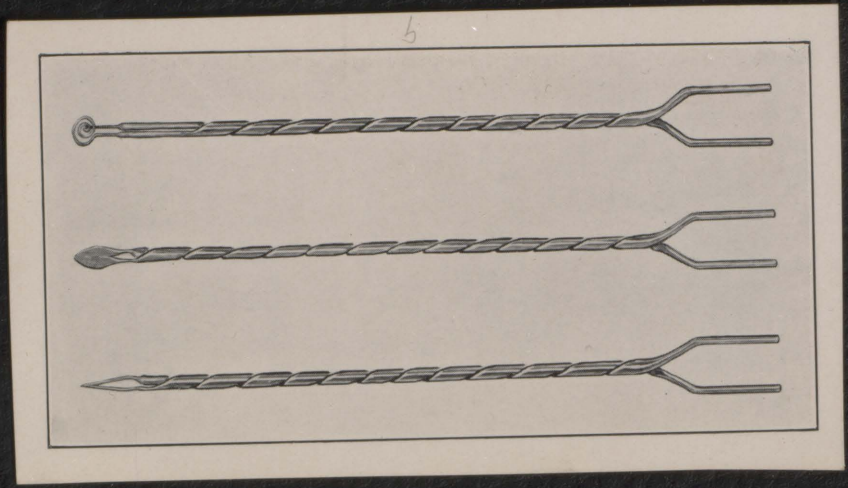

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Prolapse of the Rectum

By E. A. DANIELS, M.Sc., M.D.
Montreal, Canada

Prolapse of the rectum may be defined as a loosening and unnatural freeing either of the mucosa or muscularis (one or both) of the rectum. In this condition the supporting structures of the rectum i.e., the peritoneum, lateral ligaments, blood vessel stalks, levatores ani muscles, fascia and connective tissue are all abnormally relaxed, and thus permit the extrusion of this portion of the terminal bowel through the anal-orifice.

The classification of prolapse of this organ is based upon the degree of descent of the bowel wall. There are two varieties:

(a) Prolapse of a superfluous redundant mucous membrane

(b) Prolapse of the muscle coats of the rectum.

In this second type the mucous membrane may or may not be superfluous and relaxed. From his experience, the author disbelieves that (b) is merely an advanced stage of a mucous-membrane prolapse. As will be presently pointed out, the factors operative in producing either type of prolapse are somewhat different[1] and a complete prolapse may occur at once without being preceded by a prolapse of the mucous membrane. The mucous-membrane variety of prolapse is easily recognized. The extrusion is a short one and palpation between the thumb and index finger reveals only a thin band of tissue composed of two layers of mucous membrane. The protrusion is smooth without furrows and continuous with the peri-anal skin. The extruded mucosa is easily sucked back into the bowel lumen. Complete prolapse of the rectum presents itself as a large, thick mass and has an apex at its lowest extremity. There are a series of circular folds and there is a moderately deep sulcus between the skin of the anal margin and the wall of the prolapse. Anteriorly, the recto-vaginal or recto-vesical pouch of peritoneum is usually dragged down with the prolapse. This may contain loops of small bowel. Reduction is not easily effected as compared with the mucous membrane variety. (Figs. 1 and 2.)

FIXATION OF THE RECTUM IN THE PELVIS

Although the rectum is quite firmly fixed in the pelvis, it nevertheless enjoys a moderate degree of mobility and is capable of marked variation in diameter.[2] The rectum usually begins at the level of the

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third sacral vertebra where it is continuous with the pelvic colon, and ends where it pierces the pelvic diaphragm at a point about 1½ inches anterior to the tip of coccyx. At this point there is marked narrowing of the bowel which turns abruptly backwards at a right angle (opposite prostate in the male) to become the anal canal. The rectum lies in the concavity formed by the sacrum and the coccyx and then rests for about 1½ inches on the pelvic floor formed by the union of the levatores ani muscles and their fasciae. The anatomic fixation of the rectum in the pelvis is accomplished by the peritoneum which holds the bowel to the walls of the true pelvis, particularly in the lateral planes where the peritoneum becomes quite thickened, to form the lateral ligaments. Posteriorly, between the rectum and the sacrum (where peritoneum is lacking), there is a dense area of connective tissue (really the deep fascia) which higher up carries the superior haemorrhoidal vessels in the form of a heavy rectal stalk. In front, the recto-vesical or recto-vaginal folds of peritoneum are present as supporting structures. Below these and anteriorly, the rectal wall is adherent by dense connective tissue to the prostate and seminal vesicles in the male and the posterior vaginal wall in the female. Below, firm support is offered the rectum by the levatores ani muscles and fasciae, the coccyx and recto-coccygeal ligament. The fat which normally surrounds the rectum and fills the ischio-rectal fossae reinforces in some measure, the other supports.

Although the rectum lies in the hollow of the upper and middle part of the sacrum, its long axis forms a virtual right or acute angle to the lower sacrum, coccyx and sacro-coccygeal ligament and to the sling support offered by the pelvic floor. It is the author's opinion that the coccyx, coccygeal ligaments and fascia are of great importance in preventing descent of the rectum. Two cases of prolapse of the rectum were recently observed following upon removal of the coccyx with division of its ligamentous supports. (Figs. 3, 4 and 5.)

ETIOLOGY OF RECTAL PROLAPSE

The factors operative in producing a mucous-membrane prolapse of the rectum are, in the author's opinion, quite different from those which come into play in the development of complete rectal prolapse. The mucous membrane of the lower rectum is normally very loosely attached by fibrous and elastic tissue to the submucosa.[1] This loose attachment in certain normal individuals may easily become exaggerated. This redundancy is especially evident in the anterior and lateral walls of the lower rectum. The loose attachment of the mucosa to the submucosa may easily be demonstrated experimentally by in-

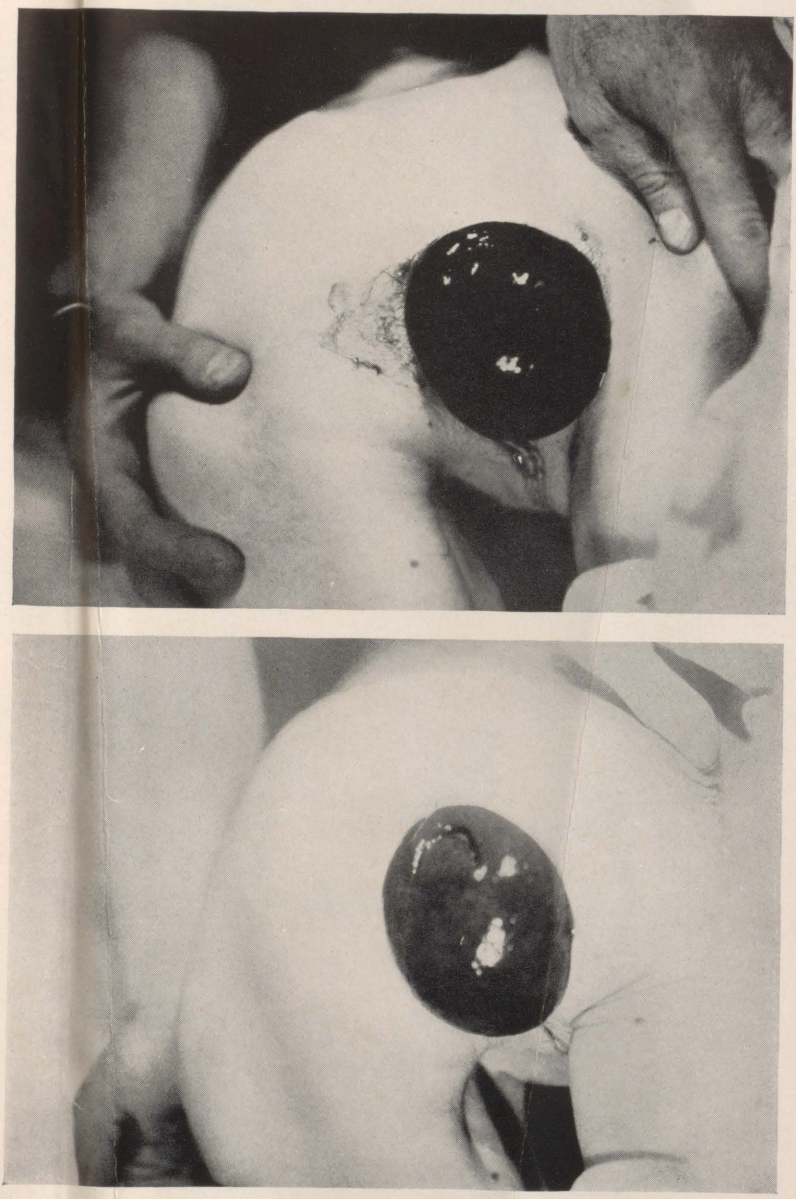


FIG. 1. Photograph of patient suffering from irreducible strangulated complete prolapse of the rectum. Woman, 58 years old. The bowel was not gangrenous. Reduction was effected under low spinal anaesthesia. The case was subsequently treated by submucous and para-rectal injections (described in the text) without any operative procedures on the pelvic floor or ano-rectal outlet. There has been no prolapse for three years.

