle age spread were only about 20 lbs overweight. Now I think that the jejunoileal bypass is an unsafe operation, as you will see as my story unfolds, but the gastric bypass is not an unsafe operation if it is done technically correctly. It worries me that we are going to see a lot of patients undergoing gastric bypass who are 10, 15 or 20 lbs overweight, and that makes me a bit nervous. Nevertheless, I think that it is good to simplify operations. I am a great believer in having simple operations. I personally can't stand much longer than about six hours. I get tired. Therefore, if any operation takes longer than six hours it makes me very nervous. We still do the standard type of operations, as you'll see.
The other point I would like to emphasize is that once you start, once
you embark on this course, you will get flooded with patients that by
and large are referrals from other patients. I, too, like to avoid the
ones the psychiatrists send me. I think the psychiatrists have little
role to play in this entire endeavor. Perhaps the approach that Bill
Jewell uses with the clinical psychologists is a good one, but for the
most part psychiatrists are sending you patients who have been total
failures. I also feel very strongly that patients can help each other.
We have formed a Fatty Club just the way that we have Ostomy Clubs, The
Lost Chord, the Reach for Recovery and all the other things that are
done along those lines. This is helpful because these patients can tell
each other much better than I what the various complications are and how
the new patients are going to feel. They also can help you, by the way,
in getting your randomized studies going. The minute you tell a patient
you are going to do an operation based on the flip of a coin or the fact
that their hospital number is even or odd (which is the way we randomize
our series) or any other kind of randomization procedure, at least the
intelligent ones seem to shy away from joining with you in that particu­
lar effort. The clubs can be helpful here in adding encouragement and
enthusiasm. Therefore, I think all these things are terribly important
and are items that must be followed if you are going to obtain any kind
of scientific information from it.

I also feel, as George Blackburn was trying to point out yesterday, that
we do need to have science in this area. Doctors are now becoming the
public scapegoat for everything, from social injustice all the way down
the line. Unless we conduct ourselves with at least some sort of scien­
tific evaluation we are open to criticism and should be criticized.
That is why we should guard against the charlatans who get into this
business. I would encourage everyone not only to read the book by Dr.
Bray, which is an excellent book, but if you want to get a shorter
reading, you can read the two-part article that appeared in July and
August, 1974, by Gordon Mann about the whole obesity problem. Mann
points out that obesity per se does not necessarily shorten the patient's
life. If it is complicated, as it very often is, by diabetes, hyper­
tension, cardiac disease, pulmonary disease, etc., it will shorten the
patient's life span, but obesity per se does not. In that article he
also points out the fact that diet therapy in obesity can be stated quite succinctly, it doesn’t work. The reason it doesn’t work is because these people do diet and lose weight but then they put it all back on again. It becomes a recurrent cycle until the point where they come to you for some kind of help. Mann further addresses the problem of the quacks who get into this business and is very concerned about the fact that there are a lot of quacks in the business of obesity. That’s why there are so many books currently available on various diets that in paperback editions have sold millions.

There is another reason. I tell the students quite often that there must be a reason why the chiropractors still stay around. The reason is because the chiropractors sit down and talk to their patients; something many doctors won’t do. If we would or could spend more time talking to our patients, convincing a patient, for example, that the only thing he needs to do is stop eating so much, stop drinking so much, stop smoking so much and get a little exercise, we probably would have a healthier nation. We would probably have a public that was happier with the medical profession. Unfortunately I don’t think that’s going to happen. Therefore, we don’t have an ideal situation. I would agree that obese people who meet the criteria, who come to you having almost invariably been overweight all their lives, having been on 10, 15 or 20 diets and having a positive attitude should be considered for this procedure.

I am sure you are all well aware of the rationale of these two procedures. Basically the gastric bypass procedure is a form of enforced and permanent starvation, that is, if the operation is done correctly. Jejunoileal bypass, on the other hand, is controlled malabsorption. That is to say, it is hopefully controlled, but sometimes it is uncontrolled and that is when you get into trouble. In 1973 I started doing patients with morbid obesity. As many other speakers ahead of me have indicated, the minute that you start doing this operation you begin to see the fat people crawling out of the woodwork. It does involve an exponential curve. You go through a learning process, get somewhat disturbed and back off from doing them. Then you take it up again because some friend of yours wants you to do one. The patient has a spectacular result so you get a little more enthusiastic and pretty soon it’s exponential.
I am going to talk today about the experience that we have had since January, 1974. When I began, I had known about Dr. Mason's procedure. I had also heard of the criticisms or comments that had been made when he first presented the material, indicating they were concerned about stomal ulcer, the fact that it was a technically difficult operation, and the hundred other criticisms that were made of it. It's very easy to criticize, as I'm sure you're all aware. I did only the jejunoileal bypasses in the first 30 patients or so, but then I said, well, as long as I'm going to start doing this, I might as well do it the scientific way. Therefore, we started a randomized study in January, 1974. By January, 1977, we had 32 patients in the gastric bypass group and 27 in the jejunoileal bypass group with three years of followup. I made an arbitrary decision that I was going to get 50 in each group and I can report that as of this time I already have 50 in each group. If it is any consolation to those of you who are protagonists of the gastric bypass, I can report that from now on we are only going to do the gastric bypass in my institution.

The operation that I used for the jejunoileal bypass procedure was the operation that had been described by Scott except for the fact that I used 30 cm of jejunum, 12 inches, and 25 cm of ileum, 10 inches, with an end-to-end anastomosis. I have drained all of the bypassed segments into the sigmoid colon and I recognize all the arguments pro and con as to where you should drain. I personally don't think it makes very much difference, it just seemed easiest to put it there. I have not been terribly unhappy with the operation from the standpoint of getting pneumocystis or from a lot of gas in the bypassed segment. I really don't think that's been a major problem with the procedure.

The first seven patients that we did in the gastric bypass group were done with a retrocolic loop gastroenterostomy. I was impressed by the fact that the patients would overindulge and vomit. We finally learned how to control it. But I also had two patients who had vomiting intradigestively. One patient in particular used to wake up at two o'clock in the morning and vomit pure bile. She also had a great deal of heartburn. I finally did a fundoplication on that patient even though she had had a gastric bypass about a year earlier. Fortunately, it worked.
She got rid of her heartburn, although she hasn't gotten rid of all her other problems. Nevertheless, I decided that I was having too much problem with the bilious vomiting. Therefore, I went to the Roux-en-Y technic with a retrocolic type of anastomosis. One of the nice things about it is that you don't have to drag that gastric pouch down below the transverse mesocolon. You can just tack that loop of jejunum right up to the mesocolon where it lies. I have had no problem with this in terms of either alkaline gastritis or stomal ulcers.

I've been doing this particular procedure since the fall of 1974, and I think it is a perfectly safe operation. The reason it is safe is twofold: 1) there aren't too many parietal cells in the fundic pouch and 2) the gastric remnant or the bypassed segment does produce acid so that the gastrin mechanism is shut off effectively after the patient eats. Consequently, there isn't any hypersecretion from that little 5% pouch even if there should be enough parietal cells. I did not, until recently, start measuring the size of the pouch but I do think it is a good idea to measure the volume that the pouch will accept along with the pressure measurement, as Dr. Terry has indicated.

There are two other points that I would like to make before going on. One is the fact that we are basically in trouble because of the lay concept of instant repair. Everybody in this country believes they can over-indulge in whatever they wish and then they come to the body shop known as a hospital and we mechanics put it back together again so that they can be perfectly normal. As Harry Schwartzmann once said, "The medical profession will always be in trouble because what the American public wants is immediate cure at low cost to old age with full sexual prowess," and that's an impossibility. The other point I would like to address concerns the various things we do surgically around the operation: 1) The use of parenteral antibiotics. Previously, I had used them only in the jejunoileal bypass patient and then because we opened up the colon, but because of the high incidence of infection in the gastric bypass group, I started using the antibiotics with them also. I do use oral antibiotics on the patients undergoing jejunoileal bypass procedures but we do not use oral antibiotics on the patients undergoing the gastric bypass. I use parenteral antibiotics the night before and the day of
surgery and then stop them. 2) The use of mini-dose heparin. I do use low-dose heparin on all these patients. We have a regular protocol that takes about five working days to complete. Therefore, the patients are in the hospital for a week prior to their operation and as soon as they are admitted they are put on low-dose heparin. Although in non-obese patients, I use low-dose heparin at a rate of 5,000 units every 12 hours, in these patients, because of their size, I use 5,000 units every 8 hours. The patients do not bleed excessively. I really have not had any problems with low-dose heparin in my total experience except for two patients on whom I did a Whipple procedure. Interestingly enough when we discovered that they were bleeding intra-abdominally postoperatively their coagulogram showed obvious total heparinization even though they were on low-dose heparin. Aside from this, I have been rather pleased with low-dose heparin. I think it is something that we need to look at very critically. I am sure many of you are well aware, there are now at least three lawsuits that are on the basis of pulmonary embolus and one death postoperatively due to a pulmonary embolus where low-dose heparin was not used. 3) A third feature of it is that we do concomitant or simultaneous procedures with the bypass operation, taking out the gallbladder, tying tubes, taking down the ovaries, clipping the vena cava, etc. I have clipped the vena cava in three patients who had documented pulmonary embolus prior to having the operation for morbid obesity, and again have had no particular problem with that. Naturally, I too, stress the early ambulation and respiratory support. Extubation is carried out as soon as the patients meet the criteria of their respiratory mechanics.

The criteria for the inclusion in this study is that they be more than 50 kg over their ideal weight. We do a whole battery of endocrine studies but like Bill Jewell I suspect we are going to stop doing all that material of the patients once we stop doing the randomized series because we haven't found anything positive in them. We do a whole host of absorption tests and we have not found absorptive deficits, as you might expect we would, but we don't find anybody with supervalues either. They just overeat, it isn't that their bowel does such an excellent job.
I prefer to have patients who have concomitant conditions, diabetes, hypertension, hyperlipidemia or whatever, because then I feel as if I'm doing something more than cosmetic operations. Nevertheless, I have a number of patients who have not had anything more than their morbid obesity. I still get psychiatric evaluations. However, I just received a memorandum before I left from the Psychiatric Department saying that their Consultation Service will no longer see the patients. As a matter of fact, I feel that those of us who do this operation are really the patient's psychiatrist anyway. As many of you know the patients will call you up about everything, their alcoholic husband or their kid who is on drugs or the fact that they can't get it up or get it down or whatever.

Of course, the patient has to be willing to participate in the protocol. We have got to have informed consent. We go through the whole rigmarole with a written document, the first statement of which is in capital letters, "THIS OPERATION MAY KILL YOU," because I think the patients ought to understand that this isn't a picnic that they're going on, it's a real risk.

We have had an interesting distribution in terms of males in that we have had quite a number of males. However, when the gastric group is broken up for some reason there were many more females. This explains why the average preoperative weight and height are a little bit lower in the gastric bypass group than in the jejunoileal. Nevertheless, they are all sizable candidates. The smallest patient I've operated on weighed 223 lbs, that's a 110 kg woman. She was 4' 9" tall, so I thought she fit the criteria. She was just a little butterball instead of a big butterball. I have done some patients over the age of 50. The oldest patient I've done actually was 62. She was a severe diabetic and had had two myocardial infarctions and had hypertension. I really felt as if she needed the operation as a therapeutic measure more than anything else. I was gratified to find that she did very well.

In regard to the concomitant conditions many of them had had hypertension. This has been hypertension that has been documented by arterial lines, on the operating table, and it is true arterial hypertension. Many of
them have been pickwickian and several have had cardiac disease. Many have also been diabetics. By diabetes I mean patients who either have overt diabetes, either taking pills or insulin, or patients whose glucose tolerance curve is abnormal when they come into the hospital. Several of the patients have had hyperlipidemia.

Now we can look at the complications. There have been no operative deaths. In fact, in the somewhat more than 120 procedures that I have done since 1973 I have only lost one patient. She died from a pulmonary embolus on her 15th postoperative day. That patient, by the way, had an error in the medication in that the low-dose heparin was stopped on her eighth postoperative day and none of us picked it up.

There have been two late deaths that I think are very interesting. One late death in the jejunoileal group was in a man who lost 250 lbs in ten months. First of all, he came back every six months and I kept yapping at him about the fact that he was not eating properly because he was losing more than 15 lbs a month. I think 15 is an outside limit, I prefer having them lose only about 10 lbs a month. I tried to explain to him, as I do to all of the patients, that they must keep their nutrition up and their oral intake must be adequate. This is particularly true in the jejunoileal group because if you add, on top of controlled malabsorption, an exogenous nutritional deficit the patient surely will get into liver trouble, and he did. At ten months he absolutely refused to let me put it back together again, even though it was obvious to me and everybody else that he was not faring well. At 12 months he didn't keep his appointment at the clinic and I called to find out that he had been admitted to another hospital jaundiced and in renal failure and kidney failure. He did, in fact, die just one year after he had had the operation.

The patient who died in the gastric group represents one of the vitamin deficiencies to which Dr. Jewell has already alluded. This was a patient who was three months past her gastric bypass operation. She came to the clinic and just said she wasn't feeling very well. She didn't have any specific complaints. She still had a bit of vomiting when she overate. She did not have any diarrhea. She was losing weight satisfactorily, in
fact, in three months she lost about 56 lbs. But we went ahead and admitted her and the only thing abnormal we could find was that her serum potassium on admission was 2.9. We treated her with glucose-containing materials and high amino acids and she seemed to straighten out and was eating on the third hospital day. She suddenly collapsed on the bathroom floor and when the nurses found her she was dead. We did get an autopsy, thinking that surely she had had a pulmonary embolus. However, she did not have a pulmonary embolus and I suspect that this represents a vitamin deficiency that we perhaps just kicked over the rest of the way by giving her glucose-containing fluids when she first came in.

