As a rule, however, the patient should be entirely in bed and should be treated like a gastric ulcer. Duodenal ulcers are of the same nature as gastric ulcers, in that they occur in that part of the alimentary tract which is exposed to the gastric juice before its acid is neutralized by the bile and pancreatic juice. Cases of duodenal ulcer do well on the Lenhartz diet; did time permit I could quote a considerable number. I often combine with it $\frac{1}{4}$ oz. of olive or almond oil three times a day, following the method introduced by Cohnheim and by Walko, and advocated by Dr. Hertz. On this plan $\frac{1}{4}$ oz. of oil is given every three hours and increased to 1 oz. or 2 oz. Nothing else is given, except water for thirst, until there is no blood in the stools. Cream is then given, and the foods of the Lenhartz diet are added gradually, but not the rice. The oil is now reduced to 1 oz. before each meal.

I have tried this method thoroughly, and can recommend it for patients who will endure it, and not a few will. To others it is exceedingly nasty, and fails on that account. Whenever retching or vomiting is caused the treatment should be altered. If the patient does not respond to one or other form of dietetic treatment, the possibility of the symptoms being due to gall stones must be carefully reconsidered.

The indications for operation in duodenal ulcer are the same as in gastric ulcer. It is to be noted that a proportion of cases of ruptured duodenal ulcer have had no premonitory symptoms. As in gastric ulcer, a certain number of cases relapse after operation—25 per cent. in 28 cases operated on by Drummond and Rutherford Morrison and 15 per cent. in the series recently published by Mr. Moynihan, which includes many cases that cannot be regarded as severe. If those surgeons who advocate surgical treatment as a routine could do a year or two's work on the medical side of a hospital, they would learn that many cases get well as out-patients, and most of the remainder as in-patients, with rest and dietetic treatment.

REFERENCES.

- ¹ Bogen, H.: *Arch. f. d. ges. Physiol.*, 1907, cxvii, 150.
- ² Bolton, C.: *Proc. Roy. Soc. Med.*, 1909, II, iii, Path. Sect., 54.
- ³ Clairmont, P.: *Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, 1909, xx, 330.
- ⁴ Cohnheim, P.: *Zeitschr. f. klin. Med.*, 1904, lii, 110.
- ⁵ Drummond and Rutherford Morrison: *BRITISH MEDICAL JOURNAL*, 1909, ii, 67.
- ⁶ Gorhardt: *Zentr. f. Stoffw. u. Verdauungskr.*, 1909, N.F. iv, 336.
- ⁷ Katznelson, H.: *Arch. f. d. ges. Physiol.*, cxviii, 327.
- ⁸ Leube: *Deutsch. med. Woch.*, 1909, xxv, 961.
- ⁹ Quincke: *Ibid.*, 1882, viii, 79.
- ¹⁰ Rosenfeld: *Zentr. f. Stoffw. u. Verdauungskr.*, 1909, N.F. iv, 336.
- ¹¹ Saundby, R.: *Lancet*, 1891, i, 353.
- ¹² Sternberg, C.: *Zeitschr. f. Heilk.*, 1907, xxviii, N.F. viii, Supplement-Heft (Marz, 1908), 280.
- ¹³ Strauss: *Zentr. f. Stoffw. u. Verdauungskr.*, 1908, N.F. iii, 39.
- ¹⁴ Spriggs, E. I.: *Med.-Chi. Trans.*, 1907, xc, 283.
- ¹⁵ Spriggs, E. I.: *Proc. Roy. Soc. Med.*, 1909, II, iii, Therap. Sect., 81; *BRITISH MEDICAL JOURNAL*, 1909, i, 825.
- ¹⁶ Walko, K.: *Centr. f. inn. Med.*, 1902, xxiii, 1113.
- ¹⁷ White, W. Hale: *Lancet*, 1906, ii, 1189.
- ¹⁸ Winternitz: *Schmidt's Jahrb.*, 1906, ccxci, 235.

DOES GASTROSTAXIS EXIST AS AN INDEPENDENT DISEASE?

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It is well known that profuse haemorrhage may occur from the mucous membrane of the stomach apart from gastric ulceration. For example, in severe anaemias, in infective disease, in purpuric conditions, and in toxæmias associated with diseases of the liver and kidneys, it is a common experience to find the mucous membrane of the stomach intact when vomiting of blood has occurred during life.