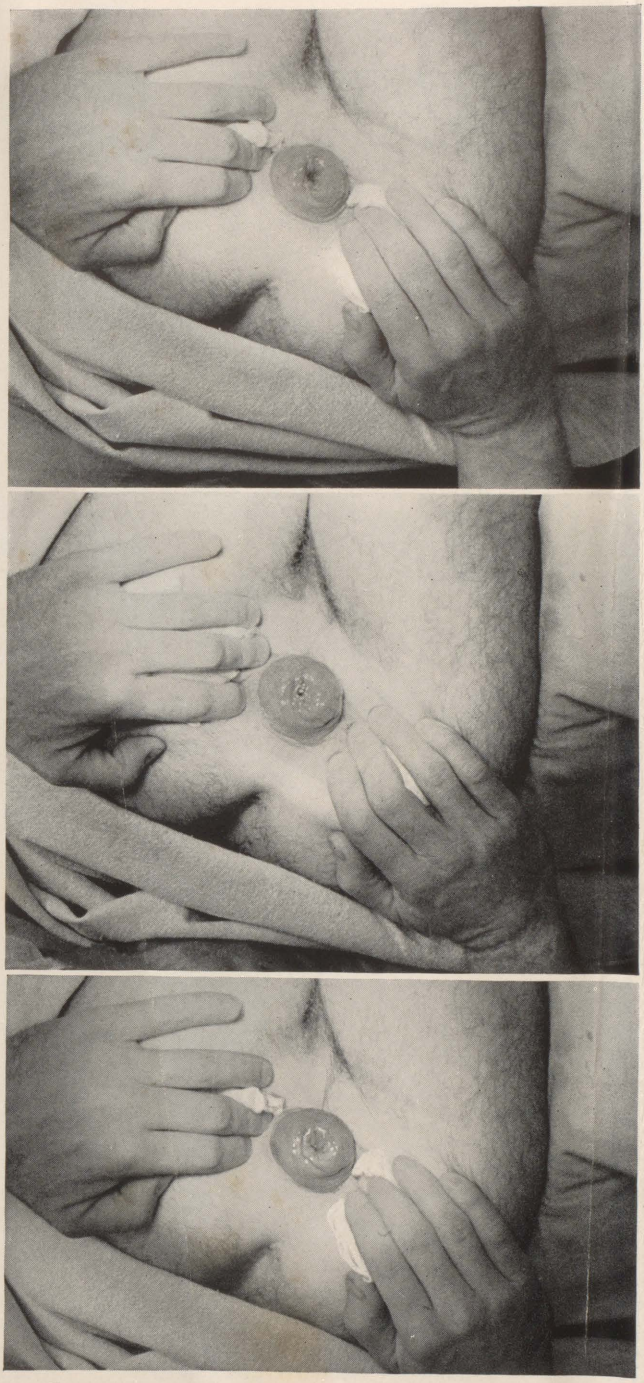
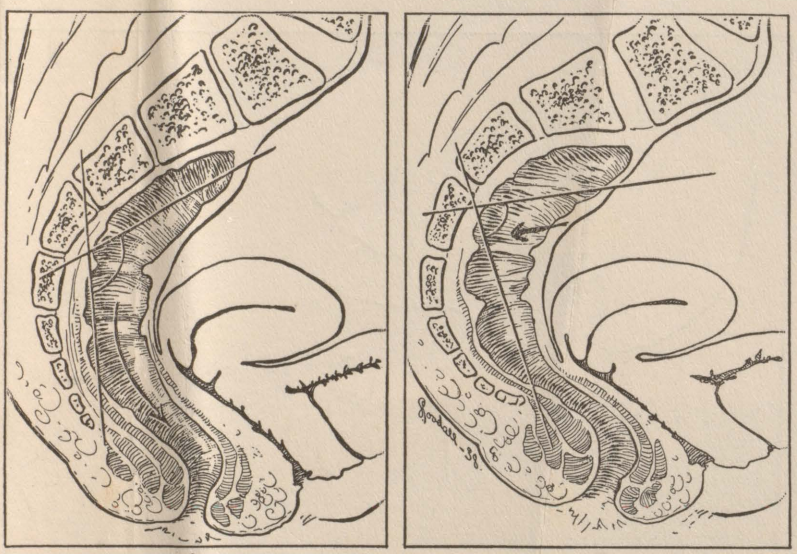


FIG. 2. Complete prolapse of the rectum. Treated by submucous and para-rectal injections followed by plication of the ano-rectal musculature described in the text.

jecting either water or normal saline submucosally. It will be observed with what ease the redundant mucous membrane may be floated off its submucosal bed. A loosely attached, redundant mucous membrane in the next stage becomes quite superflous and in concertina-like fashion rises up into the ampulla recti. It may now be easily seen that if the anal orifice permits, this redundant superfluous mucous membrane may be extruded and the condition known as mucous-membrane prolapse of the rectum becomes established. The extrusion



A B

FIG. 5. A. This is a diagrammatic representation of the flat type of sacrum found in many of the author's cases of rectal prolapse. The long axis of the rectum forms an obtuse angle with the long axis of the lower sacrum and coccyx. The bowel does not fit into the "sacral bowl" found normally. The arrow indicates the direction of gravity directed through the anal orifice, naturally predisposing to prolapse.

B. Showing the normal type of sacrum. An acute angle is formed, and gravity directs the long axis of the rectum almost in a right angle against the lower sacrum and coccyx thus preventing prolapse through the anal orifice.

of a redundant mucous membrane through the anal orifice depends more upon the anatomic condition of the anal tube than upon the degree of the redundancy. In normal individuals the anal canal (i.e., the distance from the skin of the integument to the ano-rectal line, a distance normally of approximately 2 inches posteriorly and 1 inch anteriorly) is long, narrow, slit-like and tubular. The anal canal moreover, at the ano-rectal junction (opposite the prostate in the male) as already pointed out, bends abruptly backward and its long axis

forms an obtuse angle with the long axis of the rectum. This can be verified in doing a digital examination in such individuals, when, in traversing the anal canal, the tip of the finger points towards the umbilicus and in females is especially evident as it follows the posterior vaginal wall. A sharp angle or ridge is thus encountered at the ano-rectal junction and in entering the rectum the finger turns abruptly backwards towards the spinal column. Such a tubular anal canal, with its long axis in a different plane from that of the rectum, acts as a

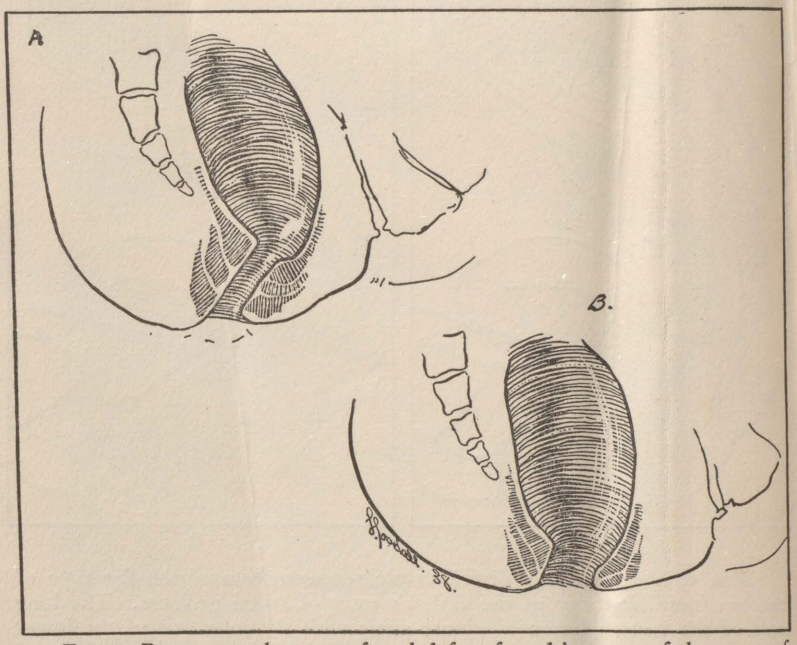


FIG. 6. Represents the type of anal defect found in many of the cases of rectal prolapse.

A—Represents the long tubular type of anal canal (Normal).

B—Represents the abnormal type predisposing to prolapse.

formidable barrier to the extrusion of any of the components of the bowel wall, such as a redundant superfluous mucosa. When this occurs, the whole of the lower rectum and ampulla recti on proctoscopic examination will be observed to be crowded and filled with this redundant, concertina-like mucosa. However, upon questioning the patient no history of prolapse or extrusion is recorded. The patient on the other hand will be conscious of a feeling of fullness as if a foreign body were present in the rectum. (The author would use the name of "concealed mucous-membrane prolapse" for this variety.)

A

B

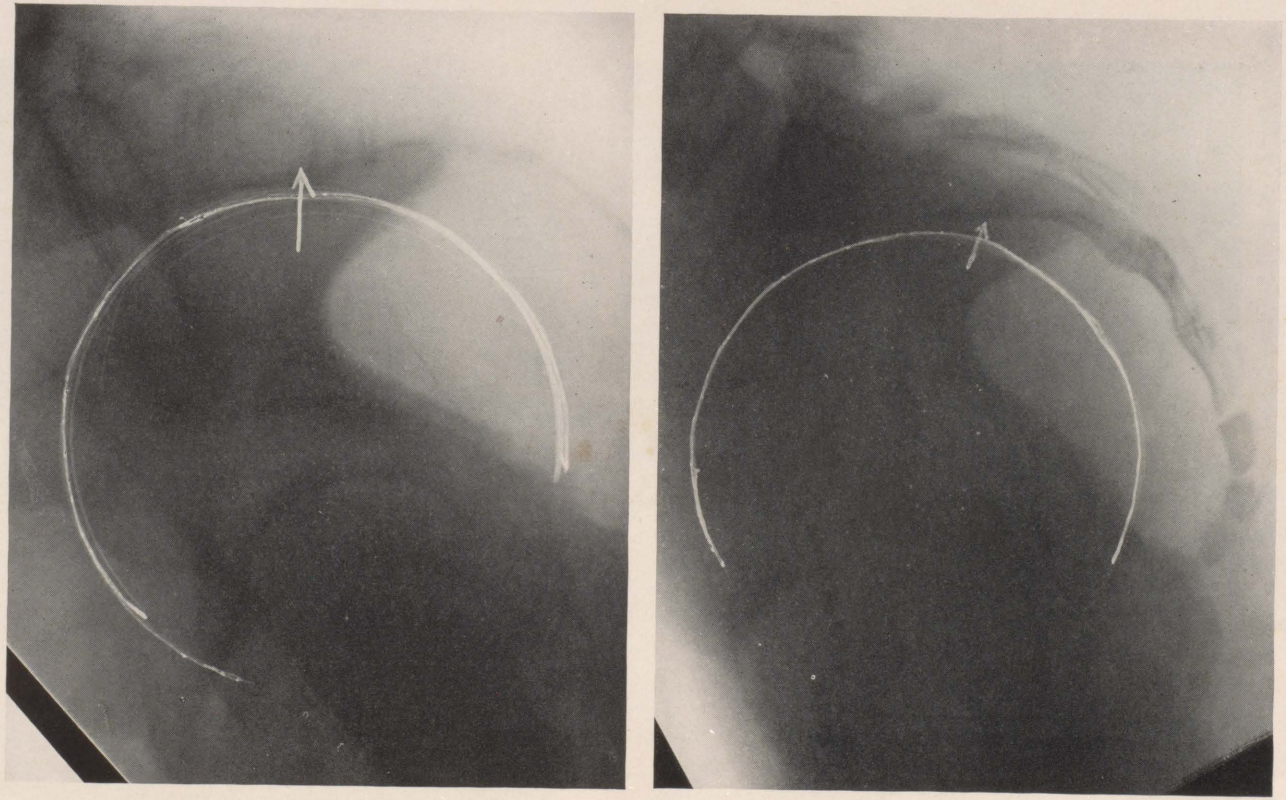


FIG. 3. A, B, and C. X-ray studies of the sacrum. The inner surface of this bone is quite flat as compared with the normal. This is more obvious upon digital examination through the rectum. The arrows indicate the flat area. The straight coccyx and anal deformity may or may not accompany this type of sacrum. The latter defects may be present singly as described.