The rehospitalization rate is interesting. Four patients have been rehospitalized from the gastric group. Two of the patients are ones who had leaks and we will come to that later. There was one other patient in addition to the one who died. The ten patients who were rehospitalized in the jejunoileal group represent a variety of things. One of the patients was rehospitalized about seven times for hypokalemia in the 18 months that she had had the jejunoileal bypass in place. Many of the other rehospitalizations in the jejunoileal group are patients who had undergone subsequent cholecystectomy. I personally believe that the jejunoileal bypass is a lithogenic operation and that almost 100% of these patients will eventually develop gallstones. One patient in the gastric group has had a reanastomosis and that is one of the patients who leaked. She did not require a second operation as the leak was not a major leak, but she developed so much fibrosis around the anastomosis that it eventually got down to a measurable 2 mm by endoscopy. Although she was happy because she went from 412 lbs down to a svelte 119 lbs, I wasn't particularly happy because she vomited about two or three times a day. Therefore, we took her down and interestingly enough she is back up to 300 lbs, so we're probably going to have to go back in and do something. There was one jejunoileal bypass that we took down in a patient who had a combination of electrolyte imbalance and beginning liver failure. I convinced her that she ought to be redone and she accepted this. We've had our complications and I think it's important to recognize that this procedure is not without complications. Nevertheless, in the last 25 patients we have only had one wound infection and
that was a jejunoileal bypass patient. Wound infection in these patients is disastrous. Ever since we put the gastric group on antibiotics, we've only had one infection. We do, by the way, transverse incisions in these patients, both the gastric and the jejunoileal group. I believe you can get perfectly adequate visualization with a transverse incision. I don't have the four corner retractor that Dr. Gomez has, but that looks like it would be interesting because then maybe I wouldn't get so tired.

We have had dehiscence in each group. We have had two anastomotic leaks. In the second anastomotic leak the patient did have to be reoperated upon. The rapid pulse business is true, it does work and it gives you an idea that something is wrong. Unfortunately, we weren't smart enough to tumble to the fact and we actually didn't get back in until five days down the pike. This was in a patient who had a Roux-Y anastomosis. One of the nice things about the Roux-Y anastomosis is that the leak doesn't probably present as much of a problem as it does when you've got a loop. This is because you don't have the bile and pancreatic juice coming out, you simply have saliva and gastric juice. In this patient we were able to close the leak and put a gastrostomy tube into the bypassed segment and actually started feeding that patient by her own GI tract on the third post closure day of the leak and she did very well.

We have had a couple of subphrenic abscesses in the gastric group and one in the jejunoileal. Urinary tract infection has been a significant problem and interestingly enough all of them have been females. We have had no pulmonary embolus in either group that we have recognized. They may have been there but we certainly haven't recognized that, that is, in the early stage. We have had some in the late stage and I'll get to that in a minute. The three other complications in the gastric group are splenectomies. That is a part of this operation and must be recognized as such.

In regards to the late surgical complications, nausea and vomiting is an integral part of the gastric bypass, and I think the patient should know that. Basically what we are doing is helping them in their behavioral
training. We are teaching them that they just can't overeat. Diarrhea, of course, is a major complication of the jejunoileal bypass. Fifteen of our patients had severe diarrhea. We do the same as Bill Jewell. We expect diarrhea in all of these patients, we only call it a complication when they have more than six bowel movements a day and require medication.

There are 20 patients on medication in the jejunoileal bypass group. This is one of the reasons why I think this operation will probably go by the wayside. Even though you can get the diarrhea under control, it often requires a very significant amount and type of medication. Also I think that that is perhaps another indication for gastric bypass. Although I don't like the gastric bypass operation necessarily in people who aren't too smart and don't understand what we are trying to do, let me tell you that the jejunoileal bypass in a dumb person is a disaster. They simply don't understand the disease process. They also don't understand the medication needs. If they really are going to end up having to take a lot of medication, lack of intelligence is really a contraindication for doing the jejunoileal bypass.

As far as kidney stones are concerned, it is interesting that we have not seen a single kidney stone in a gastric bypass patient. We have seen it in 4 of 27 in the jejunoileal bypass patients. I think that is because the jejunoileal bypass group does represent a metabolic disorder that you have created on top of a metabolic disorder that already existed. In addition they are losing weight and can go into periods of relative dehydration, depending upon how much diarrhea they have. On the other hand the gastric bypass patients, even though they may not be able to take in solids without knowing about it, seem to be able to handle liquids fairly well. Consequently they keep their water balance up, their urine flow high and therefore do not get the concentrated urine that produces kidney stones.

We have had some late pulmonary emboli. These have all occurred in patients who have gone home and languished in the bed. Therefore, we do try and get them into vigorous ambulation exercise programs as soon as possible. In fact, we start it the day after their operation or on the night of their operation, and continue running them for the rest of
their lives, which I think is terribly important. We have done 10 reoperations for various reasons such as cholecystectomy and hernia repair.

We have taken some liver specimens intraoperatively and then later at one year postoperatively. In the jejunoileal bypass group the specimens look even worse at one year. If Dr. Drennick were in the audience he would clearly point out that at one year they look terrible while at two years they begin to come around to be halfway decent. On the other hand, in the gastric bypass group the liver is really getting to be pretty normal at one year postoperatively. We haven't seen the liver problems in the gastric bypass group that are seen in the patients with jejunoileal bypass.

In regard to weight loss, there are 18 gastrics and 22 jejunoileal bypass patients in this series that have had more than a year of followup. The gastric bypass group had a mean weight loss of 51 kg at one year, while the jejunoileal group had a mean loss of 57.9 kg. This is not significantly different. These patients do lose weight at the same rate, as Dr. Jewell indicated. In summary I can say that gastric bypass is technically more difficult although with the new modifications that statement may have to be erased. I do think that their anastomotic leak will still be at a somewhat higher rate than the jejunoileal group because of the fact that you are using the stomach as part of the anastomosis. On the other hand, the list of disadvantages of the jejunoileal bypass is tremendous - malabsorption, medication and the rehospitalization rates are extremely high. I again say that I personally believe that the jejunoileal bypass operation is lithogenic and all these patients will eventually develop cholelithiasis. And, of course, liver disease is there as a specter of death continually. In conclusion I would say that all patients operated upon for morbid obesity require close supervision postoperatively. That is particularly true of the jejunoileal group, and considering all factors, the gastric bypass is a more satisfactory operation for morbid obesity.
HIATUS HERNIA, REVISIONS AND RELATED PROBLEMS

Edward E. Mason, M.D.

The problem of hiatus hernia and reflux is due to certain incompatibilities of gastric bypass and reflux operations. There are competing objectives. The antireflux operations, Nissen fundoplication, Hill, Belsey, and so forth, have to do with wrapping the esophagus and forming a valve. This requires some redundancy of the stomach. The objective of gastric bypass is to make the upper segment as small as possible, to take down the fundus, to get rid of the angle of His, to do things which seemingly would be productive of reflux. Certainly it does not leave a redundant pouch to wrap around the esophagus.

Another complication exists in that a successful antireflux operation by definition prevents vomiting and belching. A successful gastric bypass with a gastroenterostomy requires a small pouch and a small stoma which slows up emptying. The obvious potential hazard here is a closed segment. The area of the lesser curvature with the upper branches of the left gastric artery must be preserved. If the stomach is transected this is the blood supply to that upper segment. If you interfere with that blood supply the upper segment will undergo necrosis. I am very concerned about some of the pictures that were shown about the technique yesterday. I wonder if they truly depict what the surgeons are doing. I am worried about devascularizing that upper segment. It may be that if the stomach is stapled in continuity there is enough blood flow through the staples to feed the upper segment even after its complete devascularization. However, I wouldn't depend on it and I don't think in that regard that you need to go that high on the lesser curvature. I think maybe the speakers yesterday are leaving a little bit more than those illustrations suggested.

To get back to the competing objectives in gastric bypass, you are trying to leave blood supply on the upper aspect of the lesser curvature. At the same time the antireflux procedure requires pulling down on bites of tissue from the sling of Helvitius to recreate the angle of His. The fundoplication is around the esophagus. It should not be around the lesser curvature but nevertheless you are working in this area and there
is a real risk of interfering with the blood supply. In spite of this a Nissen fundoplication can be performed if the patient has demonstrable reflux. The TA-90 stapler can be applied and the volume measured. The volume can be made less than 100 ml and staples can be discharged. The gastroenterostomy can be set up and in this situation the stoma can be made somewhat larger than the usual 12 mm, and perhaps even as large as 25 mm. I wouldn't dare do a Nissen fundoplication and then make a stoma that was quite as tight as Dr. Gomez or I normally make it. I think you have to compromise a little bit if you are going to combine an antireflux operation with gastric bypass.

There is an alternative. When I was at the University of Minnesota, Dr. Wangensteen was very excited about the treatment of esophagitis due to acid peptic disease. Esophagitis, he said, was just like duodenal ulcer. It is caused by too much acid and pepsin that, in some patients, goes up instead of down. Although the patient would develop acid peptic esophagitis rather than duodenal ulcer the treatment was the same; a distal gastric resection. Over the years, however, we began to learn, and we are learning this now about gastric bypass, that some patients still have reflux, only now they reflux duodenal contents and bile. Duodenal contents can be just as devastating as acid and pepsin.

Dr. John Thomas, who was a resident with us, did some work using a model that Fletcher Miller (who is another colleague of ours from the Minnesota days) used for the creation of esophagitis. He used the Heyrovsky-Grondahl procedure which is like an anastomosis between the fundus and esophagus. It is a pyloroplasty on the cardia and the purpose of it was to create reflux and esophagitis. You don't do this in people. This was an experimental model. Then to be absolutely sure that there was reflux, Fletcher incorporated a hiatus hernia. He enlarged the diaphragmatic opening and moved the stomach up into the chest and then he studied various procedures to see their effectiveness in the treatment of reflux esophagitis. I had John Thomas perform a series of these procedures and we had three groups. There was the model with reflux; another group that had the model plus gastric bypass with the standard loop gastroenterostomy; and finally, a third group that had a Roux-en-Y gastroenterostomy.
When John brought his photographs of the open specimens of the esophagus and showed them to me as a "blind" observer, I could pick out the animals that had had the different procedures. The control dogs had small ulcers which we thought were acid peptic ulcers. The animals with reflux and a loop gastroenterostomy with gastric bypass had inflammation of the esophagus. It was mild but they didn't have the ulcers and it looked like something that had been reported in the literature as due to bile reflux. The Roux-en-Y gastric bypass animals had a clean esophagus. This bit of information suggested that the Roux-en-Y probably would be a desirable procedure if it did not increase the secretion of acid and interfere too much with the secretion of pancreatic juices. This was never published because John went off to take a cardiovascular-thoracic residency and took the information with him. I can't seem to get him to write it up and submit it but maybe sometime he will. Harrington and Moody (Total duodenal diversion for treatment of reflux esophagitis uncontrolled by repeated antireflux procedures, Ann. Surg. 183:636, 1976) are using this approach. The Roux-en-Y drainage of the stomach is a very good way to treat reflux. You can build a dam like the Nissen fundoplication or you can take the other approach and drain things away so there is nothing there that will reflux.

The experience with gastric bypass is, I think, following the experience with gastric resection as you might expect. We have overlooked the problem of reflux. We are assigning many of the problems of our patients, the vomiting for example, to things that perhaps are not really the cause. Maybe the patient is not responsible for all of the early symptoms which do improve with time. Maybe a lot of our patients really do have an afferent loop syndrome. Certainly on fluoroscopic examination, you see the barium move into both the afferent and efferent loops. If the afferent loop has a good peristaltic wave that shoots the barium back up and it doesn't get around the corner to the efferent loop, it squirts up into the stomach and the patient may vomit. This can also happen after eating. We need to talk to our patients further to find out more about the details of their symptoms. We should use a lot more esophagoscopy and we ought to get more information preoperatively about signs and symptoms of reflux. Then, if we know that a patient has an incompetent cardia and esophagitis with or without hiatus hernia, we can approach
this in a different way. We can do a Roux-en-Y, which I believe would be preferable, while retaining the ideals of a good gastric bypass procedure. In this fashion the small pouch with the moderately small stoma can be made so that it won't have any duodenal contents to reflux.

I am intrigued by the fact that Ward Griffen's average weight loss as given in kilograms is better than the average weight loss that we or that John Alden and Jim Seay have had. Maybe we can even afford to have a little bit bigger gastroenterostomy stoma with the addition of the Roux-en-Y. As you can see, there is still work to be done. In fact, there is no end to the work. We are still working on the treatment of duodenal ulcer and we have just gotten started on the treatment of obesity. I think there may be a place for routine use of the Roux-en-Y.

I am concerned about cancer in the stomach. I mentioned this to Dr. Holmgren who is here from Stockholm. I have seen some reports, one of these comes from his area, that indicate an incidence of carcinoma of the stomach as high as 14% in patients 15 years post Billroth II type procedures. These patients also have bile reflux and changes in the gastric mucosa adjacent to the gastroenterostomy. There may be a problem here. There is experimental evidence to support the hypothesis that bile reflux breaks down the mucosal barrier to carcinogens. The combination of reflux of bile contents into the adjacent gastric mucosa plus carcinogens will increase the risk of carcinoma of the stomach. We are all probably ingesting carcinogens. The addition of some break down in the defense mechanism such as occurs in the immuno incompetence produced by intestinal bypass in a general way or perhaps the local effect in the stomach just adjacent to the gastroenterostomy in gastric bypass are things we need to be concerned about. It might be well to follow closely our patients who are ten years post gastric bypass with frequent endoscopy to see if there are any changes and to be able to pick up those changes and do something about them before carcinoma develops. It might be well to quit doing loop gastroenterostomies for a number of reasons and switch over to the Roux-en-Y type of procedure. It would take a little more time but I doubt that it would be any more risky although it is a little more complicated. These are, of course, speculations but I think we need to speculate. Generally speaking, if you can think of something that might happen, it usually does happen.
I would like to turn now to the subject of revisions. There are at least three general reasons for failure to lose weight after gastric bypass. (1) The operation wasn't done properly, or (2) the operation was performed properly but through chronic stretching of the pouch it has increased in size or the stoma has increased in size or both, or (3), the operation was performed properly and has remained as it was but the patient is such an aggressive consumer that he really wasn't a good candidate for this kind of treatment in the first place. I am not quite sure about that third one. I think it is perfectly obvious that if you completely close off the gastroenterostomy the patient will lose weight. Going from that reasoning there must be some size opening like the carburetor adjustment on your car that will be just right for mountain driving or whatever kind of driving a person needs to do. In addition, there may be combinations of those problems. Revisions can obviously be made of the pouch or of the stoma or both. The pouch can be reduced in size by freeing up the greater curvature if it was not adequately freed up at the first operation. You can use a GIA stapler and take a nick out of the pouch or a big chunk out of the pouch.

