a blood examination made, and most of the doctors in Alabama are able to do it, and are doing it. During the past few years the physicians who have referred these patients to me are sending them in early, and it is rare, indeed, that any are brought in during the last stages of the disease. I have seen people brought in and die within a few hours without operation, their condition being so extreme when they reached the hospital. And they are dying now from purgatives, and we know it. When a man says it is a baby act to say that purgatives produce peritonitis, he is contradicting the statistics of the best observers in the world. The figures show that only $3\frac{1}{2}\%$ of those who were given purgatives escaped perforation, and they also show that less than $1\frac{1}{2}\%$ of those who had the Murphy-Oschner treatment from the inception of their appendicitis had either a perforation or a peritonitis. (Applause.)

Monihan says that spreading peritonitis is an aperient peritonitis. The purgative makes the pathology. Without purgatives, but with a morphine, peristaltic rest treatment from the beginning of their attack, they do not have perforation.

In the twenty-two and a half years that I have been doing surgery, my appendicitis cases have run into the thousands, perhaps. I have seen only one case that had a perforation without a purgative being administered previously. He was operated on within eleven hours from the onset of his first pain, and he had a perforation. This was the only one that I have seen who did not have a purgative and who did have a perforation.

The fact is that 75% of all the cases of peritonitis that come into the hands of the surgeon are due to appendicitis—the 25% remaining arising from all other causes. Eight per cent of the people who are attacked with appendicitis die under the present regime, when not 1% ought to die. I did not say that the general practitioner was responsible for it. Ill-timed operations will add to the pathology, not only in point of time, but in extent of operation.

The principal thing that this paper has dealt with is prophylaxis of peritonitis from appendicitis. The imperative thing to do is to give the patient the advantage of the present-day scientific knowledge, and if he will not accept the advice given as a result of using all of the means indicated to help his condition, in that he should be operated on immediately, then turn him over to some doctor who believes in calomel and salts (if there be any in this day and generation) and let him sign the death certificate.

We hear about drainage, tubes and gauze. I wish to warn against the use of either tubes or gauze. The ordinary dental dam will get out more fluid from the abdomen than any other form of drainage, and will be conducive of fewer post-drainage adhesions. There is less liability of fecal fistulae or pressure necrosis than follow the use of drainage tubes.

There is something I want to say about the post-operative treatment of these cases in closing.

In these septic conditions there is a drying up of the tissues and an acidosis that hastens death. To combat these an intravenous solution of saline and the hypodermoclysis of distilled

water should be used. I have tried the distilled water under one breast and the normal salt solution under the other, simultaneously, and the distilled water has been absorbed twice as rapidly as the saline. In desperate cases the hypodermoclysis should be started as soon as the patient is brought to the operating table.

With reference to the anesthetic: Infiltration anesthesia and nitrous oxide narcosis should be used. The severer cases should have the major part of the operation done under infiltration anesthesia, which can be employed without any trouble to operator, or pain to patient, if you have given him previously a masking symptom dose of morphine. (Laughter.)

In the presence of post-operative ileus, the doing of an enterostomy under local anesthesia is frequently a life-saving proceeding and should under no circumstances be neglected.

CONCLUSIONS IN THE STUDY OF INTESTINAL STASIS.*

By JOSEPH E. JOHNSON, M.D.
Memphis, Tenn.

To develop into an intestinal surgeon is a process of evolution. We begin by removing the appendix for all pains in the right side, and feel ourselves quite a surgeon. As we pursue our practice longer with the same clientele, we see a goodly number returning with no relief. We begin to observe more closely, we discover adhesions with which the appendix is blamed. Later, we learn to diagnose the different ptoses of the viscera and in these cases we note the obstinate constipation and the violent toxemia. After a futile attempt to relieve these patients by medical means, we advance into the next stage, the mechanical stage. Then we attempt to correct the ptoses by suturing the stomach and colon to the parietal wall, to hold them near their normal position. Dr. Morris says that by the time he learned to do the operation well he discarded it, and his observation has been that of most surgeons who have given this subject serious consideration. In my early replacing or hitching up operations it was unsatisfactory to do it through a median incision, so I conceived the idea of making a large transverse incision down to the rectus muscles and, by raising these from their sheath, continue through the

*Read in Section on Surgery, Southern Medical Association, Ninth Annual Meeting, Dallas, Tex., Nov. 8-11, 1915.