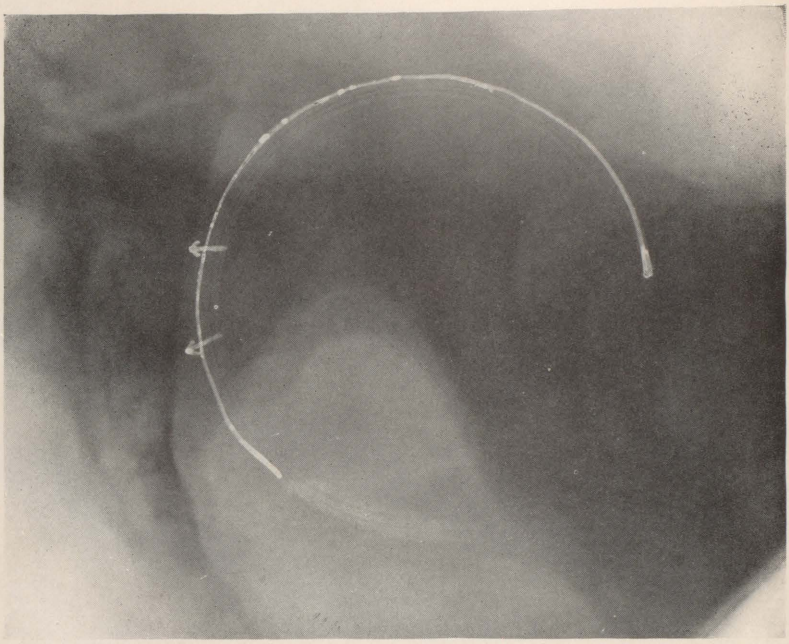


FIG. 3C. See legend on previous page.

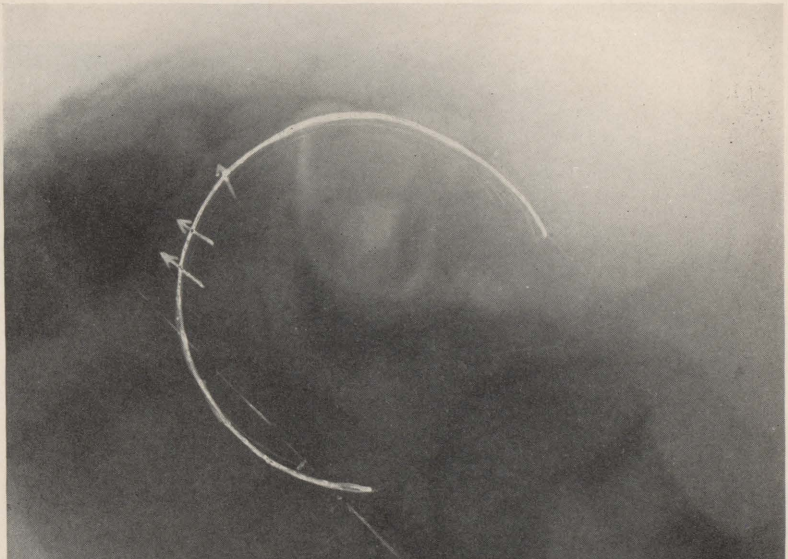


FIG. 4. Normal Sacrum. This photograph taken from a pelvis showing a well-hollowed sacrum, especially apparent on digital examination and stereoscopic study.

Backache will frequently be complained of in this "internal" non-extruding type of mucous-membrane prolapse. Constipation is a common occurrence since this redundancy fills the lower rectum and precedes the stool. Such individuals frequently develop a low-grade proctitis with anal fissures and pruritus. There may be a mucoid or muco-purulent discharge.

In the other type of individual, where the anal canal is not tubular and angulated but short, stout and patulous a mucosal redundancy will

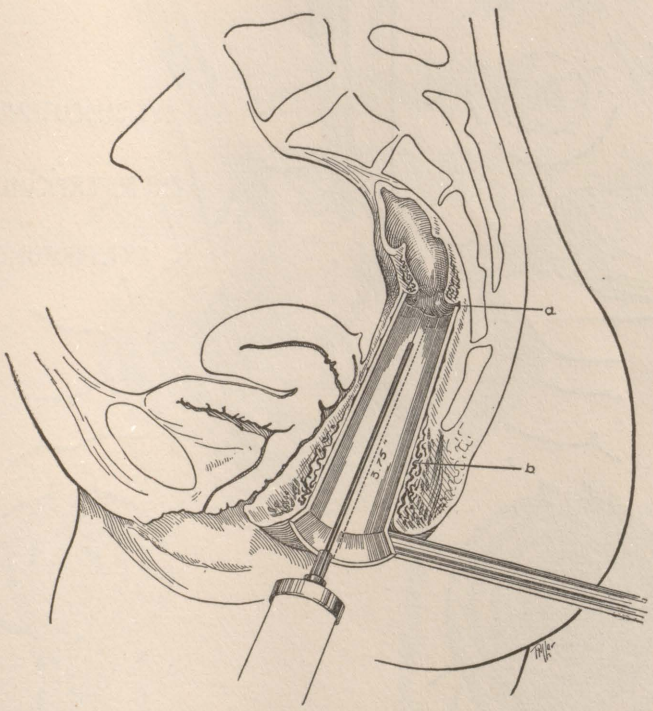


FIG. 8. Submucous injection of redundant superfluous mucous membrane with sclerosing solution.

be easily extruded. In the former type, digital examination is not easily carried out and the ano-rectal musculature grips the finger as a long, tubular, constricting structure. In the latter type, digital examination is easily carried out, and the anal canal is recognized as a loose orifice, easily traversed by the examining finger and not possessing the ano-rectal angulation already described.

Prolapse of the rectum in children may present itself as a mucous-membrane type or as a complete prolapse including the muscle coats.

The plane of the infant pelvis is rather vertical and the inner surface of the sacrum quite flat.[3] The coccyx possesses very little of a forward tilt and may lie almost in a straight line with the sacrum. The anal orifice, too, has a more posterior position than that seen in adults, and a line drawn down from the tip of the coccyx will be found to be

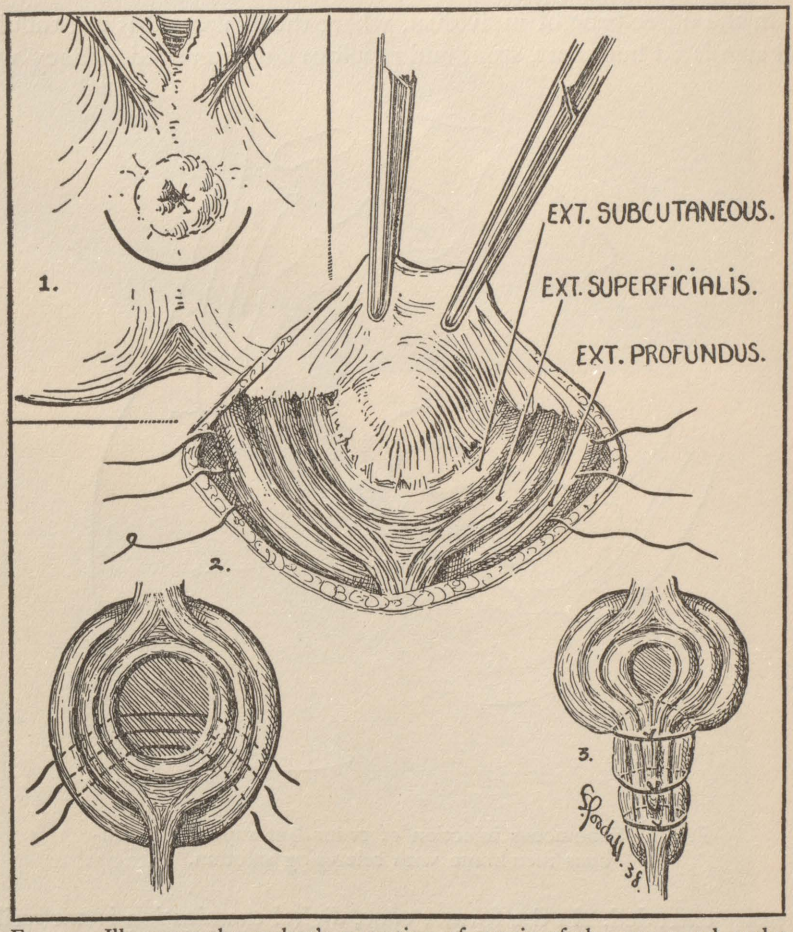


FIG. 13. Illustrates the author's operation of repair of the ano-rectal outlet described in the text.

almost in a straight line with the anal orifice. It will thus be seen that although children often present a mucous-membrane variety of rectal prolapse, conditions are favorable for the development of a complete type of descent. It is thus apparent that in infants undue loss of weight, diarrhoea and straining at stool are very often sufficient to

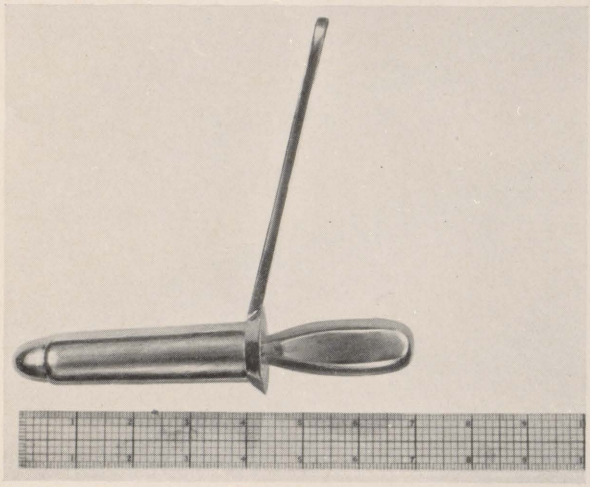


FIG. 7. Author's adult pattern of proctoscope.*

* Brass & Bronze Manufacturing Co., Limited, 269 Craig Street West, Montreal.

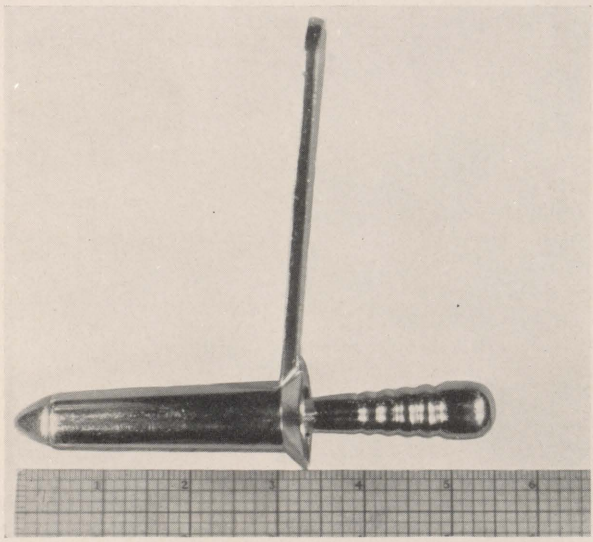


FIG. 9. Author's pattern of infant proctoscope* used in injecting the submucosal space.