You can measure the volume; and I might just put in a plug for this right now. Even if you think you can't make the volume any smaller, even if you have done an operation that looks as small as it could possibly be and you wouldn't do anything anyway, I think at this stage of the game you have an obligation to measure the volume. I am as guilty as anyone about this. We have the biggest series of unmeasured volumes in the country but it is not right. We need to know. If we have a patient who comes to us with a big pouch we need to know whether we left it big or whether it got big through stretching. It is like the problem with reflux. You operate on a patient for reflux and do a fundoplication but the patient comes back with recurrence of the reflux. Did they develop a recurrence or did you leave them with reflux when they left the operating room. We need to know the volume. We need to get all kinds of information.

You can take out part of the pouch if you measure the volume at the time of revision. If it is less than 100 ml, maybe 60 ml, and you can see that the pouch just can't be too big, perhaps there is a big stoma. You
can free up the gastroenterostomy, cut in on one side of it and do just the reverse of a pyloroplasty; instead of making it bigger you make it smaller. You cut in on one side and extend the Hofmeister, so to speak, and you can reduce it to a stoma size of 9 mm. I never make a 9 mm stoma at the first operation. I am afraid to because of the edema that usually follows in my experience with my sutured anastomoses. It too often causes obstruction and delay in discharge from the hospital. It may be that Dr. Gomez can make a 9 mm stoma with the staples by putting a Hurst dilator or a certain known size tube through and squeezing down to the TA-30. You can end up with a stoma that is small and not have any difficulty because there is no variation here. The standard deviation of this procedure will be so slight that he won't have any problems. I think there may be some real advantages to the staple types of anastomoses in that regard. You may have better control but certainly on revision you can cut in on one side and narrow the stoma to 9 mm for the simple reason that when you finish, the lining of that stoma is all healed. There is no new fresh tissue turned in. The fresh tissue is out at the side where you cut into the gastroenterostomy and pulled it together. You can do this with suture techniques or you can do it as Ken Printen does by putting the Hurst dilator down and simply snugging up the redundant area of the gastroenterostomy with the GIA stapler.

You can revise the pouch alone, the stoma alone, you can revise both of them, but the chances of success in achieving a desired weight loss are less for a revision than they are for the first go around. This is because you have eliminated all of the best candidates for gastric bypass and you are now working with a mixture of poor operations plus poor candidates. Also, many of these patients have lost some of their weight so they are no longer going to be able to lose as much weight after the revision as they could have lost after the first operation.

The results are somewhat discouraging. You see some patients who have fantastic results following a revision, but this usually is in a patient where you did a very bad job the first time. It is absolutely astonishing how big a pouch I have left in some of these patients. I cannot believe that I would ever have left such a big pouch. I think maybe with the
kind of exposure that Dr. Gomez showed us not only are the chances of success increased but the risk is reduced.

You can't dissect in those scarred areas freeing up the pouch without risking some injury to the stomach that you may not recognize. It is possible to avulse a suture and because of the irregular scar tissue around where the suture was on the stomach wall, you can overlook a perforation. This is like operating on somebody with diverticulosis. You can cut out the tic and wonder why you have trouble with your anastomosis. Well, you simply have left a hole there.

Because of the increased risks of revisions it might be that we should admit failure in some morbidly obese patients and simply emphasize behavior modification, protein augmented fasting, and some of the things that we were admonished to do earlier. Perhaps we surgeons should be doing more before we operate on some of these patients. Let me mention one other miscellaneous item. That is the gastroplasty. A number of you have asked me about the gastroplasty. We performed 56 gastroplasties in 1971. This was an operation in which we freed everything up. Then we started at the lesser curvature and transected the stomach, leaving a channel on the greater curvature. This is analogous to the Billroth I, because the upper pouch is simply emptying into the lower pouch. Patients with Billroth I did not lose weight but patients with Billroth II did. That is why we designed gastric bypass to simulate a Billroth II. We should have known that if we tried to simulate a Billroth I that the weight loss would not be as good, and it was not. I am not sure that it was entirely because of the postprandial dissociation between passage of food and passage of digestive enzymes and bile which is one of the explanations. It may be that the upper pouch was too big, the stoma was too big, they stretched too much, and that it could still be a good operation.

I am continually asked why I haven't tried this or that manifestation. My answer is that I don't want to do it because I haven't done it in the animal laboratory and I seem never to have time to get these things done. At this meeting you have heard of some 800 patients who have had their stomachs stapled in continuity without revision. There, consequently, is no necessity any longer to wait for dog work to find out if
that is a good procedure. It has been demonstrated to be a safe and effective procedure. There is some question as to whether stapling will pull out and allow communication with the distal stomach. This has occurred in a few patients.

Another question has come up a number of times about wrapping the upper pouch and going back to the gastroplasty. I think it is quite obvious that if you wrap that upper segment with Marlex mesh, put some underwear on it and fix it so it can't enlarge and so the stoma can't enlarge, that you get away from some of the things that may have defeated us in the use of gastroplasty. I hope that Dr. Granek, and perhaps others who are studying this, will find out about it. That might be a way of simplifying things.

With respect to individual cases we had a patient that didn't lose enough weight. She also had a hiatus hernia before she had her gastric bypass. We tightened up the crura and did the gastric bypass. She did not lose enough weight. She had recurrence of her hernia and last week we operated upon her and measured the pouch and it was 600 ml. Therefore, we freed everything up and wrapped this pouch around her esophagus. A very loose wrap but a big wrap with several layers. The volume of that pouch is now 60 cc and she no longer has any symptoms of reflux. It is early, I admit, but I think this will solve both of her problems. It is possible to do this but, believe me, it is a risky business and I don't think it is the best way to go. I think the Roux-en-Y way is better.

I have been quite concerned about doing Roux-en-Ys the way Ward Griffen does them. You notice on Ward's picture, he makes the enteroenterostomy on the right side of the long loop. However, a lot of the problems with afferent loop syndrome, duodenal stump blowout, and all sorts of things related to obstruction of the afferent limb have been due to peristalsis which ends up pushing that afferent limb posteriorly. It can kink the afferent limb and obstruct it. I think it probably is safer to bring the afferent limb around back of the long limb and bring it down to the side. I have been making functional end-to-side anastomosis by cutting
the corner off the afferent limb and using the GIA stapler, making a broad anastomosis.

Harrington had a patient who had a partial gastric resection with a Billroth I, vagotomy and he had reflux esophagitis. Harrington treated this by bypassing the duodenum with a Roux-en-Y. It looks like a gastric bypass the way he does them except that he has too big a pouch for a fat patient.

With regard to the revisions, out of 593 gastric bypasses that had been done by April, 1977, 74 (12.5%) of patients have had 81 revisions. When the stoma alone was revised, the initial mean weight went from 141 kg at gastric bypass, to 121 kg at the time of revision and to 110 kg at one year post revision. The total weight loss in one year was still only 31 kg. That is with both operations, only 31 kg on the average. That is not very satisfying. There are some patients who had very good results from revision but there are too many of them that it didn't seem to help. For those that had revision of both pouch and stoma, the initial weight was 160. Their total weight loss was 46 kg. A little better, but there were still a lot of failures.
You have been treated to a lot of facts and science this morning and I have another lecture on fiction. I get to talk about vitamin deficiencies after gastric bypass. You have already heard everyone proclaim there aren't any metabolic complications after gastric bypass so I won't be talking very long.

What we are going to do is show you the results or partial results of the questionnaire we send to people dealing with the kinds of things they eat now that they didn't like prior to surgery. We will show you our experience on four patients who did develop what we feel is definitely a neuropathy based on vitamin deficiency and hopefully some input and answers about what really happened to these patients.

I think before I start doing anything, the business of eating in the postoperative gastric bypass patient demands that you can't do what the popular song nowadays does and that is take the money and run. You have to follow the patients and, more importantly, you have to spend a fair amount of time talking with them preoperatively and postoperatively not about how much weight they can lose but how much and what kinds of things they can expect to eat. A lot of these patients, as a matter of fact probably all of these patients, who undergo a gastric bypass are going to go home and immediately challenge the doctor. They will go home, sit down and will try to drink a whole six pack of beer or try to eat half of a pizza or whatever they are used to eating. They are going to throw up and get sick. I think you might just as well accept that when you discharge the patient. The counseling that you give the patient preoperatively and postoperatively should allow that patient to say, "OK, fine. The doctor was right. Now, let's get back to normal and see how much and just what types of food I can eat." Most patients learn this and learn it very quickly. Whether they accept it or not is a different story. But they do learn their capacity.

In the postoperative period a fair number of folks eat a fair amount of "junk" throughout the day. However, they have cut down considerably
compared to what they used to be able to eat. This represents maybe a
couple of mouthfuls of popcorn as opposed to three bowls every night.
In regard to the vegetables again we have people eating more of this
group than we found in the preoperative evaluations. We have some folks
who do not eat any fruits and vegetables, and there are some folks who
don't eat meat. "Never again," according to them, and that is the
truth. There are some folks who never tolerate meat after gastric
bypass. They do fairly well with fish and cottage cheese but they just
can't keep any kind of meat down. This is a consideration for which we
have counseled them preoperatively and postoperatively. With respect to
the nonalcoholic beverage group, patients continue to drink and are able
to maintain adequate fluid intake. This has been demonstrated by Dr.
Griffen in his slides. The patients do not get into urologic difficul-
ties, with either oliguria or stones after gastric bypass. This is
because they are able to drink and most of the time that is the way they
get on immediately after their discharge from the hospital. There is a
significant group of people (about 20%) who can never tolerate milk, ice
cream, butter or any dairy products after gastric bypass. Sometimes it
makes them sick to look at it. Most of the time it makes them sick to
swallow it, but the patients really do not get malnourished.

We can't demonstrate serious nutritional difficulties after a gastric
bypass in spite of the fact that some of the people can't tolerate
various types of food. We did liver function studies on all of these
people before hand. You will note that they are almost always abnormal
preoperatively. As the patients are able to tolerate food, nothing
really changes very much. There is no difference in bilirubin despite
what everyone might have said and certainly there is no difference in
the SGOT.

Some people are never hungry preoperatively, they eat from habit. This
can become a problem. If you combine a patient who is never hungry
because he is eating from habit alone with a patient who, when he does
eat from habit alone, overeats continually and vomits, then you get a
patient who is much the same as the intestinal bypass patient; one who
everytime he eats has diarrhea and therefore decides not to eat. This
patient will react in a similar way to gastric bypass. Somewhere stuck
in the back of his head is the idea that he must eat X-amount of food; he eats it from habit three, four or five times a day and he vomits. After a while he shuts off all together, perhaps like anorexia nervosa, I really don't know, but some people just stop eating all together.

As I mentioned earlier there are some folks who really never do recognize their capacity. This has not been debilitating in any of our patients. Such patients are like my secretary's husband. He used to weigh 368 lbs, now he weighs 210 lbs. He is 6 foot 5 and all muscle. He eats six sandwiches for lunch and goes out and vomits and comes back and then drinks a couple of beers and feels much better. He is working every day but he also vomits every day. There is no question. The vomiting doesn't seem to deter him from eating; it never did and it never will.

I think that in four of our patients we have been able to document nutritional deficiencies of one type or another. We haven't found any others that we can measure except as I mentioned yesterday, the hemoglobins in pregnant females. Three of the 13 ladies who became pregnant after gastric bypass did require additional supplements of iron beyond what an obstetrician would normally give a pregnant woman in order to maintain her hemoglobin in the 12 to 13 range.

As far as the four patients who developed peripheral neuropathy are concerned, all of these patients developed exactly the same type of picture you would expect from beriberi. They presented here in various stages of neuritis, with painful soles, patchy loss of sensation, stocking glove type of loss of sensation and areas of hypoesthesia along both upper and lower extremities. In all cases we felt that this was primarily a vitamin deficiency but the reason behind it was a little obscure until we sat down and talked with the patients. We treated all four of them, by the way, with thiamine and all four patients recovered totally and are now doing well.

The interesting thing is why did four of 600 patients develop a neuritis. The reason was simple, in these four patients at any rate. One lady went home and tried to eat as much as she ate preop and began to vomit. That scared her so badly that she didn't eat at all. She had zero
intake. The others were chronic overeaters and chronic three to four times a day vomiters. They were brought back into the hospital and received a little bit more dietary counseling in regard to the volume of things that they could eat. The necessity to chew food properly in order for it to go through that small anastomosis was reinforced on the patients. The kinds of food they probably ought to stay away from were also stressed. In several instances drip feedings were given until they felt they were significantly informed to try food on their own.