peritoneum in the same line, thus giving an incision where I could do a first-class hitch-up operation. I did this a number of times, for which I have to apologize to every one for whom I have not taken it down and resorted to other means. The mechanical stage in this evolution has produced some operations which, from a mechanical standpoint, seem to be efficacious, but from a physiological standpoint are worse than useless. Ileosigmoidostomy is an example. This mechanical stage marks the blackest page of disappointment in this field of surgical endeavor. Observing our failures by our mechanical means, we advance to the next stage, which is a study of the physiology of the intestines, and note the failures, embryonic and otherwise, which cause the trouble; then we are well launched into the study of toxemia. The accepted idea of stasis is delay, so let us study this from a standpoint of delay. It is at the end of our study that we get our first real conception of the subject in hand.

I will, necessarily, quote frequently from my own paper read before the Tri-State Medical Society of Mississippi, Arkansas and Tennessee (1914), as from this study I wish now to give the conclusions reached.

Let us look at the intestinal track as a whole. We will observe, first, that at the lower portion or orifice of each division of the intestines we have a sphincter, which is so placed for a very definite physiological reason. The function of the pylorus, normally, is to retain the food in the stomach a sufficient time for it to be acted upon by the digestive juices. When it has reached a certain degree of acidity, the acid in the stomach stimulates it to relax and allow part of the stomach's contents to pass into the duodenum. When the acid comes into contact with the mucosa of the duodenum, the pylorus is stimulated to contract and remains contracted until all acid in the duodenum is neutralized, when it again relaxes, and this continues until the stomach is empty and all of the acid in the duodenum has been neutralized. Then the pylorus will lie open and the bile and duodenal contents will flow back and forth through it without causing disturbance. Water almost immediately passes through the pylorus.

The vagus stimulates the pylorus; the splanchnic inhibits it.

The ileo-cecal sphincter also has a double function. First, to hold back the digested proteins a sufficient time for absorption; and second, to prevent the regurgitation of the contents of the cecum into the ileum. The anal sphincter retains the contents of the colon until a convenient time for emptying.

The peristalsis of the stomach is well known. The peristalsis of the small intestines is a wave of dilatation followed by a wave of contraction which is regular and continuous. It is very little, if at all, influenced by cathartics or stimulants. It is controlled by a local nerve mechanism in the walls of the intestines. In the colon we have peristalsis and anti-peristalsis. With a giant peristalsis wave the contents of the cecum may move forward to the splenic flexure, or with a wave of anti-peristalsis be brought back an equal distance. Anti-peristalsis prevails in the proximal half of the colon. Forward peristalsis prevails in the distal half. The stomach is a receptacle where food is received and prepared for digestion. It should empty in four and a half hours. Food delayed in the stomach, beyond this time, will undergo putrefaction just as definitely as if it were removed with a stomach tube after four and a half hours and placed in a pan under the radiator. Patients with stomach delay are always eating bad food. The small intestine is the great eater and the terminal ileum has been proven to be the great absorber. The colon is the sewer; its mucosa has its natural defenses against the contained bacteria. It absorbs practically nothing but water. It will absorb a small amount of amido acid. Albumens or peptones by enemata are not absorbed. The mucosa of the intestines below the oesophagus is insensible to heat, cold, or pain. The peritoneal covering and even the intestine itself may be incised without pain, but pulling upon the peritoneal covering or its mesenteric attachment gives intense pain. This should be remembered before making adhesions to the parietal wall.

According to Lane's theory, the product of a finished digestion, if delayed a short time in the terminal ileum, will reach the next stage, that of nutrefaction, the absorption of which is the source of

the toxemia which in turn causes visceroptosis. I agree with Lane as to location, but differ with him, radically, as to the source of the toxemia.

Now, let us look at stasis in the accepted sense of delay and apply our study of physiology. There is one physiological fact with which we are all familiar, and that is that if you irritate the intestinal track at any point, all the sphincters above, beginning with the nearest, will make an effort to protect the irritated part against the contents of the intestines above. To illustrate: we take a case of chronic appendicitis which is causing a great deal of trouble. There is disturbance in the upper abdomen which is so pronounced that we make an incision so as to explore this region. When we remove such an appendix we find no inflammation or infection. It may be atrophied and kinked. We are frequently in doubt as to whether we have removed our pathology when we remove such an appendix. The patient is subject to so-called biliousness or spells of toxemia and has lost weight, also has the nervous disturbances which go with the toxemia. We have all noted the rapidity with which these patients return to health and weight after removing such an appendix. We have all noted that in cases of hemorrhoids or ulcerated rectum the picture is very much the same as in chronic appendicitis—loss of weight, general toxemic condition—and upon removing the irritation the return to health and weight. Now, why?