* Brass & Bronze Manufacturing Co., Limited, 269 Craig Street West, Montreal.

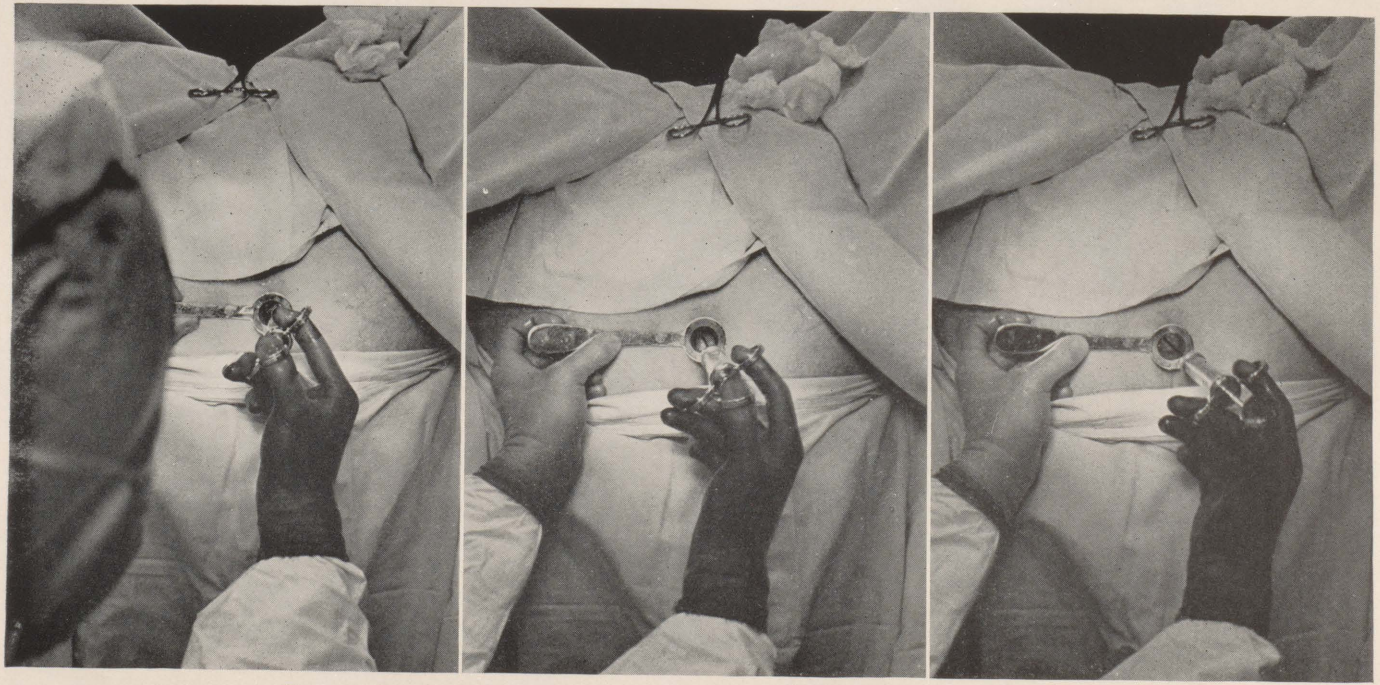


FIG. 10. Demonstrates the technic of submucous injection described in the text.

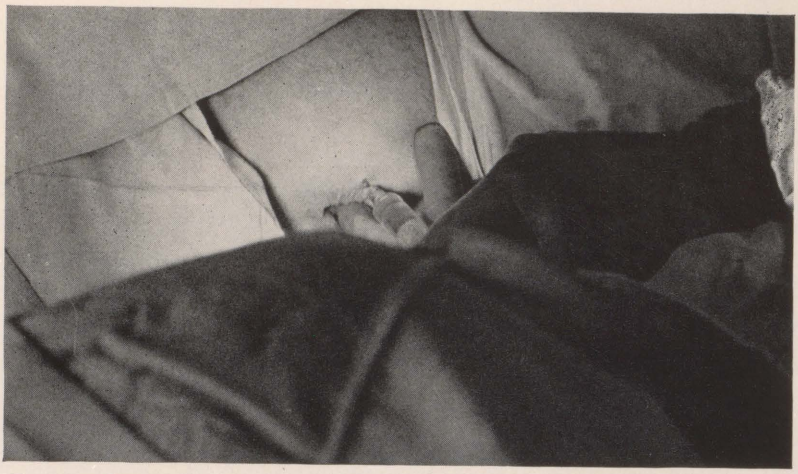


FIG. 11. Represents a pararectal injection with the finger in the anal canal guiding the direction of the needle.

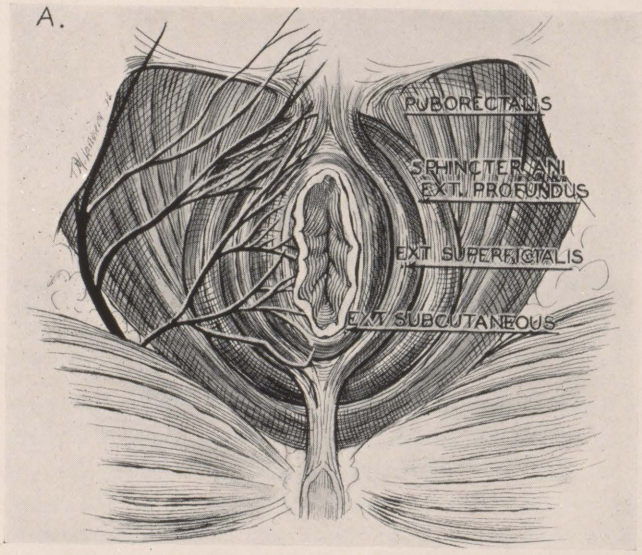
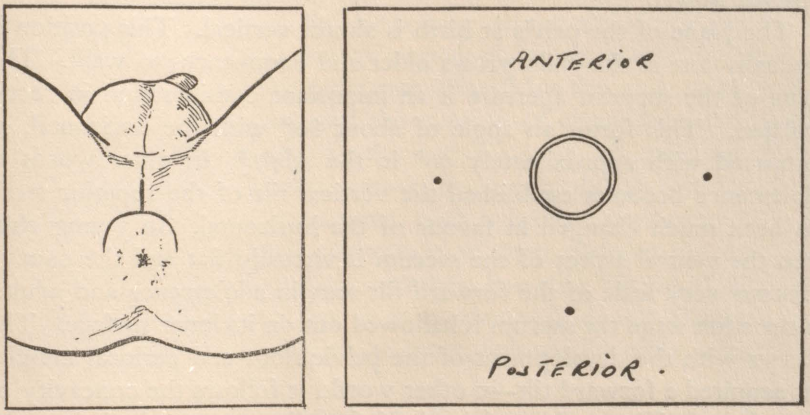


FIG. 12. Represents the musculature of the ano-rectal region viewed from below. The three portions of the sphincter ani externus muscle are shown in their respective planes. The inferior haemorrhoidal and perineal nerves are shown in position on one side. (Reproduced through the courtesy of the Amer. Jour. Digest. Dis. and Nutr., 3:775, 1936. Daniels, E. A.: Anal Fissure, Anal Spasm, and Anal Stenosis.)

initiate either an incomplete or a complete prolapse of the bowel, since the stage is set and conditions are favorable for such a disturbance.

Complete prolapse of the rectum is, in the author's opinion, not frequently preceded by the mucous-membrane variety, but more often than not commences at once as an extrusion of both mucous membrane and muscle coats of the bowel wall. A large redundant mucosal prolapse may eventually drag down the muscle coats of the rectum during the process of descent. However, in the author's cases of complete rectal prolapse, it appears that very few of these were preceded by a mucous-membrane type of extrusion. In this type of prolapse,



A B

FIG. 14. A—Represents the incision in a male patient as the initial step in performing the plastic procedure upon the levatores ani muscles described in the text.

B—Represents the three areas of needle punctures in performing the para-rectal injection described.

the whole of the rectum descends through the anal orifice. Reduction may be easily effected or, on the other hand, its return may become impossible. Obstruction of the blood supply through the prolapsed bowel occurs, edema ensues and in severe cases the lesion may present itself as a strangulation in the form of a large, bluish, edematous, deeply engorged mass with areas of necrosis on the surface of the lesion. The factors operative in initiating a complete descent of the whole rectum are mechanical in nature. One should first discuss what actually occurs to the rectum in complete prolapse and then try to tabulate certain of the conditions which would predispose to such a state. Complete prolapse may be defined as a loss of the normal degree of fixation of the rectum in the pelvis. The supporting structures of this organ become lax, stretched and inefficient and with conditions

favorable at the outlet, permit its descent. The peritoneum supporting this portion of the terminal bowel especially its lateral ligaments, the retro-rectal connective tissue, the meso-rectum carrying the haemorrhoidal vessels, the dense connective tissue between the rectum and the prostate and seminal vesicles in the male, and the posterior vaginal wall in the female, are all stretched and subluxated. The rectum now lies more or less unsupported in the true pelvis. Its excursion in almost any direction is now possible. Gravity now favours its downward descent and one has only the resistance offered by the pelvic floor and the anal tube to prevent the complete extrusion of this portion of the terminal bowel.

The plane of the pelvis at birth is almost vertical. This position is gradually lost as the child grows older and commences to walk. The plane of the superior aperture is an important consideration in rectal prolapse. This forms an angle of about 80° with the horizontal, as compared with approximately 60° in the adult.[3] In other words as adolescence becomes established the vertical tilt of the superior strait has been much changed in favour of the horizontal. In young children the ventral aspect of the sacrum is virtually flat and the coccyx possesses very little of the forward tilt seen in adolescence and adults. In the adult stage the sacrum is hallowed out on its inner surface. The coccyx with the development of the pelvic floor and perineal muscles has acquired a forward tilt—in other words, it follows the concavity of the sacrum. The anal canal in the infant lies completely below the level of the tip of the coccyx, and the anal orifice occupies a more posterior position. In other words a vertical line drawn downward from the tip of the coccyx will be found to pass through the anal orifice, whereas in the adult such a line generally will be distinctly behind it. The rectum in the infant is a straight tube possessing very little of the lateral and antero-posterior curvature seen in the adult bowel. It has a straight descent and does not, as in grown-ups, lie in and fill out the concavity formed by the inner surfaces of the sacrum and coccyx. In the adult, the long axis of the rectum therefore lies at a virtual right or an acute angle to the lower portion of the sacrum and to the coccyx and at the same time its posterior wall follows the concavity formed by the inner surface of the upper and middle thirds of the sacrum. The rectum, therefore, in trying to descend, would impinge almost at right angles against the lower portion of the sacrum and coccyx and recto-coccygeal ligament. In the infant a straight line drawn from the superior strait of the pelvis would virtually drop out of the anal orifice, and it will thus be seen that the long axis of the rectum in young children is almost parallel to the long axis of the

sacrum and coccyx. The anal canal in children is quite straight and continues directly downwards from the rectum, whereas in normal adults, as already pointed out, a rather sharp angulation backwards occurs at the ano-rectum (opposite the prostate in the male) and the examining finger, in the adult will recognize the anal canal as running in a forward direction. In grown-ups, this anatomic configuration of the anus in the presence of a well developed tubular canal acts as a resisting force in preventing prolapse.