This serves to underscore the fact that first of all the operation itself is not infallible. It is not going to produce weight loss without having to worry about what happens to the patient in the long run. You have to talk to them about it before operation. Afterwards you may risk having the patient come back into the hospital either to be educationally reoriented or to be fed because of persistent vomiting due to overeating. Another consideration is the type of diet on which you discharge the patient. Dr. Mason prefers a more liquid diet in general but I think that is a dealer's choice. I tend to be more in favor of sending the patient home on a solid diet. I know what kinds of food most folks can't tolerate but I don't know what an individual patient can't tolerate. Therefore, I let the patient make the choice. Most of the time they do pretty well and they eat what doesn't bother them.
DR. KRIDELBAUGH: I would like to ask if the Roux-en-Y demands any specific length of the Roux-en-Y limb and are they all retrocolic as the diagram indicates or are some of them antecolic?

DR. GRIFFEN: I always do it in a retrocolic fashion but I hate this saying that there is anything routine in surgery. I try to make it as short as possible. I've always made it where it lies nicely with a gentle curve. Many of them I bring around and anastomose to the opposite side.

DR. MASON: You have talked about the length of the afferent loop. How about the other loop?

ANSWER: I am trying to prevent reflux. The length of the anastomosis to the jejunum is approximately 35 cm.

DR. MASON: Is there less vomiting with the Roux-en-Y?

ANSWER: They all vomit at the beginning but as soon as they stop the over-indulgence of food and drink then they stop vomiting. I do not have them persistently vomiting longer than two or three weeks. I was impressed with the loops that they were in fact not only vomiting post-prandially but were also vomiting in between meals and what they vomited was pure bile. I said, "my God, they've got operative reflux." That is why I went to the Roux-en-Y and that with a bit of trepidation because of the problem, or possible problem, of stomal ulcers, but so far I have not seen any.

DR. BUCKWALTER: We have always done a retrocolic anastomosis, 12 to 14 inches.
DR. MASON: Dr. Gomez told me that he is doing some Braun intestinal or jejunojejunostomies instead of the Roux-en-Y and he can visualize the entire excluded stomach with the fiberoptic scope as long as the stoma is 12 mm in diameter or more.

QUESTION: But not with the Roux-en-Y?

DR. GRIFFEN: You can't. With the fiberoptic you can get up through the end-to-side anastomosis and all the way up to the stomach but not with a Roux-en-Y. The incidence of cancer of the stomach de novo is decreasing, the incidence of cancer of the stomach occurring in gastric remnants following surgical procedures for bleeding ulcers is increasing, and it is particularly prevalent in the Billroth II type of anastomosis. I personally believe it is totally related to the bile reflux into the gastric mucosa, which we know injures the gastric mucosa, and probably is the etiologic factor in gastric ulcers. That's why I'm not too terribly concerned about gastric cancer in the excluded pouch because I don't believe it's got much bile reflux. In patients in which we've looked into their excluded gastric remnants, the pouch looks like perfectly normal gastric mucosa.

DR. MASON: That might be an argument for not doing a pyloroplasty. We have two experiences of finding a very fatty liver at the time we did our gastric bypass. When we operated on the patients less than a year later with a good weight loss we found the liver to be completely normal. I only like to say this in order to dispel the idea that the liver damage is due to malnutrition. I don't think that's it at all. It's a unique feature of the ileal bypass, not due to the weight loss as such.

QUESTION: Does gastric bypass create a blind loop syndrome?

DR. MASON: We cultured the excluded segment both in dogs and in some humans and by and large the bacterial content is very low and comparable to what you would find in a normal stomach. In the dogs, interestingly, if there was some overgrowth of bacteria you could immediately get rid of it by giving a dose of histamine. In other words, the acidity of the excluded loop without any neutralization by food, is sufficient to keep
the bacterial count down. I do recall one patient that had quite a bit of trouble with diarrhea who was achlorhydric before he had his gastric bypass. I think this is a potential problem. I think you can get overgrowth of bacteria in the excluded segment if you happen to have a patient that doesn't secrete acid.

QUESTION: What discharge eating instructions do you give them?

DR. MASON: I think the most important thing to tell them is that everything they swallow has to be liquid. I don't send them out on a liquid diet necessarily, I mean, they can eat anything they want to if it is liquid when it goes down.

QUESTION: Are you starting all your patients on vitamins now?

DR. MASON: Yes. I think that is important to put them on multiple vitamins during the first month or so.

QUESTION: What kind of vitamins do you use?

DR. MASON: Multiple vitamins. Sometimes there is trouble with the tablets so you have to put them on liquids. You can calibrate your stoma that way.

QUESTION: In the patient with a previous history of gastric ulcer, would you be reluctant to do a gastric bypass?

DR. MASON: Dick, any patients have duodenal ulcers?

DR. HORNBERGER: Four or five, but they haven't been active for a time and I don't worry about it.

DR. MASON: Bill?

DR. JEWELL: I think about the same. We've only had a couple.

DR. MASON: Ward?
DR. GRIFFEN: I haven't encountered any, but I would go right ahead if they had them. Most overly obese patients don't have duodenal ulcers. They're sort of a happy bunch.

DR. MASON: John, any duodenal ulcers?

DR. ALDEN: Yes, but they were people who had the ulcers in their distant history. If an ulcer has been fairly recent, I would avoid doing it. It is only when I have heard they had their ulcer a long time ago and the preoperative upper GI is normal that I go ahead and do it at this stage.

DR. MASON: Joe?

DR. BUCKWALTER: No, I haven't seen any of these patients.

QUESTION: Has anyone who uses the TA-90 to create the pouch had any of the TA-90 staples give way postoperatively?

ANSWER: Not so far as I know.

ANSWER: We've had it fall apart just while we were doing it, that is, it was the suture technique, but I think that the reason was, as Dr. Printen pointed out yesterday, we had parts from two different instruments. You've got to be sure they are all matched. I think that is why ours fell apart, because our nurses mixed up our instruments.

DR. MASON: We've had two patients .... one of them drank a pitcher of water the first postop day and ripped it out, but it leaked into the distal segment, so it wasn't too serious although it did mess up the operation. It gave a very interesting x-ray. I think the other one was about the same sort of thing, except it was a smaller leak. I want to say one thing that I didn't say yesterday, and this reminds me of it. A lot of the problems with leaks in these patients arise because they develop acute gastric dilatations. This is often caused by the lack of compulsiveness about the nasogastric tube during that early period when the stomach is atonic and also because the resident or somebody will put
a sign on the forehead of the patient that says "DO NOT MOVE NG TUBE." Boy, once that sign is there, nobody will move it except me, and I come around and I'm always fooling with those tubes, trying to get them to work better. I've looked over those leak patients and not infrequently you look back to the nursing notes and see: 10 cc per 8 hours, 50 cc, and then all of a sudden the patient says, "Oh, I've got this terrible pain. It feels like my bladder burst." That's another sign. When they leak it runs into the pelvis. When they start complaining about their bladder you better look in the stomach.
INTESTINAL BYPASS COMBINED WITH OR CONVERTED TO GASTRIC BYPASS

John F. Alden, M.D., Paul H. Beckman, M.D., and Edward J. Drew, M.D.

DR. ALDEN: Without stapling instruments I wouldn't think of converting an intestinal to a gastric bypass. It would take too long and it would be too difficult. I would also like to make certain that you understand that I am not speaking of desperately ill patients with jaundice and hypoproteinemia. I only do this with patients who are still active and functioning and fairly well off physically, who either have too many kidney stones, polyarthritis, or too much bloating and gaseousness and distress and too much diarrhea. When I do a simultaneous operation like this I am dealing with a relatively healthy patient who just has problems with the intestinal bypass that are intolerable. In the 35 patients that I have done, the success rate has been high. I can't break down the figures because I haven't assembled them, but I can tell you I am very satisfied. The patients as a rule don't have a marked weight loss after the substitution of the gastric bypass for the intestinal bypass. They probably will drop, perhaps, an average of 15-20 kg or more after this operation. A few patients in which I think I made the gastric pouch too large have gained weight. I might add that I have never made a gastric pouch too small, and I suspect you can't make one too small, but I certainly have made a lot of them too large.

DR. MASON: So then your experience is 35 patients with conversion?

ANSWER: Yes.

DR. MASON: Have you done any adding on, have you had any patients with both gastric and intestinal bypass?

ANSWER: We have experience with two patients. The first one was a nurse who works at our hospital. She had a 14/4 jejuno-ileostomy with very poor weight loss and eventually regained to a weight higher than her weight at the time she had her 14/4 jejunoileostomy. She was quite upset about it. We talked about it and decided that she would take a chance and I would be willing to do this operation in stages, so I did a good gastric bypass on her. She has been extremely healthy. She has
had no problems of any kind. She keeps her jejunoileostomy. I see her regularly at the hospital. She recently won a beauty contest, so she is just fine. Her liver was absolutely normal, I want to add, when I did the gastric bypass in addition to this ileal bypass. The second patient, done by one of my partners, was a different story. It was a patient who had a 14/4 jejunoileostomy and had done rather poorly with a lot of diarrhea. Her liver didn't look very well when we did it, but having been pleased with the nurse's results we took a chance. She understood fully what we were going to do. About three or four months later she just wasn't doing well at all and we took down her 14/4 jejunoileostomy. She now lives with her gastric bypass. Her weight loss hasn't been particularly good, but she is regaining her health.

DR. MASON: Thanks, John. Paul, do you want to tell us about your experience. Dr. Beckman is from Davenport and he has had some experience with one of my patients, I believe.

DR. BECKMAN: Right. I wish she had remained one of your patients. When Dr. Mason asked me to speak I started wondering to myself just what my qualifications were, and I decided they were somewhat as follows. I had a series of one, which makes me an expert. The procedure has been banned in the area in which I live since 1974 and perhaps my third qualification is that on diet alone I have lost far more weight than my patient did in two operations. At any rate, the patient at hand is a 42-year-old white female, 4' 11" tall, with a preoperative weight of 240 lbs. At that weight she underwent a gastric bypass in April, 1972, along with repair of a huge ventral hernia which really was disabling to her. Her pregastric bypass weight was 243 lbs. Initially she lost about 20 lbs over the next few months, but in September, 1973, the patient's weight again was 240 lbs. After due consultation in Iowa City by the patient she refused to accept the offer of a revision of the gastric bypass and consulted me again. At this point she was markedly obese, again having a huge ventral hernia, and was semiambulatory because of multiple arthritic pains from her weight. After fully explaining the hazards to her, we elected to go ahead and do the intestinal bypass superimposed on the already existing gastric bypass. Incidentally, the preoperative lab findings were all normal, as were the x-rays, including
gallbladder series and an upper GI which showed a very adequate gastric pouch. Probably if I hadn't known what had been done to her I would say she had a normal gastric volume with a well functioning enterostomy. At any rate, we went ahead and did a 14/4 end-to-end jejun ileal bypass and she underwent a very uneventful postoperative course. She was fed by mouth three days after operation. Incidentally, a liver biopsy done at the time showed what our pathologist described as a moderate degree of fatty changes. Her large incisional hernia was actually the major technical difficulty. It was multiloculated and it took more time to do that than it really took to do the bypass. The third postoperative day she took food and fluids well. She was discharged on the seventh postoperative day. She was having two to three loose stools a day at time of discharge.

Over the next six months she had apparently very little trouble, and lost 80 lbs. She was readmitted in May, 1974, for correction of multiple electrolytic and metabolic disturbances. These were satisfactorily treated with parenteral fluids and albumin. The only other admissions she required were for elective repair of recurrent incisional herniae and two other bouts of electrolyte deficiency, one of which was definitely attributable to an apparent viral gastroenteritis which in spite of all our warnings she had refused to report until it was four or five days in progress.

The most striking and memorable admission was for severe hypocalcemic tetany. However, she recovered from this and apparently in questioning her this episode was also caused by lack of the patient's compliance in that she for some reason quit taking her calcium supplement several days or a week or more before she came in and again had an enteritis which was not particularly related to the bypass. It did not present any problems itself except for the incidental electrolytic disturbances.

At this time the patient appears clinically well. It has been 42 months since her last procedure. Her weight stabilized at about 135 lbs. She is normotensive with normal serum electrolytes. The only abnormalities existing at this time in our lab work are a total protein of 6 with an albumin of 3.0-3.5 and a persistently elevated alkaline phosphatase
between 150 and 180. This is in spite of liver scan which was normal and a GGTP that wasn't too far off. I don't know at this point whether or not to just let her stay with an alkaline phosphatase of 150 to 160. She is clinically well, as indicated, with no history of jaundice in the past of any kind. The conclusions I have based on this series of one are not very sweeping or very grandiose. At least in this case intestinal bypass produced satisfactory sustained weight loss but at a considerable cost to the patient for postoperative problems. It did not appear to me that any additional problems were encountered because of the superimposed gastric bypass.

DR. MASON: Dr. Drew is from Des Moines and he has had some experience with several of the patients who have been operated upon here with gastric bypass who failed to achieve adequate weight loss and then had added on intestinal bypass. Dr. Drew.

DR. DREW: I have not performed any gastric bypasses. However, in doing several hundred intestinal bypasses I have had six patients who had gastric bypass with unsatisfactory weight loss and later had a small intestinal bypass. Of the six, one had a lipectomy alone, five had jejunoileostomies without disturbing the gastric bypass. In three the weight loss has been satisfactory, one is too early to evaluate, and one was later reversed. I had one patient who is a 48-year-old white female who originally had a jejunoileostomy performed in 1967. This was done by Dr. Swenson in Omaha. She said it was a jejunoileostomy. At that time they were doing jejunocolostomies and it could have been that rather than jejunoileostomy. In any event, she went from around 283 to 160 lbs. In the interim someone did a hemorrhoidectomy and she said the anal sphincter was disturbed. She couldn't control the diarrhea, and I think Dr. Swenson reconstituted the jejunoileostomy in 1970. She continued to gain weight and went from 160 back to 283 lbs. In 1973 she came to Iowa City and had gastric bypass. Her weight fell from 283 down to around 230. About that time I saw her. She importuned me to have her jejunoileostomy reconstructed and I demurred thinking she should have another procedure such as removing the abdominal panniculus, removal of the humeral and femoral lipodystrophies, and elevating her breasts which extended downward to the hips. The weight did not change. Two
years later she had another jejunoileostomy and her weight has fallen from 230 to 160, which represents a weight loss of around 30% from the time the second jejunoileostomy was performed and 120 lbs from the time of the original operation in 1967. When she weighed 280 she had enormous arms, chest and neck. She later had reduction mammoplasty. She has also had an abdominal lipectomy and the excess fat removed from the arms and thighs. She isn't ready for Atlantic City but there is now quite a difference. Her arms, abdomen, and the thighs are much smaller. She is employed full time as the manager of a laundromat in Omaha and quite happy with her present state.