In the condition known as gastrostaxis, however, there is said to be an oozing of blood from the mucous membrane of the stomach not only in the absence of any ulceration of that organ, but also in the absence of any other disease in which bleeding is likely to occur. Gastrostaxis is also said to possess a definite clinical history simulating that of gastric ulcer—namely, pain, vomiting, and haematemesis occurring in attacks in either sex, but

especially in young females. It seems to me that the actual existence of gastrostaxis as a common clinical entity is hardly established on a sufficiently sound basis, and that it is extremely important for the question to be definitely decided one way or the other, because it is obviously absurd to treat a case of gastrostaxis by operation, whereas in the haemorrhage of gastric ulcer life may often be saved by timely operative interference.

For some time my attention has been particularly directed to the difficulties in the way of proving that this disease exists, and my object in writing this paper is to emphasize these difficulties and to encourage others to pay especial attention to the condition of the stomach at all *post-mortem* examinations of patients who have died of haematemesis. I am continually on the look-out for cases of gastrostaxis, but have never yet encountered such a condition on the *post-mortem* table, in all cases having found one or more acute gastric ulcers.

The evidence that gastrostaxis exists is threefold, namely: (1) That at certain *post-mortem* examinations of patients who have died of haematemesis no lesion has been found in the stomach to account for the bleeding. (2) That at certain operations performed upon such patients an ulcer has not been found. (3) That gastrostaxis possesses a definite clinical history. I shall critically examine each of these statements in turn.

Haemorrhage from the mucous membrane of the stomach is due to one of two pathological conditions:

1. There is some lesion of the walls of the blood vessels, allowing of the escape of blood, which breaks through the surface of the mucous membrane into the cavity of the stomach. There are two distinct conditions of the mucous membrane produced:

(a) If the haemorrhage be profuse enough to kill the patient it is quite common to find at the autopsy multiple erosions of the mucous membrane. These erosions are quite superficial, the bases of the glands being preserved, and form slight depressions in the surface of the mucous membrane as if the latter had been lightly rubbed off with the finger. When the stomach is hardened in formalin they can hardly be seen by the naked eye unless there be any altered blood adherent to them, but they are easily recognized microscopically. These erosions are of no importance, except that they may help to keep up the bleeding, and they do not develop into ulcers because the stomachs of such patients are kept in the resting condition, the secretion of gastric juice being therefore at a minimum, and secondly, the gastric cells themselves are healthy, and there is no tendency towards their further digestion by the gastric juice.

(b) If the haemorrhage be not profuse the condition found after death, which is always due to the primary disease, is one of multiple small or large interstitial haemorrhages into the mucous membrane, with perhaps a little coffee-ground material in the cavity of the stomach. Such interstitial haemorrhages appear as black patches on the inner surface of the stomach, which on microscopic examination are seen to be portions of mucous membrane infiltrated with blood which has not escaped externally. These haemorrhages cause no symptoms, and the patient is fed in the usual way, with the result that the stomach is actively digesting, and the infiltrated patch of mucous membrane is digested owing to the destruction of the gastric glands, and removed, leaving an ulcer. These two pathological conditions occur quite independently, and constitute two distinct types; but there is no doubt that at the same time one may see all transitions and combinations in different cases.

2. The haemorrhage is a secondary event resulting from ulceration of the mucous membrane. The ulcer is the primary condition and the haemorrhage due to a secondary opening up of a blood vessel. There is a great deal of confusion with regard to the conception of what is an erosion and what is an ulcer, and the two names are sometimes applied to precisely the same condition.

Both the terms "ulcer" and "erosion" imply a loss of substance, and when the lesion is once formed, there can hardly be a clear distinction between them, the difference being only one of depth. From the point of view of morbid anatomy, therefore, an erosion may be regarded as a superficial ulcer.

From the point of view of general pathology, we are not yet in a position to formulate precise definitions of these

two conditions. I think it will be granted by all that serious haemorrhage will not occur as the result of an erosion, so that if a patient die from gastric haemorrhage any lesions which may be subsequently found in the mucous membrane, and which are microscopically proved to be erosions, must be a result of the haemorrhage, which is therefore the primary condition. The lesion must be examined microscopically, because, as I shall show later, fatal haemorrhage may occur from an ulcer which is so shallow as to be indistinguishable from an erosion by the naked eye. To whatever pathological condition the term "erosion" be restricted is immaterial, because it is quite obvious that if a small artery be opened up and death result from profuse haematemesis, the lesion extends to the submucous tissue or deeper, and should undoubtedly be called an acute ulcer. This is the class of case with which I am dealing in this paper. It is often stated by writers that a slight abrasion or erosion existed, in the centre of which a blood vessel had been opened. Such a lesion is undoubtedly an ulcer, and a great deal of misconception arises from the use of these ambiguous phrases.