Age of Onset. The author believes that complete prolapse of the rectum becomes initiated in early childhood and adolescence due to a retarded development of these adult characteristics of the pelvis and rectum. Stretching of the supporting structures of the rectum thus takes place over a long enough period of time to enable the prolapsing bowel to overcome the resistance offered by the anal outlet (usually a short vulnerable one) and the process of complete rectal prolapse has become established. In the author's series of cases of complete prolapse of the rectum examination has nearly always revealed a rather flat inner surface of the sacrum with a coccyx possessing very little forward tilt. In a high percentage of these patients the anal orifice was observed to be almost in a straight line with the tip of the coccyx and not definitely anterior to it as observed in normal adults. The anal canal too, was found to be short, vulnerable, easily traversed and quite lax.

The pelvic floor formed by the levatores ani muscles and their fasciae and especially that formed in front by the puborectales portion of this muscle is an important consideration. The pelvic floor acts as a sling in supporting the rectum especially anteriorly. In parturition rupture of this support will favour descent of the rectum, particularly so if the abnormalities described should be present.

The long, tubular type of anal canal already described in which the sphincteric muscles are well developed and quite powerful acts as a definite barrier to the extrusion and descent of the rectum. As already pointed out, certain individuals possess very little of this type of anal configuration, but have a rather short patulous, "sawed-off" anal orifice. This latter type of anal canal with or without the abnormal type of pelvis already studied definitely facilitates the descent of the rectum.

From this description it will be seen that certain anatomic and mechanical factors when present will favour and predispose to complete prolapse of the rectum. These conditions, above outlined, initiate a downward excursion of the terminal bowel, stretching of its supporting structures and its eventual extrusion through the anal orifice. It will thus be seen that a good pelvic floor in the presence of a long,

tubular, more or less rigid, anal canal will offset an infantile type of rectum and pelvis in preventing prolapse. It is for this reason that complete prolapse of the rectum will often spontaneously follow parturition, coccyxgectomy or fistulotomy where sphincter tissue has been sacrificed. Large protruding haemorrhoids or a mucous-membrane prolapse producing an eventual relaxation of the anal sphincters in the presence of the above predisposing mechanical factors may initiate a complete prolapse of the rectum. Complete prolapse thus following spontaneously upon any of these factors, however, must pre-suppose the presence of either the abnormal type of pelvis or anal tube already described. In the author's experience loss of weight and loss of the fatty tissues surrounding the rectum will not in itself favour prolapse of the bowel unless the pelvis and outlet are such as to favour extrusion of the rectum.

Examination of a case of complete rectal prolapse during the act of extrusion of the mass presents a characteristic appearance. This may be likened to the appearance of the uterus during the last stages of labor in which the lower uterine segment has become obliterated. The lowest cone-like segment of the uterus becomes part of the upper uterine segment and undistinguishable from it. In the same fashion the slit-like anal canal has become obliterated as shown in the diagram. One sees merely a bulging spheroidal structure impinging against the perianal tissues with only the thinned-out and stretched subcutaneous portion of the external sphincter visible under the skin, as the last barrier. A complete corona of dilated external haemorrhoidal veins may be seen between the skin and the fibres of this subcutaneous muscle.

TREATMENT

The treatment of prolapse of the rectum first implies that a careful study and diagnosis of the patient has been arrived at. It is essential that the operator determine whether the lesion is a mucous-membrane one or whether one is dealing with a complete prolapse of the rectum. An effort must be made to see the condition in the prolapsed state. One should then encounter no difficulty in determining whether the prolapse consists only of mucous membrane or includes the muscle coats as well. The bony structure of the pelvis must be carefully studied and the amount of relaxation of the anal orifice determined. If the patient is a woman who has borne children the presence of a ruptured perineum must be sought for. One should examine a case for any evidence of fistulotomy or other rectal operative wounds.

In studying my cases of rectal prolapse, I was struck by the peculiar type of anal canal already described which a high percentage of these

patients presented. There was this short "sawed-off" type of anal orifice in a vertical practically straight line with the coccyx (anus normally far to the front) easily traversed by the examining finger which seemed to "fall" at once into a roomy ampulla. This has already been compared to the obliterated lower uterine segment seen in the last stages of labor. In many of these patients the inner aspect of the sacrum on digital examination presented itself as more or less of a flat surface by comparison with the normal. X-ray studies in these patients, particularly the stereoscopic views, bore out this finding. The coccyx in my cases of rectal prolapse had very little forward tilt as found normally. In other words, it pointed directly downwards in a straight line with the long axis of the sacrum instead of having a forward curve in a hammock fashion. In two of my patients suffering from complete prolapse of the rectum, coccygectomy had been performed six months previous to the onset of symptoms. Of the etioligic factors above outlined found more or less consistently in my cases of rectal prolapse, it was my considered opinion that the mechanical anal defect already described was the more constant and the more important of the predisposing factors outlined.

One cannot reconstruct the pelvis nor remove the predisposing factors operative in producing rectal prolapse, but with the knowledge at hand concerning this condition one can carry out certain conservative but effective measures based upon the etiology outlined in order to correct or alleviate the condition. These measures have been very gratifying in the author's hands in a series of 22 cases of complete rectal prolapse and in well over 50 cases of mucous membrane prolapse (incomplete) treated during the past four years. The principal underlying treatment consists of fixing in the pelvis the descending portion of the bowel which may be only mucous membrane or include all the muscle coats. One attempts a "restitutio ad integras." In prolapse of the rectum, the supporting structures of the bowel which keep the uppermost part of the rectum from about the level of the third sacral vertebra fixed in the hollow of the sacrum have all become subluxated, stretched and inefficient. These supporting structures have been described.

The author's plan of treatment consists as follows:

A. Obliteration of the redundant superfluous mucous membrane by a sclerosing solution.
B. Fixation of the rectum in the true pelvis by surrounding this organ with an area of dense inflammatory scar tissue, employing a sclerosing solution.

C. Repair and reinforcement of the ano-rectal sphincteric musculature.
D. Perineorrhaphy operation in either males or females to supplement A, B, or C.

The author has thus far never had to resort to the radical operation known as recto-sigmoidectomy which, on the basis of this work, would seem to be the only radical operation suitable for the permanent cure of this condition, should the above conservative measures fail. In his series of patients the conservative measures to be described have been sufficient to either completely correct the disturbance in the majority of cases or to give sufficient relief to make the patient's life agreeable.

A. In dealing with either the incomplete or complete type of rectal prolapse, the writer carries out a thorough plan of injecting the redundant mucosa with a 5 per cent solution of phenol in sweet-almond oil in the submucosal plane. In many cases of complete rectal prolapse there may be very little or no mucosal redundancy in which variety the whole rectum descends at once without being preceded by a mucous-membrane prolapse. In this type as well, the author carries out his plan of producing some degree of submucous indurative reaction as the first step in dealing with the lesion. A long tubular proctoscope (Fig. 7, author's pattern)* is employed in carrying out the submucous injection. From 10 to 30 cc. of 5 per cent phenol in sweet-almond oil is injected submucosally employing a St. Mark's Hospital type of Luer Lock syringe. The whole of the redundant mucous membrane is thus floated off its submucous bed. (Figs. 8, 10.) This technique is described in detail under reference No. 1. At the completion of this procedure the whole of the ampulla recti is packed with vaseline gauze through the proctoscope and the instrument slowly withdrawn. This packing is left in from 24 to 48 hours and assists in effecting a submucous indurative reaction and at the same time assists in fixing the redundant mucous membrane to the submucosa. This procedure may be all that is required in the mucous-membrane type of prolapse of the rectum but on the other hand will merely be the first step in dealing with the complete variety. In infants suffering from mucous-membrane prolapse of the rectum the author has employed this technique,[3] employing his small infant proctoscope* (Fig. 9) and using from 5 to 15 cc. of 5 per cent phenol in sweet-almond oil. One may have to repeat this treatment after a period of weeks or months. No anaesthesia is required for the submucous injection.

B. In complete prolapse of the rectum one carries out the next step[4] in dealing with the lesion. This consists of a para-rectal injection of

* Brass and Bronze Manufacturing Co., 269 Craig St. West, Montreal.

quinine sulphate in dilute sulphuric acid. (Fig. 11.) (Quinine sulphate 12 gr. dilute sulphuric acid 30 minims, water 30 minims, the whole sterilized by boiling.) This measure is carried out under low spinal anaesthesia, within a week following the submucous procedure described under A. A long, 4-inch, 18- to 20-gauge needle is employed and three points are selected—two lateral and one posterior, each about an inch from the subcutaneous portion of the external sphincter. Between 9 and 12 cc. of this solution is employed in carrying out the para-rectal injection. The solution is injected laterally on each side of the rectum, both above and below the levatores ani. Posteriorly the solution is injected into the retro-rectal fascia between the rectum and the sacrum. Between 3 and 4 cc. is injected in each plane. On rare occasions, in fat robust individuals, the author has employed as much as 5 cc. in each plane. A firm binder with a thick perineal support is placed over the perineum and the patient returned to his room. Within several days when the finger is inserted into the rectum a fairly marked para-rectal reaction will be noted. This para-rectal technique first employed by Swinford Edwards and more recently by Mr. Gabriel[4] of St. Mark's Hospital, London, has proved very valuable as a step in the author's plan of dealing with complete prolapse of the rectum. One cannot employ a very large quantity of this solution because of its toxicity but no untoward effects have thus far been observed.