Another patient was around 40 and weighed 285 lbs. She had a gastroplasty performed in 1971 in Iowa City. I appreciate that Dr. Mason has changed his technique since that time. She had no significant weight loss. A year later she had a jejunoileostomy. Subsequently she developed a pyloric obstruction for which the old area of gastroplasty was resected, and a partial gastrectomy was performed. Subsequent to that she developed another pyloric obstruction and a Heineke-Mikulicz was performed. Because of the adhesions in the upper abdomen it was impossible to find the vagi without considerable trouble and nothing was done. Following the pyloroplasty she developed another pyloric obstruction and a gastro­jejunostomy was performed. In the meantime she became tired and shopped around, as patients are prone to do. She developed a hernia, went to a different surgeon who fixed the hernia, did another gastric resection and reconstituted the jejunoileostomy. Now she has made a complete circuit. In April of this year her weight is 285 lbs, the same as it was six years previously. I think the surgeon who completely reconstituted her jejunoileostomy was a little over-zealous and he could have compromised and perhaps not reconstituted the same full way.

Another patient had a fracture of her left femur. She had had a lot of bone operations. She was a patient in the Department of Orthopedics at the University of Iowa. She was 5' and weighed around 230 lbs. She was crippled and had considerable trouble ambulating; it was felt that her weight was a definite impediment. She was referred to the Department of General Surgery and had a gastric bypass in October, 1973. Her weight fell from 273 to 180, it later went back to 210, and at that time she
had an intestinal bypass and a lipectomy. Her weight fell from 210 to 163 and at the present time it is 170 lbs. This represents a 40 lb loss in the 18 months or 20% of her weight from the time of the jejunoile-ostomy.

The next patient weighed 365 lbs. She had a stomach bypass in the spring of 1972. She had a rather turbulent postoperative course, according to her description, and lost about 50 lbs in the hospital. Eighteen months later she weighed 272 and had a large abdominal panniculus. An intestinal bypass and lipectomy were performed in November, 1973. Her weight has fallen .... in the first six months it went to 195 lbs, but at the present time it is 181 lbs. This represents a 50% weight loss from her initial weight, and approximately 33% weight loss from the time of the small intestinal bypass. At the present time she is quite happy. She had some difficulties with hypoglycemia. She was referred to Dr. Buchwald at the University of Minnesota who prescribed some protein supplements which apparently corrected her low blood sugar, and she is quite happy today. Incidentally there is a familial tendency in this patient. Her oldest daughter weighed 300 lbs and also had an intestinal bypass. She didn't have anything done to the skin to remove excess fat, but she now weighs around 160 and is quite happy, as well as her mother. A second child by a different husband is about 8 years old now and is on a collision course with obesity. She weighs almost 160 lbs. If things go on as they did with her mother and sister, she will ultimately have to have something performed.

Another patient had a stomach bypass about six years ago. She weighed 350 lbs then and lost very little. She came in requesting a small intestinal bypass. Her daughter had had one with gratifying consequences. However, she has a disabling arthritis in her right hip. Her hip is flexed and her knee is flexed, and I was afraid she couldn't stand the diarrhea. She is confined to a wheelchair most of the time. Therefore, I compromised and removed the large abdominal panniculus which extended almost halfway to her knees. She lost about 30 lbs. That could be attributed to removing the panniculus and if it weren't for her age and her incapacity and her arthropathies she would probably be a good candidate for further surgery.
There are about 300 reports in the Index Medicus about the surgical therapy for overweight and hardly any advocate removal of the panniculus at the same time. I have done it on practically all my patients, and I have devised this technique which might be of some interest. I don't think removing the panniculus contributes to the morbidity or mortality, and it gives the patient the feeling that something has been accomplished right away. I take a bar from an Albee orthopedic table, put it in the sterilizer, use Backhaus towel clamps, clamp the panniculus and suspend it from this bar. The drapes are arranged so you are working in a sterile field. I remove anywhere from 5 to 45 lbs.

All of these people are told to take baths a couple of times a day with Hexachlorophene soap preoperatively. I haven't had much problem with wound infections. They also have bowel preparation with Terramycin and neomycin. After the skin is brought together I put two large rectal tubes in and connect them to Gomco pumps. That keeps the wound fairly dry and the incidence of seromas or hematomas has been minimal.

My last patient is 40 years old and had a gastric bypass performed nine years ago. He was confined to The University of Iowa Hospitals for several weeks preoperatively and his weight was brought down from 380 to around 300 lbs by dietary measures. Before having the operation he was permitted to go back on a regular diet. At the time of intestinal bypass his weight was 320 lbs. Nine years later his weight is 295 lbs. One leg has been amputated from a farm accident. He is too early to evaluate, but his wife has had the procedure and is quite happy, and he thinks it will help him with his weight.

DR. MASON: All right, it looks as though we have nine patients, then, that teach us something about this combination. Who wants to summarize this?

DR. ALDEN: Well, I would just like to say that if you have a notion that you want to play God and try to do this, it is okay, but you better have a pretty good agreement with your patient. If you decide to add a gastric bypass to an intestinal bypass, don't do it if the liver isn't perfect. If the liver is excellent, I think it means that the intestinal
bypass is doing essentially nothing at this stage and the patient has recovered metabolically, nutritionally and anatomically from that previous surgery. Probably, then, you might get by with the gastric bypass imposed upon the intestinal bypass, but for heaven's sakes if the liver looks bad, back off.

DR. DREW: That sounds like an awful lot of surgery to take a jejuno-ileostomy down and do a gastric resection for a bypass simultaneously. How long does it take when you do that?

DR. ALDEN: It sounds big and it looks big when you write it up in the operative report, but it is not much of an operation with the stapler. It certainly is less than two hours.
We definitely would like to have all our patients be in a status where sooner or later they require a panniculectomy. We do abdominal lipectomies or panniculectomies. There are also thigh, arm and breast reductions. I think one thing that can truly be said about them is that they are not cosmetic operations. When you take 25-30 lbs. off someone in one fell swoop ala Attila the Hun, you really are not making the individual prettier. Nevertheless, I think it is very important to recognize for the patient's benefit and also sometimes for the benefit of visiting with other physicians that these are really functional kinds of operations. Many people can't get around very well with 25-30 lbs. of excess skin hanging down. Often in society, just as the morbidly obese patient is discriminated against because he is so big he can't function, so is the person who carries around a 30 lb debilitating apron. The patient looks odd, there is no question about it. There are no clothes you can put on the patients that keeps them from looking funny. Consequently, these people feel the same kind of discriminatory effects on jobs and the like that they would feel had they not lost weight.

Our experience is largely with abdominal panniculectomy at the University of Iowa. We have done 105 total panniculectomies of various types, shapes and sizes. Eighty-five of them have been of the abdominal variety. We are pleased with results of upper extremity lipectomy although surprisingly, unless the patient is grossly obese we don't have much trouble with hanging arms. This is particularly a problem in women, but if they will exercise, the arms do tend to firm up so that they can fit into dresses. That is usually the determining factor in operating on a patient's arms.

In regard to the abdominal panniculectomy, we have been doing a relatively standard operation. The skin fold or crease underneath the abdominal panniculus, which is always present, is usually the area where the individual will get intertriginous skin infections. Therefore, it is important to make sure that this area is free of infection before we accept the patient for panniculectomy. The initial incision is made
there. The amount of skin and subcutaneous tissue that is removed generally falls into a wide ellipse of tissue which ranges anywhere from 5 to 45 lbs, depending on how much weight the patient has lost.

There are several ways of doing abdominal panniculectomy. The worst complicating technical feature is the lack of blood supply, usually to the upper flap, primarily because of other incisions. Many of these patients have had cholecystectomies, or C-sections in addition to their bypasses. They have had this operated on and that operated on and they often have a lot of incisional scarring. Consequently, the blood supply to a tremendously large flap on such patients has been lost. Therefore, as a general rule, we do not mobilize the skin and subcutaneous tissue from this intertriginous incision all the way up to the costal margins. We found that when we did this we lost the tissue right around the umbilicus, which takes 33 to 35 days to heal. On those patients who have no incision anywhere else in the abdomen I think it is perfectly fine to mobilize up to the costal margins, circumscribe the umbilicus and replace it so that it sits in its normal position. For the patients with other old abdominal incisions, I feel very strongly that we must make the first incision in the inguinal crease. Then we must plan with a marking pencil in order to determine how much skin and subcutaneous tissue can be removed without elevating such a large flap that the blood supply may be jeopardized. As I say, most of these are functional panniculectomies. You are not doing them so that the lady can wear a bikini, just so she can wear overalls of the proper size.

We have had an appreciable wound infection rate with our abdominal panniculectomies although really not as high as we thought initially. At one point we adopted a policy of keeping everyone who had had a wound infection at their original gastric bypass on antibiotics prior to their abdominal panniculectomy. We no longer do this on a routine basis since we have found that only 8% of the patients who have wound infections at their primary surgery have wound infections with their panniculectomy.

I want to make note of the kind of operation we do on the thighs. It is really a bit different than what we had been doing. John Alden removes a diamond shaped piece of skin and subcutaneous tissue that is actually shaped with a little tail on the medial end that extends in between the
legs. Initially we took the redundant medial thigh tissue by making a long incision all the way down to the inner aspect of the calf just below the knee, and freeing up a large skin flap that went posteriorly. Finally we just pulled everything around and cut off the excess. This has not held together satisfactorily, and certainly is not satisfactory from a cosmetic standpoint since it is a very difficult incision to close without leaving a very wide scar. The incision is usually under a fair amount of tension. It is difficult to keep the patient immobilized, which you have to do if you are making longitudinal incisions for the thigh panniculectomies. If you don't, they pull apart. In addition many patients get swollen feet when you have completed the procedure. It is nothing that is insurmountable and support hose have been able to handle the situation. Nevertheless, the patients must stop in the middle of the day, the same as somebody that has chronic circulatory problems of a venous nature in the lower extremities, and elevate their feet. Otherwise they get pedal edema. I am sure this is due to interruption of lymphatics by that large up and down incision and the mobilization of all the skin.

But we have used Dr. Alden's technique with relative ease and it works well. You perhaps don't get as much of the skin out of the area in between the legs. However, you are able to usually pull enough up so that it gives a satisfactory result and once again the patient is able to wear clothes. That is what the whole story is about with this particular panniculectomy.

Infections of a serious nature in the panniculectomy are devastating infections. The patients stay in the hospital for quite a long time. What we have really is an 8% incidence of reinfection of the wound, in other words, a wound infection at gastric bypass and a wound infection after panniculectomy. You notice I didn't mention drains of any type in abdominal panniculectomy. We sort of wax and wane. Most of the time we feel that if you observe meticulous hemostasis even if we elevate a large flap, we don't need a drain. I have put in sump drains, but don't anymore since it hasn't done a thing to prevent the development of seromas.
Of course, the buttocks are another area in which some of our patients are interested in having work done, and this, by the way, is about the only area of the obese patient on which our plastic surgeon will work. He claims he was trained to be an aesthetic plastic surgeon and that meant you removed grams of tissue, not kilograms. Consequently, he says he doesn't know really what to do with a gargantuan abdominal panniculus. Buttock lifts, certainly in the younger patient, are something that can be done with relative ease. What you end up doing, of course, is tucking the upper flap under and reproducing the contour of hopefully someone who looks better than with all the previous sagging tissue. We have done very few of these, as I say, this is in the realm basically of our plastic surgeon. Those that have been done have been relatively successful and probably more aesthetic than any of the rest of the things we do.

The incision we use for our patients who have the fat and subcutaneous tissue hanging under the arms starts below the elbow and is carried well back onto the posterior chest wall. We feel that using an incision this long allows us to do several things. First of all, we are able to effectively remove this great mass of hanging tissue. In addition, the majority of these ladies also have a fair amount of hanging tissue on the lateral chest wall. With an incision that is carried down as far as ours you are able to gather up some of that tissue and remove it at the same time. This becomes important especially if the patient has breasts that are of the size that sag far enough for her to want a reduction mammoplasty. With such an incision you can remove back tissue, which you can't get with a routine reduction mammoplasty. That's the story with the various types of panniculectomies we have performed, as I say, primarily in four areas, the abdomen, the thigh, the arm and the buttocks.
STOMAL ULCER
Edward E. Mason, M.D.

Stomal ulcer was the first problem we were concerned about. With a small fundic segment there isn't enough parietal cell mass to cause a stomal ulcer. Thus the secret of success in weight loss is also the secret of preventing stomal ulcer.

If a stomal ulcer does occur, it may respond to medical management. The excessive stomach can be removed and the gastroenterostomy refashioned. However, if the stomach has been divided at too low a level, there may not be enough acid-secreting tissue in the excluded segment to inhibit the antrum. I haven't documented this, but I think this is one of the reasons we should try to get serum gastrin levels whenever this sort of problem does arise.