When moderately stretched out at an autopsy the thickness of the stomach wall is about 4 mm., the mucous membrane being about 1 mm., so that an ulcer reaching to the muscular coat must necessarily look shallow and especially so in proportion as its area is large. This shallow appearance almost forces the observer to call the lesion an abrasion or erosion and to consider it as of little moment. I do not forget that much of the congestion of an acute ulcer probably disappears after death.

Acute ulcers vary in diameter from about the size of a split pea to an inch. Small acute ulcers and the scars of acute ulcers are very easily overlooked at *post-mortem* examinations, and an ulcer may be cut through in opening the stomach, and so obliterated. All writers agree upon this point, and I think there is no doubt that many stomachs have been described as being free from any evidence of ulcer owing to the employment of a not sufficiently minute method of examination. For such an investigation the stomach should be placed in warm water so as to relax the muscular coats, and first examined in front of a light for opacities in the mucous membrane. It should then be pinned out on a board after being moderately stretched, and the surface should be well washed with a camel-hair brush, and carefully examined all over by means of a lens. Even now a scar may be missed. The specimen should then be hardened for twenty-four hours in 5 per cent. formalin, and dried by washing in methylated spirit. Stellate scars can be much easier seen in the dry state, and if the specimen be photographed they are seen with greater ease still. The radiating lines are exaggerated by the photographic plate.

Hardening in formalin, of course, takes the colour out of the specimen; but I am presuming that the specimen has already been examined in the fresh state and all information possible gained from the presence of blood. The scars of acute ulcers are not always stellate in form. The only evidence of a scar may be a slight opacity of the mucous membrane which is adherent to the muscular coat at that spot.

If the ulcer has perforated the muscular coat and been at all extensive, the resulting scar will be a small surface very slightly depressed below the surrounding mucous membrane, but otherwise looking quite like it. The surrounding mucous membrane may be slightly puckered or not. The scar is always very much smaller than the ulcer from which it has resulted, and it often tends to be oblong, or like a small fissure, and may be quite invisible till the stomach is stretched. The area of such a scar may be less than a fifth of that of the ulcer preceding it, as I have been able to show experimentally.

A point of the greatest importance is that in the centre of a scar of this kind, almost invisible itself, may be found a tiny opening leading into an artery. One such case was only recognized by a piece of blood clot which protruded from the hole; this clot had been tunnelled by the blood, and formed a tiny projection from the scar. It is not sufficiently widely known that profuse haemorrhage may occur from an ulcer which is almost healed.

The following three cases illustrate the fact that death from profuse haemorrhage may occur at any stage in the evolution or cicatrization of an acute ulcer.

CASE I.—*Haemorrhage from an Acute Ulcer in Its Fully Developed Stage.*

The patient was a servant, aged 30 years, who was admitted to University College Hospital on February 19th, 1908. She had no previous history of any stomach disease or indigestion. On the morning of the day of her admission she suddenly vomited half a pint of blood, and had five such attacks during the day. She was blanched and had a small feeble pulse of 120 in frequency, but was well nourished and apparently otherwise healthy. During the next two days she had three attacks of haematemesis, melaena occurring. The vomiting stopped, but the patient died on the sixth day of her illness. At the *post-mortem* examination an acute ulcer was found on the posterior wall of the stomach, situated $1\frac{1}{2}$ in. to the right of the cardiac orifice and 1 in. below the lesser curvature. It was sharply punched out and extended to the muscular coat of the stomach. It was round in shape, $\frac{3}{4}$ in. in diameter, and looked quite shallow. In the centre of the base was an opening into a small artery.