C. This next step is carried out either at once following the para-rectal injection but is better deferred for several weeks to a month to see whether steps A and B have not proved sufficient to correct the lesion. This operative procedure is indicated when there is marked relaxation of the ano-rectal outlet as the result of stretching by the protruding bowel. This operative procedure is based upon the anatomic studies of Milligan and Morgan on the anatomy of the ano-rectal musculature.[5] This procedure is carried out upon the sphincter ani externus muscle by first dissecting out its three component parts, as pointed out by the above authors. (Fig. 12.) The external sphincter, as shown in the diagram, extends up posteriorly around the ano rectum for a full distance of two inches. It would be impossible to describe the detailed anatomy of the external sphincter and the pubo-rectalis portion of the levator ani in this paper. The reader is referred to the work of Milligan and Morgan[5] and to the work on the anal canal[6] published by the author of this paper. A circular incision between the coccyx and the anus is made and one dissects down upon this area exposing the component parts of the external sphincter muscle as follows: First the subcutaneous portion which runs circularly around the anal

canal (beneath this one can see the longitudinal muscle of the bowel), one next comes upon the superficial portion of the external sphincter which runs backwards to find its insertion in the coccyx. Deep to this one finds the fibres of the sphincter ani externus profundus which fuses with the pubo-rectalis portion of the levator ani and the longitudinal muscle of the bowel to form the ano-rectal ring. In the author's plan of treatment the subcutaneous portion of the sphincter is folded over employing through and through sutures of No. 1 twenty-day chromicized catgut and next carried deeply through the superficial sphincter, into and picking up the profundus portion of the muscle in the same fashion. Two or three of these sutures are employed in this fashion, thus plicating this musculature. With the finger in the anal canal an actual tightening up of the orifice will be observed and effected. The skin is then closed with fine linen and no drainage employed. (Fig. 13.)

D. The perineorrhaphy operation in either male or female to supplement either A, B or C is carried out as a last stage when the above measures are not effective, either alone or collectively. In the female the ordinary type of perineorrhaphy, pulling up the levatores ani and tightening these, thus building up a firmer perineal body, is carried out. Such a procedure can and has been done even in nulliparae.

In the male the dissection is carried out between the bulb of the urethra and the rectal wall through a T shaped incision over the perineum as shown in the diagram. (Fig. 14.) The levatores ani are exposed and tightened by through and through sutures or over plication employed No. 1 chromic catgut. It is imperative when carrying out this perineorrhaphy that the suture be carried through the outer longitudinal muscle of the bowel wall, thus pulling up and holding the rectum as well as re-enforcing the levatores ani muscles. After the last two procedures the patient is kept on a very low residue diet and the bowels confined for a full week. Walking is not permitted for three weeks.

The author in his series of cases has never had to carry out the operation of recto-sigmoidectomy, since practically all of his cases were either greatly improved or cured by the above measures described. Recto-sigmoidectomy, which is a very radical procedure, has a definite place in dealing with the intractable type of prolapse. Fortunately such a case has not come to hand and the author feels that the conservative measures outlined in this paper will deal with the average case of prolapse of the rectum.

SUMMARY

A study of rectal prolapse has been carried out by the author in a series of cases taken from hospital and private practice. The etiology of this lesion has been investigated and in the author's experience certain factors have been consistently present. These have been described. A conservative plan of treatment has been outlined.

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VISCERAL ALLERGY

JAMES R. GOODALL, M.D.
and
R. M. H. POWER, M.D.
Montreal, Que.
From the Wards and Research Laboratory of St. Mary's Hospital

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VISCERAL ALLERGY

JAMES R. GOODALL, M.D., AND R. M. H. POWER, M.D., MONTREAL, QUE.
(From the Wards and Research Laboratory of St. Mary's Hospital)

ALLERGY is a new science with unlimited possibilities. It may be of interest to abstract the first paragraph of a previous article to convey a broader concept of this difficult subject. The local and general manifestations of allergy are due to a local or general hypersensitivity to certain provocative substances, allergens, which either gain entry into the circulation by absorption through the alimentary tract, or by bacterial invasion, or by hypodermic injections; or they may act locally by contact, inhalation or any other mode of approach. The presence of an allergen either in the circulation, or by contact, in sensitive individuals, produces in the sensitive tissues certain protective antibodies, or antigens, which are liberated by these cells when the allergen invades their domain, and the resultant is a form of irritation expressed either as an extravasation or a spasm. The extravasation is due to definite changes in the capillary lining epithelium, and the spasm, it is thought, is due to irritation of the neuromuscular mechanism. These antibodies are called allergins. They are a protective device. The results in the sentient patient are protean in their symptomatology. The cells or organ affected determine, to a great extent, the nature of the symptoms. In the nose, the common manifestation is hay fever, or hemorrhages due to plasma, or cellular extravasations, respectively; asthma, due to involuntary muscular spasm and extravasations; urticaria, due to focal skin dystrophies; local edemas, due to subcutaneous outpourings; eczema and prurigo, due to disturbance of nerve nutrition; and disturbances of the genital cycles, due to spasm or derangement of the normal secretions. These are the commonly recognized manifestations. But there is no organ or system of the body which may not be the seat of allergic symptoms. It was shown, in a previous work, that the sex organs are commonly affected; that the migraine is an expression of brain allergy, and cases of spreading paralysis and coma were quoted, of undoubted allergic origin.

It is proposed to detail the cases in which unusual and startling trains of symptoms arise out of involvement of specific organs in allergic states, producing conditions closely simulating the well-recognized organic diseases. These will be dealt with in the following order: Intestinal, hepatic, peritoneal, cardiac, muscular, and cerebral.

Intestinal Allergy.—In a paper entitled "Mucous Colitis"[1] Goodall named allergy as one of the frequent causes of colitis. Further observation has confirmed this opinion beyond refutation. The condition may be a more or less permanent, chronic state, or it may be periodic in its

manifestations, just as allergic states, as described and accentuated in a previous paper on allergy of the pelvis.[2] It may be added that the symptoms may be fixed in one part of the bowel, or may shift, affecting now one section of the colon, now another, and it may be further stated that the condition may be spastic or hypersecretive. The dominant symptom in the majority of cases is not acute pain, but a burning sensation. At times the pain is quite severe, occasionally crampy. There is generally tenderness over the ascending or descending colon. Backache is an invariable accompaniment. Constipation is the rule. The stools are small in the spastic type, large and covered with inspissated or glary mucus in the hypersecretive type. In this respect, mucous colitis of allergic origin does not differ from colitis from other causative agents. The pain or tenderness may be fixed over the cecal region, thereby resembling appendicitis or pyelitis; or a pendulous cecum may closely resemble right tuboovarian trouble. If the pain is permanently fixed at the hepatic flexure, cholecystitis may be suspected. When the transverse colon is affected, the symptoms may cause one to suspect gastric disease; and involvement of the splenic flexure may give the patient severe precordial pain, with a fixed idea that she is suffering from cardiac disease. Sigmoid involvement closely resembles the ovarian diseases, or may cause one to suspect diverticulitis, or pyelitis. Not infrequently an extension of the process to involve the rectum, causes tenesmus and exacerbation of any hemorrhoidal symptoms. The fact that the symptoms most frequently are not fixed, but pass from one colic area to another, almost at once leads one to suspect the correct cause. Palpation of the colon reveals general or local tenderness over the colonic area. The colon may be distended, or more frequently, present the hardness of a hawser. Constipation is the rule, with occasional bouts of bowel hyperactivity. There is always improvement after such a development. Upon awakening, there is a general abdominal soreness, and upon rising, backache and occasional articular rigidity.

A painstaking history, both personal and familial as to allergic susceptibilities, will greatly help in determining the proper cause. Alternating periods of recovery and recrudescence characterize the majority of cases. Patients are generally much more uncomfortable at the before or during the menstrual epoch.

Referred gastric symptoms are not uncommon. These are generally of the nature of nausea and eructations several hours after meals, or tremendous bloating.

Direct gastric symptoms are very varied, and may simulate organic gastric diseases so closely as to defy clinical differentiation. It is only by a process of exclusion, by tests of gastric function, which is not disturbed in allergic diseases, and by a suggestive personal and familial history of allergic manifestations, that the true nature of the dysfunction may be opined. In infancy, one of the commonest manifestations is pylorospasm, and regurgitation. In childhood, incipient hunger, followed after a small quantity of food, by an anorexia; in adult life,

symptoms simulating gastric ulcer without hematemesis or the acute local tenderness, torpitude and flushing of the face after meals, palpitation and profound muscular and mental lassitude.

Hepatic Allergy.—Probably the most interesting type of spastic allergy is that which affects the bile passages, simulating hepatic colic and probably pancreatic regurgitation. These cases are moderately common. They have come under observation in the last year. The symptoms are those of hepatic colic. The pain is right-sided, referred to the breast, shoulder and back; occasionally (in one case) the pain had the distribution also of involvement of the pancreas, in that the pain radiated also distinctly to the left of the mid-epigastric line. The history suggests gallstones so closely that all the patients were operated upon for this diagnosis, without finding stones at operation or in skiagraphs. The history of one of these cases will do more to clarify this syndrome than any other enumeration of findings. The patient, a physician's wife, mother of three children, had five distinct and severe attacks characteristic of hepatic colic. X-rays were negative. Tipp test negative. But owing to their characteristic onsets and the severity of the symptoms, non-opaque gallstones became the final diagnosis. Operation revealed a perfectly healthy gall bladder and bile passages. The gall bladder was removed. Patient made an uneventful recovery. Three months later she had a severe recurrence, and six months later another. She then came under the writers' care for a menstrual disturbance of allergic origin. She gave a familial history replete with allergic manifestations. Her husband, the doctor, is also allergic, all her children are allergic. She herself had a minus 18 basal rate, and was devoid of gastric free hydrochloric acid. She had most distressing attacks of migraine. After being under observation, but before the diagnosis was made, she had two severe colic attacks. The second lasted almost three hours. In the attack, three-fourths of a grain of morphine only aggravated the symptoms. Morphine, it is interesting to note, raises the intrabiliary pressure. She now learned that these attacks invariably followed the use of compound tablets of aspirin, phenacetin, caffeine and codeine, which she took for her migraine and facial neuralgia. Since this determination, she has been wholly free from biliary attacks. Since being placed upon XXX minims of hydrochloric acid and 4 gr. of thyroid (Burroughs Wellcome) t.i.d.p.c., she is no longer appreciably allergic, being free from migraine and eschewing aspirin. It may be interpolated that aspirin is a very common allergen in susceptible patients.