We have had an incidence of 2% of stomal ulcers. I think it will be lower now that we are making smaller pouches. You have heard some accounts today of several hundred patients with no stomal ulcers, and it appears that no one has had an incidence of stomal ulceration that is above 2%. Incidentally, I have seen reports in the literature of duodenal ulcer occurring in over 4% of the patients with jejunoileal bypass, so it is a problem with both operations to some degree. We performed gastric bypass on eight thin patients with duodenal ulcers. Within about 18 months we observed a 75% incidence of stomal ulceration. About the same time I ran across the publication by Andrew Kay of Glasgow who, on the same thesis of excluding acid-secreting tissue with the antrum and thereby inhibiting the antrum, had used a 50% exclusion operation for the treatment of duodenal ulcer but had to give it up because of the high incidence of ulcer recurrence. Wadell and Barrett reported a series of patients with antral exclusion plus truncal vagotomy who did not develop stomal ulcer but these patients did not have all of the parietal cell mass above the gastroenterostomy that would occur if you transected the stomach at a low level. Consequently, I don't think this establishes that we could get by with an inadequate gastric bypass plus vagotomy. We have talked about this. Dr. Printen recently had a very heavy patient who had a lot of other problems including severe cardiac
disease. He was anxious to do a simple procedure, and he did do a
 gastric bypass with a small pouch plus a vagotomy. He put a gastrostomy
tube in the excluded segment and found there was a marked reduction in
acid secretion. She had relief of her ulcer symptoms but died a few
months later from her cardiac disease.

Ten years ago when Chikashi Ito was with us for three years, we did a
lot of work in the laboratory in preparation for the use of gastric
bypass in the clinic. One set of experiments was presented this fall at
the International Surgical Society meeting in Kyoto. It is patterned
after the work that was done at the University of Minnesota which actually
stimulated me in seeking residency training with Dr. Wangensteen. They
gave dogs histamine-in-beeswax and created ulcers. This model was
thought by Drs. Varco, Lanning, Hay, Wangensteen, et al., to be a means
of testing an operation for effectiveness in the treatment of ulcer. It
seemed at this time to be a good model but that was because they weren't
thinking in terms of vagotomy. They were thinking in terms of removing
the target tissue, and if you remove enough parietal cell target tissue
you can prevent stomal ulcers. Then came along the operation of antrec­
tomy vagotomy and it does not protect against ulceration with histamine-
in-beeswax.

This perhaps gives some interesting information and something to think
about in terms of gastric bypass which is an exclusion operation. One
difference between these experiments and the experiments that were done
in the 40's is that there is no resection. The stomach is transected.
Part of it empties through the duodenum and part of it empties through
the gastroenterostomy. Note that with antral exclusions stomal ulcer
occurs with histamine-in-beeswax. If you exclude more than about 36% of
the stomach, you no longer see stomal ulcers because there isn't enough
acid-secreting tissue emptying through the stoma to create an ulcer.
When a gastroenterostomy is added to the pyloric outlet as in gastric
bypass the surface area through which the acid leaves the stomach is
increased. It would seem that if you did a 50% exclusion, as Andrew Kay
did, this would be an ideal operation. This is because half of the acid
would go through the duodenum and half of it would go through the stoma
and therefore there shouldn't be enough acid in either place to cause
ulceration. Of course, that didn't turn out to be the result with Kay's ulcer patients and it illustrates again the fact that this model doesn't necessarily predict what will happen with acid-peptic disease.

With even more extensive exclusion of the stomach, you increase the risk of ulceration in the duodenum. This is really not a strong ulcerating tendency. The duodenum has a greater resistance to ulceration than the stoma, simply because the duodenum contains bile and pancreatic juice and a lot of bicarbonate which neutralizes the acid. By looking at all these progressively more extensive exclusions together you can see that in antral exclusion, ulcers occur in the stoma. With more extensive exclusion they no longer occur in the stoma but a few begin to occur in the duodenum. Actually, we had two other experiments where exclusion of over 60% of the stomach never produced any ulcers. This would be consistent, of course, with the thesis that the duodenum does have better protection because of the buffering of bicarbonate.

The other model was the historically older model of common duct ligation. In regard to a comparison of the histamine-in-beeswax versus common duct ligation pattern of ulceration, with common duct ligation you never see stomal ulcers, but you do see duodenal ulcers. If one is to interpret this in terms of patient care I think it means that you should be very wary of letting any patient become jaundiced after gastric bypass. If they have exclusion of bile from the duodenum, this will markedly cut down on the availability of bicarbonate to neutralize gastric acid. Bile has bicarbonate but, in addition to that, the presence of bile greatly augments the secretion of the bicarbonate in the pancreatic juice which is stimulated by secretin. Putting this all together, a great deal of bicarbonate is dependent upon bile coming into the duodenum. That is interesting in view of our concern about the reflux of bile into the stomach and reflux of bile into the esophagus. In the duodenum bile is a very important and protective material.

Early work indicated that gastric bypass was protective. We made a Pavlov pouch and studied the effects of a standard meal on acid secretion in dogs. We then added gastric bypass, and showed that acid secretion was greatly reduced because of acid in the excluded segment washing over
the antrum. The dogs secrete with the meal, after the pouch was made. After gastric bypass the secretion is reduced because of inhibition of the antrum by secreted acid in the excluded segment. With removal of the acid-secreting part of the distal segment and conversion to an antral exclusion, a late intestinal phase of secretion is observed. Then after removal of the antrum and conversion to a gastric resection very little secretion occurs. Thus, it can be seen that gastric bypass and gastric resection have about the same effect on gastric secretion following a standardized meal.

The thesis that the inhibition was due to acid washing over the antrum and inhibiting the secretion of gastrin was tested eight years later when serum gastrin analyses were available. By giving patients a standard 250 cc of milk to drink and measuring the response of their serum gastrin, performing gastric bypass and then, after recovery, repeating the experiment it was found that the serum gastrin level was significantly depressed.
Fellow surgeons, for the last two days we have been listening to when and how to do gastric bypass, and we have been learning how to prevent or correct complications. We have learned how to minimize some of the side-effects of duodenal shunt. Let us now look at some of the factors to which many of us have alluded. Let's look at what really makes it work, patient motivation. By this time we are convinced of one thing, and that is that it doesn't work for everybody. The patients in whom it does work really want it to work. We as doctors can exploit that. Several people have said that when the patient comes in we tell them (a) that they might die, (b) it might not work. But when they can't continue that same way of life, then we say "If you want it, we'll do it, but we make no promises." Now, I'm going to ask you, if you came into my office with a big hernia and said to me "I've got this big hernia," and I say "Boy, you're right, it's a big one," and if I go on to say "Sometimes we fix them, and sometimes they work and sometimes they don't work," what do you think? Do you think you might see another doctor?

Don't be wishy-washy. If you're in this business of obesity you have to care about the fat person. If you don't think it is serious, don't do gastric bypasses. We have heard how one man here has devoted ten years of life to this, he obviously has been serious. I think patient motivation is serious, and I have learned some things I'm going to pass on to you. The starting point of all achievement is desire. The patient comes in and says, "Doctor, will gastric bypass work for me?" Your ears and your heart have to tell you that what that patient is saying is this "Doctor, I've tried the whole reducing bit. I've been to TOPS and Flops and I've been to Weight Watchers. I have had an ileal bypass. Nothing works for me. Woe is me. Do you have something to deliver me from the bondage of this fat?" That is what the patient is saying. You already know and you have heard for the last two days, that that isn't the way it is. You know straight away that there are certain limitations that you can do with gastric bypass or anything else, and you must immediately ask the second question, "Will you work for your gastric bypass?" That is the question. Will you work?
You know what the successful doctors do? The first thing you learned in medical school was how to take a history. There's not a doctor here that would swap the physical examination, statistics, laboratory work or x-rays, for the ability to sit down and take a history. You're supposed to know how to ask questions. Learning how to ask questions is what gives you insight into the physiology, pathology and mental workings of a patient. You have to be good interrogators.

You begin at the initial interview by asking: "Why are you interested in gastric bypass?" This is a key question. What you want to know is, is this patient here to have a more successful life, to get a better job, to have a better figure? Is this patient here to be healthy and strong and muscular, or is this patient here because he is just simply tired of counting calories? Is the patient here just simply because he doesn't want to worry about it anymore? He doesn't want to think about it, he just wants it all to disappear. If you took all the knowledge of the ages and capsulized it, it would come out to one sentence, and that one sentence is "There is no such thing as free lunch." You know to do a gastric bypass without caring makes about as much sense as buying a good piano and expecting it to make you a musician, it just isn't going to work.

The second question is "What have you heard about it?". Find out if your patient talked to another patient, or have they just read somewhere that they are doing some kind of fat surgery over there in Iowa and I'm going to go up and have some of it.

Did you ever ask the patient "How much weight do you want to lose? What do you want to do with your new body?" I'll tell you what the plastic surgeons learned a long time ago. You can't fix a nose and expect to end up with a Farrah Fawcett. Gentlemen, it just doesn't work. You better find out, what is the patient's goal, and see what you can do for that patient to reach a goal. If the patient doesn't have a goal, help him find one. Discover what the patient's goal is, what they think of it. Maybe all they want to do is just be able to get up and walk around with their grandchildren, and if so 50, 60 or 100 lbs. is enough. Don't decide for that patient. Let the patient decide.
What have you done so far to reach them? One thing you have to know is: how committed is that patient? Has that patient been to TOPS? Has that patient been to Weight Watchers? Has that patient had her jaws wired or staples in the ear? Has it worked? What you want to know is if the patient loses weight, have they diligently stayed with the program and how committed are they? You have to know that before you're going to put that big zapperoo on the belly, you see, and then you have to say, "What do you expect of gastric bypass?"

I'll never forget Betty Morton. She is the reason that I became interested in motivation. After she lost 50 lbs in four months, and I was sitting there thinking, boy, this is really something, I really did something, she got me. She said, "You know, I'm really disappointed, Doctor." I said, "Betty, how can you be disappointed? You've lost 50 lbs in four months. Why, all the people say how well you've done." She said, "I know, but I thought after this operation I would never have to worry about it again." I knew right then that I had to worry and I said "What are you willing to do to make it work? What are you willing to do?"

Ladies and Gentlemen, every one of you sitting here are here right now because of the choices you made in your life. Sometime take an hour off and sit down and look at where you are and start tracing your life back. You will see that you are where you are because of the choices you made from the time you got up in the morning until you went to bed at night. The greatest power in your entire life is your power to choose. What are you going to do for that patient? Ask yourself, "How could I help this patient make the proper choices that will lead to reaching the personal goal that that patient has set up? And where do we start that?" What do you insist on? You insist on counselling with gastric bypass patients, attendance at the gastric bypass meetings, and thorough commitment to the success formula.

Let us talk about the success formula. Someone already mentioned this, and it is absolutely true. If the patient knows what to expect and if the patient sets the goals; if the patient comes in and literally pleads with you and begs you to do that operative procedure, he will no
doubt be in your success column. He will be a successful patient. In Arlington there is a monthly meeting of gastric bypass patients, I've only done 45 so far. Half of them come to the meetings and they are successful. The meetings are arranged and chaired by the patients, but I do attend regularly. We talk about the topic of this discussion, that is, what the prescription for success in life after gastric bypass is. We also talk about principles and rules. One of the most important things is built-in satiety, "that's all I care for." Gentlemen, when you sit down at that table and they pass those vegetables and meat around or they ask if you want dessert and you say "No, thank you. That's all I care for." you remember every time that the obese person who is coming into your office would consider that a blessing. They never have that opportunity. It is a blessing to say, "that's all I care for." If there is any primary effect that you will accomplish for patients with gastric bypass successfully, it is that you reduce the pouch. What you eliminate for those patients is the self-hate of overeating, and they hate themselves. The talk about jolly fat people, it just isn't so! They are not looking around and laughing their head off because they are jolly and happy. They hate themselves, and part of the reason is because of what happens at meal times. They say, "If I eat that, I know I'll hate myself. I shouldn't eat that. Oh, I'll eat it, and eat it anyway, and then I'll go home and hate myself." Isn't that right? Has anyone seen anybody do that? Another thing you eliminate is the humiliation those people feel about public opinion. What someone says to you isn't what hurts. And what happens to you isn't going to hurt you. It is your opinion of what someone says, and these people have a very low opinion. They are sitting there thinking "I know what that guy's thinking. He thinks I'm eating like a pig, yeah, I know, I know. If I eat another piece of cake you know what they're going to say, don't you? 'No wonder he's big and fat, look at the way he stuffs himself.' They live with that, day in and day out. When you make a small pouch you eliminate that. They can now say "He can see I don't eat too much," and just getting rid of that negativism is a big help for these patients.

The third thing is, if you're hungry you suffer. If you don't think these people suffer when they're hungry, you just get in a cave with a
hungry lion and find out. It is important to emphasize regular eating habits. You have to find out if this person wants to be healthy. You have to talk about making them build a healthy body of lean muscle, strong bones and attractive skin. We are not talking about loss of fat. That's already been talked about. You see, inside every fat person, and you all know this, there is a lean person who is longing to be free. You have to concentrate on that lean person but not only that, you have to help that fat person concentrate on that lean person. You have to make that lean person the self-image and not that big old fat stuff on the outside. Regular meals are important because you say that with a regular meal you are feeding that thin person. It's the kind of diet that maintains that lean body mass, that's where it all evolves. If they work on that, the fat will take care of itself, because when you eat regularly, if you have a small pouch you can't overeat and if you have a small stoma you can't eat too often.