CASE II.—*Haemorrhage from an Acute Ulcer Advanced in Healing.*

The patient was a man, aged 49 years, who felt a sudden pain in the abdomen whilst at work on September 3rd of last year. The pain was followed by faintness and the patient vomited up two quarts of blood, according to his own statement. He had epigastric pain after food, which he still continued to take, the pain coming on either at once or two or three hours after ingestion, and he had melaena. Five days later he vomited up a pint of blood, and on the sixth day was admitted to University College Hospital. It was found that seven years previously he had been in the hospital. The notes of the case showed that he then had pain three-quarters of an hour after food in the epigastrium, but no haematemesis. The vomiting often relieved the pain, but not always. This attack of pain and vomiting lasted seven weeks, and the patient recovered.

During the seven intervening years he never had any pain or vomiting until the present illness, but he had what he described as a little indigestion on and off. From the third day after admission he was treated by immediate feeding as recommended by Lenhartz. He did well for two days and then suddenly vomited up 24 oz. of blood and died six days later, on the eighteenth day of the disease, after gastro-enterostomy had been performed. At the *post-mortem* examination an acute ulcer was found on the small curvature of the stomach at a distance of $2\frac{1}{2}$ in. from the pylorus. It was irregularly circular in outline and 5 mm. in diameter. It was rapidly healing, the base was smooth and almost level with the surrounding mucous membrane, which showed puckering, and the edges of the ulcer were smooth and showed fine lines radiating on to the surface of the ulcer. In the centre was an open vessel which had bled. Near this ulcer was a stellate scar resulting from a healed acute ulcer of an earlier date. A tiny acute duodenal ulcer which was granulating was also present.

This case also illustrates, what is a well-known fact, that acute ulcers tend to recur in the same individual.

Such acute ulcers normally heal in a few weeks, exactly as they do in animal experiments. They may produce no symptoms; pain and vomiting without haematemesis may occur, or pain and vomiting with haematemesis. Sudden haematemesis or perforation without preceding symptoms constitute well-known clinical types of this disease.

CASE III.—*Haemorrhage from an Acute Ulcer almost Healed.*

The patient was a female, aged 69 years, who, after climbing up seventy stairs, was suddenly seized with nausea, and vomited up about an eggcupful of blood. On the following day (February 1st, 1910) she vomited a pint of blood, and was brought to University College Hospital. A fortnight before admission she had had a "bilious attack," and since then had pain in the stomach. Last August she had a similar attack without haematemesis. With the exception of these two attacks, separated by an interval of five months, she had no stomach disease at all. She vomited blood several times, and had melaena for the first five days, after which mouth feeding was commenced. She did well for four days. On the next day bleeding again commenced, and the patient died on the eighteenth day of the illness. At the autopsy four cicatrized acute ulcers were found on the small curvature of the stomach. Two of the cicatrices were stellate, and two were round, and in the centre of one of the latter was a tiny hole leading into a small vessel.

This case illustrates the occurrence of haemorrhage from an ulcer which is healed except for a small hole in the centre leading into the bleeding vessel. An interesting point about the case is that only two of the scars were found at the autopsy, the two remaining ones being found on the following day after drying the stomach as described above.

Before excluding acute gastric ulcer as the cause of haematemesis one ought, therefore, to exercise the greatest care, and be fully acquainted with all the conditions which are to be looked out for. Such cases as those I have quoted above are common enough, and all observers agree that they may be overlooked at the

autopsy with the greatest ease. The lesions are often called abrasions or erosions because they look so shallow, which is not surprising considering that the thickness of the mucous membrane of the stomach is only about 1 mm., and the base may be almost filled up, as these ulcers are often advanced in healing. The older physicians taught that bleeding might occur from the stomach in the absence of any ulcer, and many such cases were called "vicarious menstruation." Within recent times, however, Hale White¹ was only able to collect 10 cases from the literature, in which it was stated that no *post-mortem* lesion was found to account for the preceding haematemesis; one of these cases was under the care of Dr. Hale White himself, who was unable to find any evidence whatever of ulcer, erosion, or bleeding point at the autopsy. This is in striking contrast with the large number of cases of haemorrhage due to acute ulcer which have been recorded, and which are so well known as not to need reference.

Turning to another aspect of the question, a second piece of evidence quoted in favour of the occurrence of gastrostaxis is the fact that several cases of haematemesis have been recorded in which the patients have been operated upon, the interior of the stomach examined during life, and no ulcer found. These cases fall into three groups: (1) Those in which no lesion whatever was found. (2) Those in which a single bleeding point was found. (3) Those in which multiple bleeding points were found. The first group contains the greatest number of cases.