Two other cases gave a history somewhat analogous. A fourth came into the surgical wards of St. Mary's Hospital and under the care of the surgical resident, Dr. MacCormick, who has had an extensive training in allergy at the Children's Memorial Hospital. X-ray and other tests were negative. Amyl nitrate and later adrenalin gave immediate and complete relief. Later it was found that the allergen was a food product. Its deprivation prevented a recurrence. In all these cases the condition seems to point to a spasm of the sphincter

of Odie, and an elevation of intrabiliary pressure. Corroborative proof of this was found in the exacerbation of the condition by the use of morphine and its immediate relief by the antispasmodics, and by making the patient nonallergic, either by correcting the existing endocrine dysfunction, or by finding the causative allergen which disposes to allergic susceptibility. It was clearly pointed out in the previous paper, quoted above, that endocrine dysfunction often becomes the exciting factor in predisposed individuals, and the correction of the endocrine dyscrasia removes the susceptibility to allergens. It was also pointed out that the allergic state is familial or acquired, and is, in these cases, the constant subsoil which comes to the surface when the general health is lowered by disease or deranged function. Nothing in the human metabolism is left to hazard, each function being under the control of another function and it, in turn, under another. The final arbiters are the endocrines. Dysfunctions of these are the

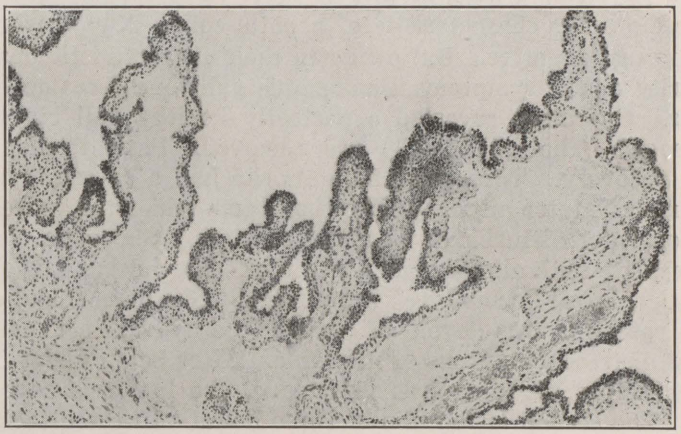

Fig. 1.—Coagulated and liquid edema of the fimbriae. The empty spaces and dilated subfrimbrial lymph channels were originally filled with liquid lymph. Coagulation has occurred only at the periphery.

necessary precursors of metabolic upsets. Under these dysfunctions of malnutrition of cell and body generally, familial, or acquired, allergic susceptibility may become a dominant factor in diseased states.

In the patient who had severe pain referred also to the left side, the inference was drawn that the intrabiliary pressure caused a back pressure along the duct of Wirsung into the head of the pancreas, thereby causing acute edematous pancreatitis, a condition so clearly described by Dr. Edward Archibald in "Acute Edema of the Pancreas" (Ann. Surg. November, 1929).

Allergy of the Peritoneum.—There are a few of these cases that are of minor degree. In a previous paper allergy of the pelvic cavity was fully described, especially when combined with pelvic inflammatory disease. However, allergy of the peritoneum may be general, without other signs of local disease, or the allergic extravasation may be local-

ized to any special part. Especially prone to this local manifestation are the Fallopian tubes at the fimbriated ends. (Fig. 1.) In the cases where the peritoneum, visceral and parietal, is involved, the edema is variable in quantity, in one case presenting a truly water-logged condition (Figs. 2, 3, and 4). In another case described by a colleague, the peritoneum was like a soaked sponge. Under these circumstances, the peritoneum, being indifferently securely attached to its underlying

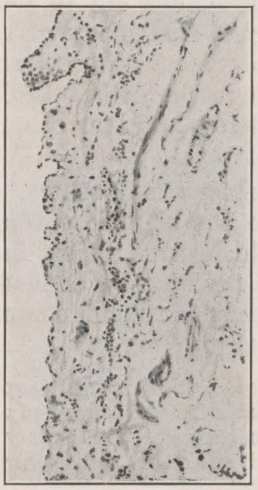

Fig. 2.—Normal peritoneum.

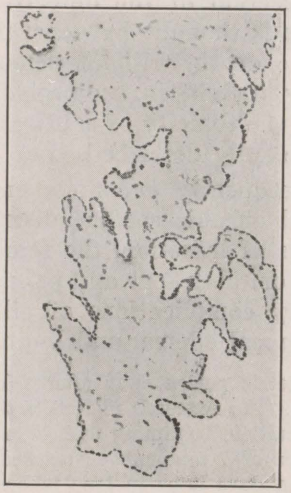

Fig. 3.—Slight peritoneal edema.

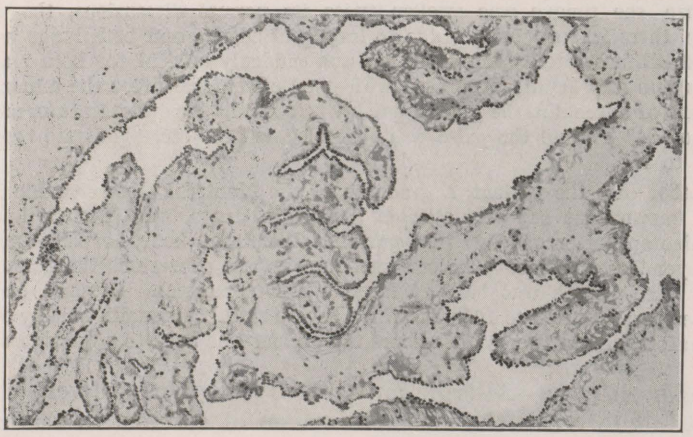

Fig. 4.—Allergic edema of the peritoneum, showing coagulum and liquid spaces.

structures, is thrown into numerous edematous convolutions which are covered by a single layer of flattened cells. Drawings of these demonstrate this condition very clearly. In other instances, the sections show the extravasated lymph in the widely dilated lymph spaces of the Fallopian fimbriae. In one case, a child of 7 years, very allergic, the peritoneum was as though it had been infiltrated everywhere by a syringe.

Cardiac Allergy.—This condition can be diagnosed only by the history of allergy, symptoms, and recovery under appropriate treatment. The cardiac symptoms may vary from precordial pain to acute pseudo-anginal attacks with a residual of pain that may last for twenty-four hours or more. Several of these cases have come under observation in women of allergic traditions, in whom appropriate treatment has immediately relieved the spasm, and removal of the offending agent, or a correction of the defective metabolism, has led to permanent relief. It is thought by general pathologists that many of the cases of cardiac death, without demonstrated coronary disease, and more especially in children, are probably allergic in character. In Goodall's paper on the toxicity of colitis, cardiac distress and fear of impending death are fully described.

How frequently one finds among physicians and others, signs of acute and chronic cardiac distress due to the allergic cardiac effect of a focus of infection, the removal of which gives almost immediate release from both fear and pain. Mention need hardly be made of the pulmonary complications of allergy: asthma, bronchitis, pulmonary and laryngeal edema, and lingual and oral swelling.

An obstetric patient was admitted to the private ward, in what proved to be false labor. For economic reasons, a therapeutic induction was attempted with castor oil and pituitrin, beginning with 2 minims and increasing 1 minim every half hour until a dose of 7 minims was reached. It proved ineffectual. She returned to the hospital ten days later in true labor. When about fully dilated, with membranes ruptured, labor pains ceased, and the resident later gave her 3 minims of pituitrin. Within one-fourth of an hour the patient was cyanotic, her lips were twice their natural size, the tongue was swollen twice its normal dimensions, the face was edematous, there was air hunger due to laryngeal edema, and both lungs were filled with moist, diffuse râles. The condition was suddenly critical for both mother and child. Oxygen and helium were used with good effect, and after the administration of 5 minims of adrenalin, the patient quickly recovered. Her temperature, meanwhile, shot up to 104° F., and the pulse was over 140, and the blood pressure rose rapidly from 110 to 150.

In another case the patient, a graduate of the Montreal Homoeopathic Hospital, who had known that she was allergic to certain drugs, was given a hypodermic of morphine on her second day post partum. Within an hour, her face was swollen almost beyond recognition. The conjunctivae were so edematous that they hung in wrinkles. The tongue was half as large again as normal. Dyspnea supervened, and her temperature rose to 105° F., with a severe chill. Adrenalin quickly controlled the discomfort, and in twenty-four hours the patient was back to a quasinormal state.

Muscular Allergy.—Allergic muscular dystrophies are very common. One has but to recall the muscular spasm of asthma, to realize what a profound and persistent effect this may produce. But one finds muscular spasm of an allergic nature under many and varied pathologic states. The writers wish to draw attention to the muscular spasms of pregnancy, which are an expression of a toxicity-producing allergic spasm. The percentage of women who suffer from these painful spasms of the legs, thighs, and feet, is very high. The spasm is acute for a time, frequently leaving a soreness which may last some hours, even days. These are so common as to be regarded as something to be expected and endured. They are an expression of toxicity, and

many spasms of a similar nature in other parts of the body, especially in the abdominal wall, intestines and uterus, are frequently complained of without ever a thought being given as to their true character. These are extremely common, and of unusual interest. They tend to recur in the same spot, when that area has once shown its susceptibility to its pregnancy change of environment. The muscles of the loins are particularly prone to this type of spasm, but any musculature may be involved. In two cases, the clinical interest is great, owing to the doubt which attended the cases until the proper diagnosis was established.

The first was a multipara, 38 years of age. Ten days before an anticipated delivery, she was seized with an excruciating pain in the right loin. Upon examination, there was no marked tenderness, slight rigidity, but the subjective symptom was intense. Morphine, ¼ gr., did not relieve at all, nor did a repeated dose effect any appreciable change. The pain gradually subsided, leaving a very marked soreness. Patient described it as a cramp. There had been no gastrointestinal disturbance, and a catheter specimen proved negative. The pain and soreness gradually wore off. A week later, I sectioned her and carefully examined all the organs of the right abdomen, without finding anything that offered an explanation. However, when coming out of the anesthetic, the patient again awoke to this fearful pain, which again was similarly localized, and would not respond to morphine. Suddenly its true character was defined, and the patient was given almost magical relief, by the administration of adrenalin.

In another somewhat similar case, the spasm of the oblique muscle could be distinctly felt. It was promptly relieved by adrenalin. Such abdominal spasms in the majority of pregnancies fortunately are of short duration, and do not require treatment. But it is well to be au fait as to their true character, and to the knowledge that they can be relieved, but not by morphine or other narcotics; antispasmodics would be a more logical procedure.

Cerebral Allergy.—Last, the effects of allergy upon the central and peripheral nervous system. In the previously quoted work on allergy, the authors described a case of allergic paresis during pregnancy, due to excessive extravasation of fluid from the choroid plexus. It is now well recognized that allergic edema of the brain is extremely common, rarely expressing itself in convulsions, but commonly in migraine, hemicrania, hemifacia, neuritis, papillary and conjunctival edema.

DIAGNOSIS

The diagnosis will rest upon the history. A meticulously careful inquiry into the personal and family history for some of the stigmas of allergy is one of the most important preliminary steps. The presence or absence of hay fever, asthma, bronchitis, eczema, prurigo, local edemas, acute poisoning by certain food producing vomiting bouts, neuralgias, and migraine, especially at the time of menstruation should be noted. These should be followed by a careful physical examination, including all the sense extension means to exclude organic disease. The basal metabolic rate should be established, and when normal, if the clinical picture of allergy is convincing, the thyroid therapeutic test will frequently prove that the metabolic estimation is wrong. The writers invariably add 10 to a minus reading and subtract 10 from a plus,

thereby getting, it is thought, a fairer record of actual states by allowing for the nervous excitation of a first test. Allergics are notably hypothyroids, and a large number of them are achlorhydrics, so that before and after meals gastric free acidity test is essential, unless one applies the therapeutic test. Anemias are frequently profound and a distinct shift to the right, especially due to eosinophiles, is not uncommonly found, particularly during an attack. A blood sugar estimate must never be overlooked.