The next problem is to lower those carbohydrates. You just can't tell someone not to eat sweets. First of all, those of you who are still doing five-hour glucose tolerance tests know that most of those patients have a reactive hypoglycemia. They have a sharply accelerated-decelerated curve, and that is attributed to hunger. Now you can't just come up and say don't eat sweets, you have to tell them what to do. Give them a low carbohydrate cookbook so they can go home and learn how to make some goodies they can eat. Tell them to put some diet grapes in their refrigerator and say, now this is mother's, just keep your hands off, or this is daddy's or this is Larry's or this is Willie's. Teach them when they go to a cocktail party how to drink quinine water and a little lime juice. You are trying to normalize a life for these people. When other people snack, they have to have something to snack also. The same holds true for drinking. You don't want them to sit on their hands, but you don't want them to eat sweets. You have to tell them what to do, not what not to do. And you must impress them with this, that Mother Nature made them. We can't help that, but we burn sugar first and when they fully understand the fact that they won't burn fat until all the sugar is gone, it is a lot easier for them not to eat sugar, because down deep what they want is to burn off that fat. Take the time to explain that to them.
There's no use going through this if you're not going to emphasize exercise. It has actually been proven that exercise results in muscle growth, hypertrophy. The average football player lifts weights so that he can be heavier. They don't go home to lose weight, they go home to gain weight, and how do they do it? By building muscles. What does that mean? Don Reeves, middle linebacker of the Chicago Bears, sent me his program. I happen to know that young man, from West Texas. He showed me how he has built up his muscle over the past five years with a high calorie diet and exercise. We all know you don't make new muscle cells which means muscle cells grow. There are two kinds of exercises and they are both important. There is regular sustained exercise. That can be walking, it can be jogging when you feel better, it can be skipping a rope, it can be swimming or playing handball or playing tennis, but it has to be regular and sustained. It is true that it helps the heart and it is true that it helps the lungs, but it does something else. It gives the patient some kind of objective improvement that they can measure. He goes home and says "Do you know what I did? I walked a mile today" or "I jogged a block" or "I jogged a block and two mailboxes." They can measure and record their level of what they are able to do. There is one thing they have to have and that is a partner. The first thing you do is tell them to go home and get an exercise partner. Do you know what happens about 6:30 when you would like to get up and go take a walk, and you don't have anyone to walk with? You say, "I think I'll wait until tomorrow morning to walk." They have to have a partner. My wife has a walking partner. She's not morbidly obese, she's just a little bit heavy. Finally she learned if she's going to walk every night she has to have a partner, because those nights she would like to sit home, take off her shoes and turn on the boob tube, the partner arrives and says "Are you ready to take your walk?" Then she has to get up and take her walk and she doesn't wait till the next day. She has to have a partner for sustained exercise.

How about stretching exercises? You know what I mean by that: body bends, toe touching, and sit-ups. Why are they important? They're important because they make you feel good. The other thing it does is that in the absence of protein depletion it helps eliminate a lot of that skin sag. Therefore, stretching exercises are very important.
Next you have to tell people how to look at weight. If I were to ask the average patient walking around in this room today how much do you weigh, there would be many people who said I weigh exactly 163, or I weigh exactly 168, or you'd say oh, I weigh somewhere between 150 and 155, somewhere between .... But if the fat person weighs every day and they concentrate on weight, do you know what happens? When they get up in the morning and get on the scales and they gained one pound, they say "It ain't working, it's not working." It's very destructive for them to weigh each day and see that weight fluctuation. They should have a regular schedule, they should keep their weekly measurements, their weekly weight, marked "Weekly Measurements" and "Weekly Weight" and "Weekly Accomplishments" of what they are able to do. They're impressed with a curve that gradually goes down. If the curve isn't going down they can see what they need to stress on their life program.

A strong self-image is important. I was 45 years of age before I learned the importance of that Bible verse that says "Love thy neighbor as thyself." What that is really saying is that you have to have a lot of love and respect for yourself if you are going to want to know how to give love and respect to your fellow man. You have got to help your patients have an improved self-image. Believe me, they have the most destructive, poor self-images of any group of patients that you will ever treat. You have to help your patients to set goals. Elliot Peters once said, "if you don't know where you're going, you will end up somewhere else." He also says that a successful life is nothing more or less than a successive achievement of worthwhile goals.

How do you help your patients set goals? There are three important questions. The patient must ask "Where am I, where do I want to be, and how do I know I am getting there?" Those are the three questions. You have to help your patients set goals. At the beginning you ask them what their goals are and how those goals are going to change. If you have goals you also know that you don't get there in one bound, but what you have are the immediate goals.

It's very effective to tell them to go down to where they buy their clothes and pick out a dress or a pair of pants that's one size too
small, just one size, put it on lay-away and say I want to be back in exactly 30 days. I'm going to pay half now and half then. When you date it you set the wheels in motion. A goal without a date doesn't mean much, that's a "some day I'm gonna ....," but when you say, "in 30 days I'm gonna," then you're going to do something, especially if you pay half down. It could be a belt, or it could be a dress pattern with the material cut one size too small. When that patient goes down there and gets that dress and that dress fits the sense of accomplishment is what puts them on the next goal. It's a successive achievement of worthwhile goals that will help that patient reach the end goal. Tell those patients to set their goals according to whatever they want to do. Maybe they want to ride a bike. Maybe they want to put on a bathing suit. We had a swimming party for these girls, and none of them had had a bathing suit on in less than 10 years. Maybe they want to have a bathing suit. Maybe they would just like to go out and play with the kids. Maybe they would like to model clothes, even if they are stout clothes; what you have to do is help them start now. The beginning is the first step of winning. You never win until you start. So tell them to begin those immediate goals. Don't be afraid to laugh a little bit, teach them how to laugh, most of them have forgotten how to laugh. Tell them, "Don't take life so seriously. No one ever gets out of it alive." Help them to set their goals.

As physicians you all know about the subconscious mind. Maybe right now and for just one minute we should talk about some things you have forgotten about: Worry and anxiety. Simply defined, they are nothing more than thinking about what you don't want to happen. A positive mental attitude and enthusiasm or successful thinking is thinking about what you want to happen. You've all heard that. What does a guy say when he knocks the ball in the water playing golf? "You guys just knew I was going to do that." You've heard it many times. That's the secret of life. You concentrate more on what you don't want to happen and your behavior is modified toward that end-result. The successful people never think about losing and they are winners. That's what they mean when they talk about a winning attitude, then you do winning things. You know your brain, your mind, is the biggest computer you can imagine. No one has ever calculated how big a building would have to be to hold a
computer that can do all the things that your mind does, and your mind is your servant. You just work the buttons and the mind does what you tell it. It has no conscience. It just simply reacts when you push the buttons. That's what it does. It is just like the captain of the ship who picks that thing up and says "All right, this is the Captain speaking. One degree right rudder. Full speed ahead." The men in the engine room don't say, "I wonder if that dummy was up all last night. Maybe we ought to set it 2 degrees left rudder and about half speed." They don't say that. When you are going to throw a baseball and you say "throw that baseball against the wall" your arm doesn't say, "I wonder if that dummy really wants to throw it about six feet on the other side," no.

You have been building up all those things in your mind and all you do is punch the buttons for what you want. Fat people are simply exhausted worrying about their fat. That's all they ever think about .... fat, fat, fat, fat, fat. They want to think about fat, fat, fat, gain weight, gain weight, and when you do it makes you hungry, so you go have lunch, and then gain weight, gain weight, gain weight, and it makes you hungry. Isn't that right? It's true. Some of you stout people know that when you think about that it makes you hungry. And you know what the fat person is always thinking about? He's thinking about those lucky people, thinking "Boy, I'd like to look that happy" or "Look at Farrah Fawcett" "Just look at those athletes and those actresses and those actors. They never have to worry about fat." Baloney! Those thin people are imaging themselves as champions. They are imaging that beautiful body. They are thinking about how to stay pretty and how to stay strong and how to stay alive and limber. What that means when they punch those buttons is "Stay muscular, stay thin." That means, get out there and jog, don't eat that dessert. That's what the subconscious mind can do. You have to explain that to your patients. You have to help those patients understand that their attitude is going to determine what they do. Thoughts develop actions and actions develop habits, and habits, gentlemen, develop character.

"Bakeries have proved that you get a perfect cake every time if you use the right ingredients and follow the proper recipe." Gentlemen, this is the right recipe and the right ingredients. I give all of my patients a booklet called: "The success prescription for your life after gastric
bypass." We talk about it. We work with them. It gives us a baseline.
You perhaps would like to have a copy of this to tailor for your patients
in your work. It's good and it's wholesome. If you indeed would like
to have a copy of that which you can tailor in any way you like to help
your patients, please write to me.
GASTRIC BYPASS REGISTRY PLANS
Thomas J. Blommers, Ph.D., and Edward E. Mason, M.D.

I would like to briefly tell you about our National Gastric Bypass Registry. The National Gastric Bypass Registry has as its purpose the establishment of a series of cooperative studies of morbidly obese patients undergoing gastric bypass in order to assure that maximum benefit will result with minimum risk. These studies range from the simple collection of information about weight loss and its relationship to other variables such as sex, initial weight, age, etc., through studies of the frequency of pulmonary embolism, hepatitis following distinct anesthetic agents, and other complications, to specific cooperative prospective randomized studies regarding treatment in order to prevent such complications as postoperative wound infection, the formation of gallstones during the period of rapid weight loss and other studies. We feel that many problems simply cannot be studied unless you have a very large series of patients. For example, there have only been 4 fatal pulmonary emboli in the entire U of I series of over 600 pts. We are gratified that this number is so small. However, it is obvious that with so few patients any type of statistical analysis is impossible. It is hoped that by pooling the experience of several series enough patients having had this disastrous complication can be found to make it possible to study pulmonary embolus effectively and to discover ways to avoid it entirely.

As yet we have not secured large scale funding, so we have been proceeding cautiously and slowly. We hope that funds will become available in the future to hire data abstracters and to purchase some other useful equipment for data management such as a computer terminal. Until such funding does become available we are limiting ourselves to the general study and collection of data, including the study of the more common demographic, laboratory and weight loss variables. The special studies such as the study of pulmonary embolus, have as yet not been undertaken.

The results of the data collection will be sent back to the participating surgeons or surgical practices. It will compare their series to The University of Iowa as well as The University of Iowa compared with all
the other surgical practices combined. Naturally, confidentiality will be carefully guarded. At no time will the results of an individual surgical series be revealed to anybody outside of that practice. The patient's identity is also being protected through the use of coding and scrambling devices that our University Computer Center offers for just such purposes. There is a preoperative-operative registry form, and also a postoperative information form. These forms employ a mark-through duplicating process so that the individual surgeon or practice may easily retain a copy for their own purposes. Each surgeon is given a three letter code prefix when they indicate they would like to join the Registry. They may then code their patients in the code box chronologically, for example, ABC-1, ABC-2, etc. In this manner, if there is a patient that does not want to be identified by name or if for some reason a surgeon prefers not to identify his patient, data can still be transmitted by code number only.

The Registry has been operating now for a little over a year. However, as yet it is still fairly embryonic. We have eight participating surgical practices so far who have contributed a total of 108 patients. There have been three early deaths, which represents 2.7% of the patient population studied. This incidentally matches exactly the overall early postoperative mortality rate in the U of I series. The causes were congestive heart failure secondary to an MI and massive pneumonitis, 1 pulmonary embolism, and atelectasis with pneumonitis and empyema. In addition, there have been a number of different complications. The major complications included one (0.9%) GI leak, two (1.9%) subphrenic abscesses, four (3.7%) wound infections, two (1.9%) dehiscences, and one (0.9%) evisceration. There were seven other minor complications consisting primarily of atelectasis. There also was one obstruction that resolved on its own. Because of the small number of patients currently in the Registry and the wide disparity between postoperative time of followup, it is somewhat difficult to compare weight loss data. The overall average weight at time of operation is 121 kg. However, some surgeons that contributed information to the Registry operated on patient groups with a significantly higher or lower mean operative weight. In spite of this, preliminary analysis seems to indicate that weight loss for the Registry patients is similar to the weight loss that has been
reported at this workshop by the surgeons with larger series of patients. As the Registry grows more information will become available and will be reported. In the meantime we would like to extend a warm invitation to all those interested in participating to join us.

DR. MASON: In addition to the Iowa City patients there are two groups of patients outside of Iowa City that have received a questionnaire. We have attempted to select a period of time so that the length of followup would be comparable in each of these three different practices. We also have as a control our questionnaire which we sent to patients who were in the first year, second year and more than two years postoperative period. This gives us some idea of how these questions are answered and whether there are changes with time. I'll show you some surprising results: Group A is a suture anastomosis, Group B is a stapled anastomosis. Group A has by and large a rather small anastomosis, approximately 12 mm, while Group B anastomoses are about 3 cm in diameter. Thus the main difference is between a big stapled anastomosis and a small sutured anastomosis.

We asked the patients whether they had constipation frequently, occasionally, rarely or never. Although this type of questioning can be criticized it must be remembered that it was asked in a questionnaire that was mailed and came back from each patient without any coaching. I think it may have some validity simply because of the way it was sent out and answered and sent back. It looks as though constipation is a problem during the first year in our patients with small stomas. This also has some validity since one would expect a small stoma to cause more constipation than a big stoma. Over a period of time, after the first year, this doesn't seem to be so important in the frequent category but it still shows a difference in the occasional category.

The counterquestion is, of course, about diarrhea, and as you might have predicted, the patients with the big anastomoses more frequently had diarrhea. I'm just charting this as an illustration of the type of thing that I think we could do cooperatively if we get together on this. In these modern days of computers, etc., you don't need to worry too much about how much data you have to handle.
Now here's a surprising thing. Many of the patients in our group claim to have vomited blood which suggests that perhaps the small sutured anastomosis is not so good. Our fears are corroborated by the affirmative answer to the next question: Do you ever have coffee-ground emesis? It appears that there is something about the small sutured anastomosis that is causing some bleeding. These are patients that have been followed. They don't have signs or symptoms of ulcer, there was no indication to look for a stomal ulcer, but they may have stomal ulceration and we've got to get those patients in now and do some endoscopy and some further studies and see what is going on.

There is also a difference between our group and the others in regard to tar-colored stools. It certainly is consistent with loss of blood in patients with small sutured stomas. I'm always very ready to develop hypotheses, and my hypothesis would be that you can't do a suture anastomosis as accurately as that machine can do a staple anastomosis. Perhaps in all these years, we haven't learned how to make a good anastomosis and maybe we've got some ischemia and chronic inflammation and chronic ulceration. It doesn't amount to much and maybe it's not clinically important, but it needs to be looked at.