In the cases of kinked appendix, the irritation of such an appendix causes the first sphincter above it (the ileo-cecal) to make an effort to protect the irritated part against the contents of the ileum—we have an ileo-cecal spasm, which may persist even as long as twenty-four hours. With the ileo-cecal spasm and the forward peristalsis in the ileum, we have a dilatation of the ileum as in an obstruction. The pain is not in the appendix, but is an ileal colic. Secondly, as in a block system on a railroad, from the irritation in the ileum we have a protecting pyloric spasm. Thus, from an appendix which in itself shows very little trouble, we may have a decided ileal stasis and a secondary stomach stasis. This will explain the entire condition. The

same condition prevails from an irritation of the intestine anywhere from the anal sphincter to the pylorus. There is unquestionably a co-ordination, under irritation, between the ileo-cecal sphincter and the pylorus. Food taken into the stomach will cause the remaining contents of the ileum to pass into the cecum (photo) and it will overcome the spasm of the ileo-cecal sphincter. We recognize cardia spasm. We have pylorus spasm due to irritation of the gall bladder.

The toxemia, which we have from a delay in the stomach, in the ileum, or the colon, is the toxemia which gives the dull headaches and a general ill feeling, the so-called biliousness which a cathartic will relieve for the time being. Let us call this delay toxemia.

Let us look at visceroptosis or splanchnic paralysis from another standpoint. Take a patient, a young woman of seventeen, who is neurotic, fairly well nourished, subject to toxic sick headaches from early childhood, with repeated attacks of soreness in the cecal region, without colic. We give her a bismuth meal and find she has visceroptosis. Her stomach empties on time, the small intestines empty on time, the colon, though prolapsed, empties on time. The appendix is patulous. *There is no stasis*, and still the toxemia is severe. We remove the appendix, straighten up a uterus which is generally retroverted, noting that the cecum is large and movable, fine veils or membranes covering the cecum, ascending colon, and the terminal few inches of the ileum, and she is not relieved. See this patient ten years later. The condition has steadily progressed until all of the viscera are piled down into the pelvis. She has been treated by every system, medical and non-medical; she is the dread of the doctor's office. As Moynihan expresses it, "she is of attenuated form, she is querulous and introspective." She has cold hands and cold feet; she has all the symptoms of profound toxemia. If we should look, then, at this same region, we would find the Jackson membranes, veils, possibly Lane's kink, and the cecum and ascending colon so distorted that it is practically a mass of diverticular protruding through these membranes. If we stop to think we never see these membranes except in this locality, and always involving the

same structures. The case history is characteristic, the visceroptosis is as definite and uniform as in any other condition with which we are familiar, so we must conclude that there is one definite etiological factor which is always responsible. Now, what could produce such a condition? The membranes and veils are only nature's effort to build stronger walls to protect against infection. The appendix, in nearly all of my cases, had been removed with no relief.

Hertz, in 1897, described an ileo-cecal regurgitation. Case has, later, verified the finding. If we have an imperfect ileo-cecal valve which will not prevent regurgitation, this will answer our question fully. Let us look at the condition which would then prevail. On one side of the cecum, which is the cesspool of the sewerage system, with its contents one-half by weight bacteria, the other half the putred products of decomposition, we have peristalsis and anti-peristalsis in the colon. On the other side we have the great eater, the terminal ileum, which has not the natural defenses against bacteria. With an anti-peristalsis, we have a regurgitation into the ileum; we have a toxic poisoning plus a bacterial infection. The patient is literally feeding upon her own excreta. The toxemia or poison causes a splanchnic paralysis, which is just as characteristic of this poisoning as dropped wrist is of lead poisoning. The bacteria are taken up by the lymphatics in the ileum and produce a peri-ileitis, involving the last few inches of the ileum, and a peri-colitis. The infection to this patient is attenuated, and this is why the veils are soft. The veils below the ileum may become rotated and form a narrow band, which is termed a Lane's kink. It is all a part of the same process, the same cause which gave the violent sick headaches in early youth, the same toxic poisoning which has produced the last named condition. I do not think there is any doubt that in all these cases the trouble is a congenitally imperfect ileo-cecal valve. Coffey described a cecum mobile of congenital origin. He is right. Dr. Morris suggests a taint of degeneracy. He is right. I have observed a mother, a daughter and a granddaughter who have visceroptosis, and there are probably other cases in the same family. Is it not quite

possible that the shape of the internal organs may be just as much of a family characteristic as the external features?