From what I have said above, it will be at once evident that it is quite impossible to explore the interior of the stomach during life thoroughly enough to exclude the presence of an acute gastric ulcer or a scar left by such an ulcer.

Cases are recorded in which the stomach was found normal at operation and yet an ulcer discovered at the subsequent autopsy (Dieulafoy,² Stevens³).

The inability to find the lesion at an operation is therefore no evidence that an acute ulcer is not present.

If a bleeding point be found in the mucous membrane of the stomach at an operation, it appears to me very questionable whether the exact condition of such a lesion can be accurately made out, and therefore whether the operator is justified in calling it a mere erosion.

The ease with which the point can be examined at an operation depends upon its position, but still, considering the difficulty that is experienced in deciding upon the nature of the lesion by naked-eye appearances at an autopsy, it seems to me that such difficulty must be greater at an operation, whatever the position of the lesion, and that the depth could certainly not be judged to a fraction of a millimetre. Such an operation must be done hurriedly, owing to the condition of the patient, and the bleeding must tend to obscure the character of the lesion.

My experience of operations on the human subject is, however, only that of a spectator, so that I cannot do better than quote the words of a surgeon⁴ who has had considerable experience in such operations. After stating his opinion that the bleeding from an acute gastric ulcer is nearly always arterial in origin when it is severe, he goes on to say:

During life the ordinary acute ulcer of the stomach usually takes the appearance of a linear or triradiate fissure, out of which the blood trickles in a steady, continuous stream. . . . *Post-mortem*, when all the congestion and oedema have gone, the arteries, especially in a patient who has died from loss of blood, contract to such an extent that they appear to be quite insignificant in point of size, and a lateral opening, such as is usually present, is almost certain to be overlooked. Unless the blood vessels of the stomach are injected, or some special examination is made, the bleeding point is scarcely ever found in an acute ulcer, and the loss of blood is almost certain to be laid to the credit of the capillaries.

When the bleeding is slight, it comes from the capillaries or veins in the base of the ulcer. If such an ulcer be bleeding, it must be easier to find it in the living person than at an autopsy, but it must be more difficult to examine accurately. If any doubt exist with regard to the nature of a lesion after death, the question can be settled by microscopic examination. The statement, therefore, that a bleeding point is found at an operation certainly appears to me to be no more in favour of the case being one of gastrostaxis than of such a lesion being an acute ulcer. Neither is the finding of multiple bleeding

points any evidence against acute ulcer, because acute ulcers are often multiple, and one would expect that in such cases an artery might be opened in one, and that in some the bleeding would be chiefly capillary and in others venous. A case is recorded in which twenty acute ulcers were found at an autopsy.⁵

Another piece of evidence given in favour of gastrostaxis being a clinical entity is that it possesses a definite clinical history. Thus, there are said to be three symptoms—pain, vomiting, and bleeding. These symptoms occur in attacks separated by intervals of months or years of freedom from any symptoms, or at most symptoms of indigestion may occur. These attacks may occur over many years. The first sign of the disease may be profuse haematemesis. The blood is bright red, and varies greatly in amount. In each attack blood is vomited on occasions separated by a few hours or days. The disease occurs in patients chiefly between the ages of 20 and 40 years, and affects females more than males. Vomiting and pain may occur without haematemesis.

I have not the slightest doubt in my own mind that a disease of this type exists, and also that it is commonly not chronic gastric ulcer, but in every case of this sort which I have seen, and which has died, I have invariably found an acute gastric ulcer which has perforated a blood vessel. From the writings of other observers it is evident that they recognize these recurrent attacks as being due to acute ulcers, but I do not think that sufficient stress is laid upon the recurrences in descriptions of acute gastric ulcer in the textbooks.

In three cases within the last six months I have found the scars of old attacks, together with recent ulcers, which have caused death. There is no doubt that acute ulcers in the human subject heal in a few weeks, precisely as they do in animals, and that they are liable to recur in the same person. Each attack represents a new ulcer or ulcers, it lasts a few weeks at most, and the ulcers heal as in animals. I have been able to show that acute ulcers in animals may be delayed in their healing owing to motor insufficiency of the stomach leading to delayed emptying of that organ. In the same way an acute ulcer in man may be delayed in healing, as the following case shows:

A man, aged 34 years, was admitted to University College Hospital on August 28th of last year. The present illness had commenced about five weeks before admission with pain in the right side below the ribs. The pain was of an aching character and was said not to be related to food. He had a little vomiting, and on one occasion brought up a small quantity of blood, melaena occurring subsequently. He kept at his occupation, and one day whilst at work fainted, and shortly afterwards vomited a dark red clot of blood about the size of the fist. He had never had any indigestion nor gastric pain before the present illness.