DR. GRIFFEN: I've got another hypothesis. You've got some gastritis there.

DR. MASON: Gastritis? Why do we have gastritis and that other surgeon doesn't? What are we doing differently?

QUESTION: Are you going to correlate these findings with hemoglobin determinations?

DR. MASON: Well, yes, we certainly are. These patients are not, as far as I know anemic, but we've got to go back and very specifically look at that.

QUESTION: Do you know what the timing is to their vomiting? It may be that they vomit and produce a minor Mallory-Weiss sort of tear.
DR. MASON: It could be. At least we have some more questions to answer.

QUESTION: Have you sent that same sort of questionnaire to a bunch of student nurses or to another kind of control population?

DR. MASON: That's a good suggestion. As yet we have not sent it to people that don't have a gastric bypass or any kind of an anastomosis.

QUESTION: Have you sent it to people with the stapled anastomosis that is made very small?

DR. MASON: There is one man who is sending this questionnaire out who has a very small stapled anastomosis and that may be a way of trying to dissect out whether it is the size or the way the thing is put together.

COMMENT: You know, in the choledochal-small bowel anastomosis, a small anastomosis causes trouble in the common duct and the large anastomosis doesn't. The theory that people propose about that is that there is free flow back and forth and that might be something we should all think about.

DR. MASON: There certainly is a lot of precedence in the medical literature for not creating partial obstructions, and maybe we are paying a price for the weight loss, a price that perhaps we don't need to pay or shouldn't pay.

QUESTION: Could we ask Dr. Griffen whether he has seen anything unusual with his Roux-Y's?

DR. GRIFFEN: I haven't sent out the questionnaire, but I haven't been impressed with this. I think a tiny anastomosis and bile in the stomach can probably cause more gastritis than someone who has free flow and a wide open anastomosis. I think Bill's suggestion is very good. If you look at the weight loss in that first six months it is considerably more than the questionnaire group. I think it was 4 kg in the first six months for the questionnaire group and the Iowa group was about 20 kg, so obviously that is why they have more constipation. They just simply
aren't able to get enough down through the GI tract to have normal sized bowel movements so I'm sure they probably are vomiting more than the people who have a wide open anastomosis.

COMMENT: I can't believe that those statistics are that significant in terms of the patients you have mentioned. If your patients were being followed and were passing blood in the toilet or vomiting blood I am sure you would have heard about it from the patients without the questionnaires. The chances are they have interpreted the question to mean, "Have you ever vomited blood since your operation?". It may have occurred in one single episode six months ago and never again. I think otherwise you would have known if your patients were vomiting blood or passing tarry stools.

DR. MASON: We do keep reworking these questionnaires, and it's almost as tough to prepare a good questionnaire as a good examination question. In fact, it is the same sort of business, and, of course, some people think examinations never test anything. But it seems to me that even as crude as this is and as inaccurate as it is, if we include enough patients and groups and begin to see some sort of a pattern, at least it gives us a basis for further investigation of selected groups of patients. Often what happens in research is that you have a hypothesis, you start testing it and then you run onto something you didn't even suspect. At least we will have a basis for further studies on some patients instead of just running out and collecting data willy-nilly, which is what we have done to a great extent so far.

QUESTION: At the rate you are gathering statistical information here, what is your estimate that you will get some valid change in your statistics or opinions that are worthy of transmitting?

DR. MASON: When are we going to fish, cut bait or go ashore? I hope that we can begin to provide some hard facts about a few things very shortly. I hoped that we could provide it at this meeting, but it seems as though it's always a scramble just trying to get the information in, get it punched, get it on tape and then we can barely get anything run and get the slides made the day before the meeting, to be perfectly
honest. But I think we are beginning to get into a position where we can provide some solid answers.
QUESTION: How about something that is usually avoided and controversial? What is a reasonable surgical fee for this?

DR. MASON: We don't want anyone to think we are setting fees through this meeting because we are not. We charged $1,000 for the operation over the years, and then in recent years there has been a little increase and I guess it is about $1,300 now. How about some of the rest of you?

ANSWER: $2,000.

ANSWER: $1,000, but that includes following the patient for a year.

ANSWER: Same thing.

ANSWER: I guess we're the pikers. We only charge $900.

DR. MASON: It always seems as though we ought to be trying to make it easier and cheaper. One way to do this, of course, is to reduce your fees. Another way to do it is to cut out unnecessary workup and unnecessary hospitalization. We might talk a little bit about that. What would you think of doing away with upper GI's as a part of the initial workup? I see some heads shaking one way and the other. We have done a lot of upper GI's and we haven't seen much.

QUESTION: How many of them stopped you?

DR. MASON: None have ever stopped us.

QUESTION: How many of them gave you information you needed at the time of the operation that you couldn't find out yourself?

DR. MASON: I really don't think they've been of much help to us.

DR. KRIDELBAUGH: I wouldn't want to give up upper GI's because I wouldn't know whether or not these patients reflux or have hiatus hernias. After
listening to what Drs. Griffen and Buckwalter said I would like to try to individualize and certainly if anybody refluxes I'm going to think about a Roux-en-Y. We have an arrangement with the insurance agency in New Mexico where if I send them to the hospital within five days of the time of admission, all their laboratory work done preoperatively is plugged into their entire hospital bill. This saves an awful lot of money in the preparation of patients in terms of total bill for the hospital side of the procedure.

DR. MASON: Are some of the rest of you able to do that?

ANSWER: Blue Cross will do it in our state.

DR. MASON: An upper GI isn't a very good test of reflux. I think maybe a better approach is to take a good history and if they have any suggestion of reflux, then use the fiberoptic scope.

QUESTION: Would that increase your price?

DR. MASON: I suppose it would.

QUESTION: Do you do your own endoscopy?

DR. MASON: We do our own endoscopies, but then we have the cost of the instruments and nursing help and we end up with all of the same expenses.

QUESTION: I think what is important is how to manage the complications which can be so disastrous, specifically anastomotic leak. I had one two months ago and at that time it was my thought to drain it and then go in there and do that Roux-en-Y that Ward is doing and get that bile and pancreatic juice out of that corner.

DR. MASON: Ward, what would you do? Suppose the patient had a loop gastroenterostomy, would you think in the presence of a leak you could convert that to a Roux-en-Y with any benefit?
DR. GRIFFEN: I wouldn't. I've had similar situations, not in the obese, and I just close off the stomach, close off the jejunum, if you've got a bypass put a tube in the bypassed segment, and get out of there and come back another day. If you've already got a leak in an anastomosis along the suture line, I can't believe that putting a new suture line in that situation is going to heal any better.

DR. MASON: I think for the most part that we have tried to close the leak or we have resected the area that looked non-viable and resutured it, but then we put a gastrostomy in the distal segment. We put in feeding and suction enterostomies. I use Witzel enterostomies. That would be a suction enterostomy with an 18 French Malecot catheter in the distal pouch so that it won't perforate the wall of the stomach. I use a long Witzel tunnel and suture the catheter to the skin. The tube is sutured with an absorbable suture at the site of entry into the bowel or stomach such that if the tube gets pulled out it won't tear a hole in the bowel and leave another leak. We have had this problem when a patient ripped a tube out and left a hole, even though there was a Witzel tunnel. You have to be careful where you are passing that tube into the bowel. Then I use a 16 French Malecot for a feeding enterostomy.

(Added on November 14, 1977: At the suggestion of Dick Simmons of the University of Minnesota we have read the paper by Hudspeth on radical surgical debridement in the treatment of advanced generalized bacterial peritonitis in Arch Surg, 110:1233, 1975. This approach has been used in one patient with great success. Fibrin is peeled away from all peritoneal surfaces. The abdomen is extensively irrigated with saline. The leak is excised or repaired. Hudspeth does not drain the cavity. Antibiotics are used. Postoperative fever is controlled with a cooling blanket and a respirator is used to support optimum gas exchange.)

QUESTION: Have you patched any of them with an on-lay patch?

DR. MASON: That's a good suggestion. I've patched them with omentum.

ANSWER: The suggestion of converting that to a Roux-en-Y could work with a patch by dividing it down below, leaving a couple of inches,
anastomosing the afferent loop to the jejunum below and using a couple of inches to patch your blowout.

DR. MASON: That might be a good solution. I had a patient with an afferent loop syndrome who was totally obstructed and I didn't recognize the facies and what not, so eventually I operated on her. What I found was that she had blown out the inferior portion of her duodenum right at the peritoneum and it had drained down into the pelvis, as you mentioned. She was complaining of pelvic pain and I thought she had a urinary tract infection. Along with her tachycardia she had fever. What she had was a necrotic looking stomach with the second or third portion of the duodenum blown out. Boy, I really scratched my head as to what to do. I couldn't do a Whipple on this woman, she weighed 250 lbs., and I didn't know what to do. So I basically just tacked things together and drained a lot and sewed up the holes. She eventually died of chronic peritonitis and respiratory failure. She had anuria and the whole business.

DR. GRIFFEN: Well, I'll tell you what you can do. If you've got that long a loop you can bring out the afferent limb as a duodenostomy, close off the stomach and bring out the other limb so you can feed the patient and you won't have any tubes in them except the tubes you put down to drain them. I've got a patient right now with a gunshot wound that has exactly that. The distal part of her stomach is shut off, her duodenum is closed, and about 15 cm distal to her ligament of Treitz she's got a jejunostomy which is draining her bile and pancreatic juice out of the peritoneal cavity. I think you've got to get the bile and pancreatic juice away from all those places because that is the thing that is going to eat up anything you do. You can't put sutures in any of that and expect it to hold. It will just perforate again and then you are right back where you were, only you're further down the pike.

DR. MASON: The time factor is extremely important. Every patient we operate on is perforated, but it doesn't make any difference because we close the perforation right away. I mean, that's part of the operation, to open the viscus and close it. They're all perforated. You can have a perforation that's present for six hours and no problem, even 12 hours
works pretty well, but if you fool around looking for pulmonary embolism and one thing and another for a couple of days, you're really in trouble and likely to lose the patient or have the patient in the hospital for weeks and weeks with terrible problems. I don't need to go into all the complications, but .... I think if we could just bring ourselves to admitting that maybe there's a leak and reexploring the patient, even if on occasion we find that we were wrong, it would be better.

QUESTION: How often have you explored and found nothing?

DR. MASON: We've had one negative exploration. We have usually found something. It's just like an appendectomy. How many normal appendices do you remove?

QUESTION: Do you, or any of the other people who are doing a significant number of these, try to raise some of the omentum up over your staple lines?

DR. MASON: I don't because in past experience in other situations when I tried to do that it has ended up with an obstruction of the anastomosis. I'll let the omentum lie there, but I don't suture it.

QUESTION: When you've reoperated early, how obvious are the operative findings?

DR. MASON: Usually you have quite a quantity of obvious intestinal or bile contents in the peritoneal cavity, and just as soon as you open the incision you see it.

QUESTION: Can you find the holes?

DR. MASON: You usually can. I think the problems are in those instances where you have a distal closed loop obstruction and you get into the situation in which there has been ischemia of the whole duodenum, the distal pouch and everything. It's all rather bad and you don't know where it's leaking, and maybe there isn't a real obvious hole.
QUESTION: Have you had any more leaks with staples than with sutures?

DR. MASON: John, the question is "Do you have leaks with staples more than with suture lines?" We know what his answer to that is, he hasn't had any leaks. He's had 270 cases.

DR. ALDEN: I will say I feel like I'm walking on eggs and I just hate the thought that one of them is going to leak one of these days, I dread it. I don't think that staples are the problem as far as causing leaks.

COMMENT: I think we need more publications in the internal medical literature about the procedure. We're physiologists basically as surgeons. We understand the basic etiology of how it works, but it is very hard to convince some of our internists and gastroenterologists that this is a physiologic operation. There is a tremendous amount of resistance.

DR. MASON: I think sometimes maybe we have written too much about it. People sometimes complain because there is too much in the literature from Iowa and not enough from other places. I think the rest of you need to add to it. Are you suggesting that we need to publish in the internists journals?

ANSWER: That's right, gastroenterology journals and internal medical journals.

DR. ALDEN: In a different light, I wonder if we could come back to the problems of cost. Some of the workups are so enormous and so expensive. Five days in the hospital seems completely out of order for a 280 pound young healthy patient. An older patient of 400 or 500 lbs is an entirely different situation. I think we should establish different categories of risk and protocols for those categories. With a routine workup that costs thousands of dollars and days in the hospital, the waste is on some patients who really don't need it.

QUESTION: What would your total hospitalization cost be for the procedure?
DR. ALDEN: Once in a while when the patient doesn't have insurance, and I have not charged a fee I have been able to get the patient in and out of the hospital for less than $2,000.

COMMENT: That depends on the rates of the hospital.

DR. ALDEN: I guess what I'm trying to get across is, and I may be wrong, that we should not use a routine workup that isn't necessary in everybody. In other words, that protocol is good but there is nothing in there to say that we need a blood oxygen on every patient. We don't need an upper GI on every patient. We don't need all kinds of things on every patient. I think we need a chest x-ray, an electrocardiogram, an SMA 12, CBC and a type and crossmatch, but I think by listening to the patient and observing him you should be able to categorize where we spend money and not always spend it just because it is part of a routine we have established to keep up with the most expensive routine that has been established for others.

COMMENT: I would agree that there have been enough studies now to know what is unnecessary but I must say that arterial gas analysis is one of the things that I would not drop for these patients.

DR. MASON: Would you get arterial gases on everybody? I mean, there are a lot of patients, young patients, that are twice ideal that can run up and down stairs.

ANSWER: I still would get arterial gases. That's cheap compared to some of the other things that we get.