The thickened terminal ileum for which Lane operates, and which he blames for the condition, is the end result of a chronic infection. It is not the cause of the trouble, but the result. It is my belief that if the regurgitation infection and toxemia poisoning can be relieved, the splanchnic paralysis will recover just as dropped wrist recovers when the painter stops the source of the lead poisoning.

I present you a picture of a case operated upon by myself one year ago. She had a visceroptosis, some fine veils, and an appendix which was patulous. In this case I did an ileo-ceco plasty, as described in my paper of 1914. I also present pictures demonstrating an ileo-cecal spasm, which is overcome by the stimulus of taking more food. In these advanced cases we have a distorted and infected colon, a thickened terminal ileum. Nothing short of a right side colectomy will be of any avail. I have operated upon eleven such cases, ten of which were successful, showing that the operative mortality is not prohibitive, and when we consider the hopeless condition of these patients and the wonderful relief obtained, it would be justifiable with even a greater mortality. When you look at these two pictures you will readily see that you are able to recognize very nearly the age of the patient.

As a differential diagnostic point, repeated soreness in the right side preceded by toxic headaches without colic, is not appendicitis, but regurgitation infection. In appendicitis we always have the colicky pains.

In conclusion, let me emphasize that there is a toxemia from delay which is quite different in its action from the toxemia of regurgitation. The toxemia and infection of ileo-cecal regurgitation is the cause of visceroptosis and the formation of the inflammatory membranes. It has been pointed out by several of our distinguished surgeons that, though the viscera was much displaced, it still emptied on time. This is true of all regurgitation infections and toxemia in the earlier stages. We must look upon this as a progressive condition, the origin of which is congenital.

DISCUSSION.

Dr. Louis Leroy, Memphis, Tenn.—I feel, in a measure, out of place—an internist discussing a surgical operation—but I feel that the biggest field today that requires combined study of the surgeon and the internist lies in this study of stasis and reflex obstruction, because, in the first place, the internist is also always the man who is worried to death and has been with these cases of a chronic nature. Four out of five, I believe, of our chronic neurotics, and, you might say, our habitual invalids, may trace the foundation of their troubles to absorption occurring in the ileum of materials which have already left the ileum and come back, or which are being held by either visceroposis or other obstruction in some other point.

It is simple enough to class the whole thing as autointoxication and think you have said it all and let it go at that, but that is scarcely the beginning.

In this principle of reflex association of different groups of sphincters we have the explanation of many of the ileum cases. You have, all of you, I am certain, operated for appendicitis on some chronic or semi-chronic cases and failed to get anything like relief in your patients. They still have the digestive troubles, loss of appetite, chronic grumbling pains, gaseous distention, loss of weight, general mal nutrition, which follow in the wake of some cases. You attack the case, remove your appendix, and expect to get the brilliant results you have gotten perhaps in many previous cases. In this case you will not. Why? Merely because there is some irritating focus at the region of the ileocecal valve which is reflected to the pylorus, and is giving pyloric spasm from the other end of the intestine. Of some of your gastric ulcer cases the same thing is true. You attack a gastric ulcer from the surgical standpoint, after your medical man has failed, and you are sure as soon as you get drainage your patient will be all right, but this may not be true. If you go further you find some of this same pathology in the ileo-cecal region, and had this been corrected, even without ileo-cecal drainage, you would have cured the ulcer, perhaps. In other words, reflex pyloric spasm, originating in irritation in the ileo-cecal region, will give you that retention of acids in the stomach which requires but an added detail to start the ulcer and to maintain it. You will gain nothing by draining that stomach unless it has gone on long enough to give you absolute pyloric obstruction or gastric dilatation. An ileo-colostomy may permit a relaxation of the pylorus; you will not be retaining your excess of acid, and from a distant location your stomach condition will improve, and without attending to that you will be disappointed in some of your gastro-intestinal work.

The same is true in rectal conditions.