Three days later he had two fainting attacks with melaena, and vomiting of blood on one occasion. He died five days subsequently, after gastro-enterostomy had been performed and after an illness of about seven weeks.

At the *post-mortem* examination the stomach was found to be dilated. An acute ulcer was present on the lesser curvature midway between the cardiac and pyloric orifices. It was oval in shape, measuring 8 mm. in its longest and 3 mm. in its smallest diameter. The edges were rounded, quite clean, and slightly incurved. The base was clean, and in the centre was a small eroded vessel which had caused the bleeding. A certain amount of thickening of the base had taken place, and puckering of the mucous membrane at the edges was distinctly visible. The ulcer was healing, but more slowly than normal and with more thickening. In short, the healing was delayed.

The ulcer, judging from the history, was probably at least seven weeks old. When an acute ulcer is delayed in the healing the epithelium grows over the surface more slowly than normal, and the glands are not so well regenerated. There is more dense fibrous tissue in the base than normally occurs, and more scarring results. It has been known for some time that acute ulcers following burns may only give rise to symptoms some weeks after the burns have healed, and unhealed ulcers have been found as long as two and a half months or more after this time. Such ulcers are undoubtedly acute ulcers produced at the time of the burn which have been delayed in their healing, or in which the healing has been completely stopped. It is quite likely that they have been slowly extending, because perforation or sudden haemorrhage may occur as the first symptom. The importance of acute gastric ulcer, therefore, does not merely lie in the fact that it produces a definite group of symptoms during its

formation and healing, but also that it may be delayed in its healing, or extend, and so form the initial stage of chronic gastric ulcer.

The natural conclusions to which one is driven from a consideration of the above points are that gastrostaxis as an independent disease is not yet indisputably proved to exist; and, on the other hand, that acute gastric ulcer is a most important and very common malady, and one which is worthy of more consideration than appears to be given to it.

REFERENCES.

¹ Hale White, *Lancet*, 1906, vol. ii, p. 1189. ² Dieulafoy, *Bull. de l'Acad. de Mèd.*, 1898, 33-40, p. 49. ³ Stevens, *Glasgow Med. Journ.*, January, 1899, vol. li, p. 5. ⁴ Mansell Moullin, *Trans. Clin. Soc.*, vol. xxxvi, p. 80. ⁵ Affleck, *Lancet*, 1901, vol. i, p. 478.

ON IMPIANTATION OF THE URETERS INTO THE RECTUM BY THE SACRAL ROUTE,

ILLUSTRATED BY A CASE OF INVETERATE VESICO-VAGINAL FISTULA IN WHICH THE LEFT URETER WAS IMPLANTED AND THE RIGHT KIDNEY REMOVED.

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SOME years ago the question of implanting the ureters into the rectum by the sacral route was suggested to me by the desire to alleviate the discomforts of a man from whom I had removed the penis with the whole urethra up to the bladder for a carcinoma of the bulbous and membranous portions which had extended to the apex of the prostate. At that time I did not work out the idea to a satisfactory conclusion, as the patient learnt to manage himself, and preferred his condition, with its attendant difficulties, to submitting to an operation with all its uncertainties. But I learnt sufficient to be sure that, in the male at any rate, the operation would be very difficult owing to the depth at which the ureters had to be sought and the contracted area in which the manipulations had to be performed.

When, however, the case which is described in detail below was sent to me by Dr. McAllister, Vice-Master of the Coombe Lying-in Hospital, the suitability of such an operation was so evident that I returned to the attempt to arrange a feasible procedure, with the impression that the greater width of the female pelvis would make it more easy of accomplishment.

Three points suggested themselves for consideration:

The first was the preservation of the sphincteric control of the rectum, for, if the result of the operation was to produce paralysis or even paresis of the sphincteric apparatus, the patient would not be benefited, and might even be made worse.¹

Then there was the question of the uterus. Could the operation be carried out without its removal, or must it be sacrificed?

And, lastly, there was need for clearer knowledge about the ureters. Could they be easily found in this situation? After their separation would it be possible to close the openings made in the bladder?

1. The Preservation of the Sphincteric Control of the Rectum.

The direction usually given in Kraske's operation for removal of the rectum that the section through the sacrum must not extend as high as the fourth sacral foramina, so that the fourth sacral nerves may not be injured, defined the limit of the removal of bone.

The fourth sacral nerves incline upwards after their exit from the foramina, and groove slightly the anterior surface of the sacrum on which they lie. In a horizontal section, if the foramina are left intact, there is no danger of these nerves being injured. The nerves to the levatores ani run from the lower part of the sacral plexus just below the white line, and close to the ischial spine; they are completely out of the way if the knife be kept close to the bone in separating the soft parts from the coccyx and lowest part of the sacrum. The inferior haemorrhoidal nerves which supply the external sphincter, and lie almost entirely within the ischio-rectal fossae, are not likely to be disturbed, and the pudic nerves, of which they are branches, are quite safe.

The internal sphincter is probably innervated by the sympathetic, which reaches the bowel along the superior, middle, and inferior haemorrhoidal arteries. With reasonable care no serious damage to the nerve supply of this muscle was to be expected. Consequently there was no reason to suppose that the patient's power of control over the sphincters would be impaired.

2. The Question of the Removal of the Uterus.

The uterus and upper part of the vagina lay in the direct route to the trigone, and to expose the insertions of the ureters it was almost certain that the former would have to be excised. If this were done the operation would be partly intraperitoneal.

Its removal need cause no feeling of compunction, for the patient had suffered too much in consequence of her last pregnancy to desire another, and if left it was only likely to be a discomfort and an inconvenience.

My first attempt led me to think that the ureters could be dissected out and removed from the bladder without any further interference with the uterus than the division of the uterine arteries, and without opening the peritoneum. I was able to isolate both ureters and by traction to pull up a cone of bladder on either side of the vagina. The closure of the separate openings made in the bladder was commenced before each ureter was completely cut adrift, so that there was no fear of the bladder wound sinking out of sight.

But subsequent attempts showed me that I had met at first with an exceptionally easy case, and that the removal of the uterus in nearly every instance would be essential. In doing this, after the uterus had been freed on either side from the broad ligaments, it was drawn out of the wound and pulled downwards and backwards, whilst the anterior surface of the vagina was stripped off the base of the bladder. The tendency was not to strip the vagina sufficiently low, as, owing to the necessity of preserving the levator ani insertions into the bowel, this was not easy. But a suitable retractor fixed at the inferior angle of the wound gave great help. When the vagina had been separated low enough it was divided transversely, but a sufficient margin was left to permit of its easy closure by sutures.

3. Exposure and Separation of the Ureters.

This was not such a simple matter as I had expected. When the uterus was removed and the vagina stripped down, the ureters were not once exposed, nor were they readily found. At this stage there was to be seen an opening into the peritoneal cavity corresponding to the lowest portion of the pouch of Douglas, through which the uterus had been drawn out, and a quantity of fatty pelvic connective tissue. The position of a ureter was best ascertained by the sense of touch. When tissue which concealed it was grasped between the finger and thumb, it could usually be felt as a somewhat flattened but definite cord. By placing a finger in the peritoneum and grasping the tissues on either side of the opening between the finger and thumb the ureters could usually be detected lying close to the peritoneum, but at some depth in the fat. When located they could easily be exposed with dissecting scissors and separated down to their entry into the bladder wall. At first the tendency was to search for them too far to the sides of the wound, but they were usually found running longitudinally $\frac{1}{2}$ in. to $\frac{3}{4}$ in. from the median line, and tending slightly outwards and upwards to lie on the outer sides of Douglas's pouch. One point that the preliminary observations taught me was to be prepared for ureters of variable size; in one instance the ureters were so small and narrow that even after they had been traced to the bladder some uncertainty still remained until it was found that a probe passed along them from the vesical apertures, which were very difficult to enter. Another was that the separation of the ureters from the bladder required considerable circumspection. Twice at least when I thought I was cutting well wide of the ureter I found that I had divided it in its oblique course through the vesical wall, and had failed to secure its orifice on the mucous membrane. This accident was probably due to putting the ureter on the stretch to steady and define the bladder. A cone of bladder was in this way formed, and the exact situation of the ureter in it no doubt misconceived.