Dr. Isidore Cohn, New Orleans, La.—I would like to ask the doctor a few questions. Did I understand you, doctor, to say that the pain you have in some of these cases is due to pain within the ileum?

Dr. Johnson—Neal colic.

Dr. Cohn.—My understanding of the physiology

of pain in the intestine is the following: I believe a few years ago it was pretty clearly proven that the nerves of pain are in the parietal peritoneum. If that is the case, I want the doctor to explain how this comes about without pressure on the parietal peritoneum. The doctor told us about removing these kinks and curing the patients. What makes these kinks if there is no bend, no congenital membrane, no inflammatory process, thickening of walls or interference with the lumen of this intestine?

The doctor also said if there was any one here who had ever seen congenital membranes without visceroposis to holler, or visceroposis without congenital membranes, to holler loud. I have seen both, and some of these congenital pericolic membranes seem to act very much in the same way as the ligament of the liver, preventing ptosis instead of causing ptosis. The doctor also said if these things were there not to touch them at all. Sometimes if they are not doing harm it is a good thing not to touch them, but if the patient has them and there is not any other pathology to explain the condition probably touching them a little bit will help the patient.

Dr. J. S. Ullman, Natchez, Miss.—I have seen some cases of Jackson's membrane where there was no ptosis.

Dr. A. B. Small, Dallas, Texas.—I shall not detain you long. I am fully aware of the objectionable features to presenting pathological specimens away from a laboratory; but I am going to beg your indulgence, since I have a very unusual specimen in connection with a most interesting case.

Patient L. F., thirty-two years of age, young farmer from near Blooming Grove, Texas, came into my service one week ago accompanied by one of his neighbors, who informed us that he was a hopeless epileptic.

The patient, who is thin, of a bad color and inclined to be melancholy, stated that his local physicians had informed him that there had been some favorable reports in the literature following an intestinal operation in epilepsy; and that he wished to take the operation if it offered any hope of a cure; if not, he thought he might as well take a shotgun.

The history of the case is, briefly, as follows: Constipated since he could remember; at the age of fourteen he began having spells of unconsciousness every few months, the attacks increasing in severity as the years went by, and the last four years they have been violent, coming on every ten days to two weeks, when he would have from one to twenty convulsions in twenty-four hours.

The attacks were invariably accompanied by a soreness in the abdomen which persisted until the bowels could be well emptied, an undertaking which at times proved very difficult. I frankly told him that I would not promise a cure in his case, but that I would assume the responsibility of the operation per se, and that I thought he could be benefited. He agreed to become a clinic for the Southern Medical Association, so that any member of this society who cared to do so might follow up the results of the operation

in the future. So this morning at the Baptist Sanitarium, in the presence of some of you whom I beguiled, we opened his abdomen to find a well marked developmental error.

The cecum and ascending colon in its descent from the subhepatic position in embryo had failed to fuse with the retro-peritoneum, a condition designated by Wilms as "Cecum Mobile," an error not so infrequent; but here the condition is complicated by having the terminal end of the ileum adherent to the retro-peritoneum at a point over the psoas muscle, where the cecum is shelved following a normal fusion and rotation, thus preventing the ileum from entering the cecum at right angles with the body from the left. The stricture caused by this strained position can readily be seen when I hold the specimen up thus. It is about five inches removed from the ileo-cecal valve, where the ileum enters the cecum at right angles with the body antero-posteriorly.

The caliber of this structure is about the size of a small lead pencil and was entirely covered over by the cecum. Just at this point, about eight inches from the ileo-cecal valve, may be seen a denuded area on the ileum. This is the point of origin of an ileo-pelvic band (Lane's kink), the attachment of which was found on the pelvic floor. This structure makes tension across the mesentery, drawing a segment of the ileum into a V shape.

Still farther removed from the valve and including all of that portion of the ileum which was resected, is a dilatation due to damming back of the ileal flow by these two breakers shown so clearly in this specimen; one an intestinal congenital deformity, the other a definite mechanical obstructive band. The two, combined, have played havoc with this poor fellow's life, rendering him a recluse.

The operation done consists of the resection of about fourteen inches of the ileum, the cecum and ascending colon, and the proximal end of the transverse colon over to the middle colic artery. The resection being circumvented by an end to lateral anastomosis of the ileum with the lower loop of the pelvic colon. I am not prepared to say just what effect this operation will have on the convulsions in this case, but my opinion is the evil spirits have been cast out of the man by a proper drainage of the alimentary canal. Whether this will prove true or not, I shall be glad to keep you posted as to results through the columns of the Southern Medical Journal.

Dr. Johnson Closes.—I asked the question if any one had seen a case of Jackson membranes or inflammatory veils without a visceroptosis. I heard an affirmative answer from Dr. Cohn and another. Seeing a case of membranes at the time of an operation would signify nothing if not previously examined with a fluoroscope in the vertical position. When I made the assertion that I had not seen such cases, I meant where I had made fluoroscopic examinations.

Dr. Cohn asked if I made the statement that a perfectly normal appendix would excite an ileocecal spasm. I made no such statement, but stated that an appendix which was kinked or adherent and showed none of the outward evi-

dences of present inflammation will cause an ileocecal spasm. It is such an appendix which gives rise to the secondary stomach symptoms. In our limited time we can not go into the etiology of a kinked appendix.

I wish to show some skiagraphs illustrating an ileocecal spasm:

No. 1 shows the bismuth meal at the end of ten hours in the terminal loops of the ileum (the ileum should be emptied in nine hours). At that time he was given a full meal. No. 2 was taken forty minutes later, showing all of the bismuth in the cecum and first part of colon. When food is taken into the stomach the normal ileocecal

No. 1.
Bismuth Meal 8 a. m. X-Ray 6 p. m., 10 Hours
After.

No. 2.
Bismuth Meal 8 a. m. X-Ray 6:40 p. m., 10 Hours
and 40 Minutes.

No. 3.
Twenty-four Hours.

valve allows the contents of the terminal ileum to pass and in case of ileocecal spasm the spasm will be overcome by the same stimulus. Picture No. 3 shows the retrocecal appendix which was causing the trouble.

Let us remember that incising the intestines or peritoneum does not cause pain; that the pulling upon it causes intense pain. Also remember that the colicky pains in chronic appendicitis are not in the appendix, but are from the ileum dilating above an ileocecal spasm, thus causing an ileal colic. I think this explanation will prove to be true in the greater number of cases of chronic appendicitis.

PROTECTION AND CLOSURE OF INCISIONS *

By R. M. HARBIN, A.B., M.D., F.A.C.S.,
Rome, Ga.

Modern surgical technique has well nigh eliminated the question of mortality in elective operations, but surgical procedure, as frequently observed, has not reduced the period of morbidity to the extent in many cases that greater care and study would suffice to relieve, and from the standpoint of a spectator many of the larger clinics seem to ignore these apparently minor considerations, while delayed convalescence becomes of major importance to the patients; yet the outcome in the end being a satisfactory result. Haggard reports a death after an

interval operation of appendectomy from skin contamination. (Southern Medical Journal, November, 1915.)

With this apology I would like to offer a few remarks from a clinical standpoint. In the average patient, after complete sterilization of gloves and instruments, the adjustment of face masks, etc., the successful issue of a surgical wound depends on (a) protection from skin infection, (b) thorough hemostasis, (c) approximation of tissues without tension or strangulation.

(a) It is manifestly impossible to sterilize absolutely any exposed part of a living organism without damage to the tissues. While not attempting a technical review of the question of value of the various methods of sterilization of the skin, it is probable that iodine furnishes a greater degree of sterilization than any other method, but a consensus of opinion admits that there is an element of danger from infection after this method, and any antiseptic sufficiently strong to sterilize the skin would produce contact damage to the intestines.

In the absence of an ideal method of sterilization of the skin, our energies should be expended towards obviating whatever dangers that may lurk after the usual methods of asepsis have been practiced.

Common observation warrants the statement that the ordinary protective measures by towels and hooks start off well, but usually break down before the completion of operations attended by tedious manipulations.

In view of this observation and experience we have devised a method by using ten or twelve safety pin towel hooks strung on two pieces of tape about two feet long for each side, and these hooks at short intervals anchor a single or double fold towels over the edges of skin and prevents not only skin contact, but protects the underlying fat from the trauma of manipulations. Fat having scant vascularity is very liable to necrosis and infection and should receive careful protection, especially in long incisions. The towel may be folded in the middle and anchored so as to conceal these hooks by having an assistant grasp edges of incision with a towel and then transfix the towel and skin with hooks. Then the

*Read in Section on Surgery, Southern Medical Association, Ninth Annual Meeting, Dallas, Texas, Nov. 8-11, 1915.