One of the worst cases of flaming vulvitis was encountered recently, in which the urine, on repeated examination by her physician, which had always been negative, showed blood sugar of only 98 m.g. before meals and after eating testmeal record of 119 m.g. Insomnia from the pruritus was almost dementing. She responded promptly to 5 unit doses of insulin, and was free from symptoms, but not of local discoloration, in five days.

TREATMENT

It will be found that a large percentage of allergics are facultative, conscious of discomfort only when the general health has sunk below the reserve of a gland, or of any system. In these it is but necessary to build them up so that they are again within their reserve, to bring about a clinical submergence of the allergic state. It cannot be too emphatically stated that these temporary allergic states, frequently accompanied by the most distressing symptoms, are due to a vitiation of function, brought about by a great diversity of activating causes, and it requires but the removal or correction of this cause, or causes, to submerge again the constant factor of hereditary allergic susceptibility. Nor can it be too strongly emphasized that no function of the body is left to hazard. Each and every function, no matter how insignificant, is under control. This control is generally vested in the endocrines, or in katalytic substances, the reduction or absence of which precipitates a vitiation and a consequent poisoning of the system, so that the lowered nutrition which follows, permits an emergence of the hereditary taint. In the vast majority of instances, it is but necessary to correct the dominant defect to re-cover the hereditary subsoil. Thyroid is the dominant endocrine dysfunction in these cases, and fortunately it is almost always a deficiency disease, so that supplemental administration of the extract becomes a simple procedure. Whether this is merely an accidental association, it is impossible to state with any degree of assurance, but the association of the two conditions is so common, beyond any possibility of explanation by the law of chance, that there would seem to be a decided interdependence, as cause and effect, or due to a common cause. However, the establishment of these associated defects gives one a ready means of treatment, by thyroid and hydrochloric acid. The allergic response is usually prompt and complete.

Glycemia should be excluded or, if present, treated appropriately. Anemias should be overcome. Parasitic intestinal diseases should be studied. Everything should be investigated to find a vitiating cause which did not exist when the patient was formerly nonallergic.

The chronic cases, frequently allergic to many things, are the most deserving of our keenest study. They are highly susceptible cases, in which the hereditary influence is usually pronounced, and in which the hereditary subsoil is constantly, slightly, or much exposed.

They have a multiplicity of symptoms, affecting now one system, now another, dyspnea, tachycardia, arrhythmia, migraine, neuritides, gastric disturbances, colitis, spastic dysmenorrhea, menorrhagia and metrorrhagia, articular pains, etc. They constitute the great majority of our so-called neurasthenics. The contributing cause for aggravation may be economic, moral, mental, or emotional. They are not the self-pitying type, but, being conscious of their unwonted irritability, generally state that they are sorry for their husband or their children.

Frequently a change of scene, and a freedom from household cares, bring about almost magical changes. Other cases will frequently tax one's ingenuity, as well as one's patience, to the utmost limit. When improvement is brought about, it is usually temporary. Relapses are frequent and discouraging.

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Jordan

tissue are all abnormally relaxed, and thus permit the extrusion of this portion of the terminal bowel through the anal-orifice.

The classification of prolapse of this organ is based upon the degree of descent of the bowel wall. There are two varieties:

(a) Prolapse of a superfluous redundant mucous membrane

(b) Prolapse of the muscle coats of the rectum.

In this second type the mucous membrane may or may not be superfluous and relaxed. From his experience, the author disbelieves that (b) is merely an advanced stage of a mucous-membrane prolapse. As will be presently pointed out, the factors operative in producing either type of prolapse are somewhat different[1] and a complete prolapse may occur at once without being preceded by a prolapse of the mucous membrane. The mucous-membrane variety of prolapse is easily recognized. The extrusion is a short one and palpation between the thumb and index finger reveals only a thin band of tissue composed of two layers of mucous membrane. The protrusion is smooth without furrows and continuous with the peri-anal skin. The extruded mucosa is easily sucked back into the bowel lumen. Complete prolapse of the rectum presents itself as a large, thick mass and has an apex at its lowest extremity. There are a series of circular folds and there is a moderately deep sulcus between the skin of the anal margin and the wall of the prolapse. Anteriorly, the recto-vaginal or recto-vesical pouch of peritoneum is usually dragged down with the prolapse. This may contain loops of small bowel. Reduction is not easily effected as compared with the mucous membrane variety. (Figs. 1 and 2.)

FIXATION OF THE RECTUM IN THE PELVIS

Although the rectum is quite firmly fixed in the pelvis, it nevertheless enjoys a moderate degree of mobility and is capable of marked variation in diameter.[2] The rectum usually begins at the level of the

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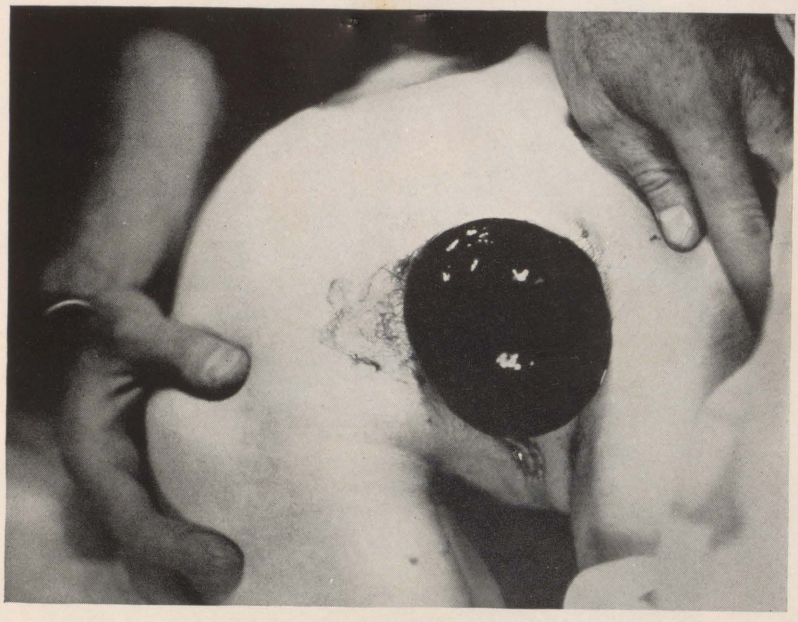
third sacral vertebra where it is continuous with the pelvic colon, and ends where it pierces the pelvic diaphragm at a point about 1½ inches anterior to the tip of coccyx. At this point there is marked narrowing of the bowel which turns abruptly backwards at a right angle (opposite prostate in the male) to become the anal canal. The rectum lies in the concavity formed by the sacrum and the coccyx and then rests for about 1½ inches on the pelvic floor formed by the union of the levatores ani muscles and their fasciae. The anatomic fixation of the rectum in the pelvis is accomplished by the peritoneum which holds the bowel to the walls of the true pelvis, particularly in the lateral planes where the peritoneum becomes quite thickened, to form the lateral ligaments. Posteriorly, between the rectum and the sacrum (where peritoneum is lacking), there is a dense area of connective tissue (really the deep fascia) which higher up carries the superior haemorrhoidal vessels in the form of a heavy rectal stalk. In front, the recto-vesical or recto-vaginal folds of peritoneum are present as supporting structures. Below these and anteriorly, the rectal wall is adherent by dense connective tissue to the prostate and seminal vesicles in the male and the posterior vaginal wall in the female. Below, firm support is offered the rectum by the levatores ani muscles and fasciae, the coccyx and recto-coccygeal ligament. The fat which normally surrounds the rectum and fills the ischio-rectal fossae reinforces in some measure, the other supports.

Although the rectum lies in the hollow of the upper and middle part of the sacrum, its long axis forms a virtual right or acute angle to the lower sacrum, coccyx and sacro-coccygeal ligament and to the sling support offered by the pelvic floor. It is the author's opinion that the coccyx, coccygeal ligaments and fascia are of great importance in preventing descent of the rectum. Two cases of prolapse of the rectum were recently observed following upon removal of the coccyx with division of its ligamentous supports. (Figs. 3, 4 and 5.)

ETIOLOGY OF RECTAL PROLAPSE

The factors operative in producing a mucous-membrane prolapse of the rectum are, in the author's opinion, quite different from those which come into play in the development of complete rectal prolapse. The mucous membrane of the lower rectum is normally very loosely attached by fibrous and elastic tissue to the submucosa.[1] This loose attachment in certain normal individuals may easily become exaggerated. This redundancy is especially evident in the anterior and lateral walls of the lower rectum. The loose attachment of the mucosa to the submucosa may easily be demonstrated experimentally by in-



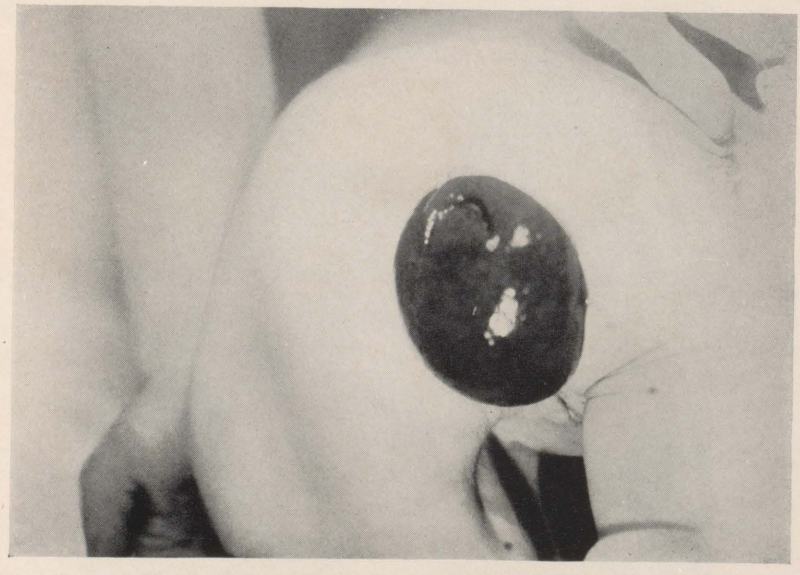


FIG. 1. Photograph of patient suffering from irreducible strangulated complete prolapse of the rectum. Woman, 58 years old. The bowel was not gangrenous. Reduction was effected under low spinal anaesthesia. The case was subsequently treated by submucous and para-rectal injections (described in the text) without any operative procedures on the pelvic floor or ano-rectal outlet. There has been no prolapse for three years.

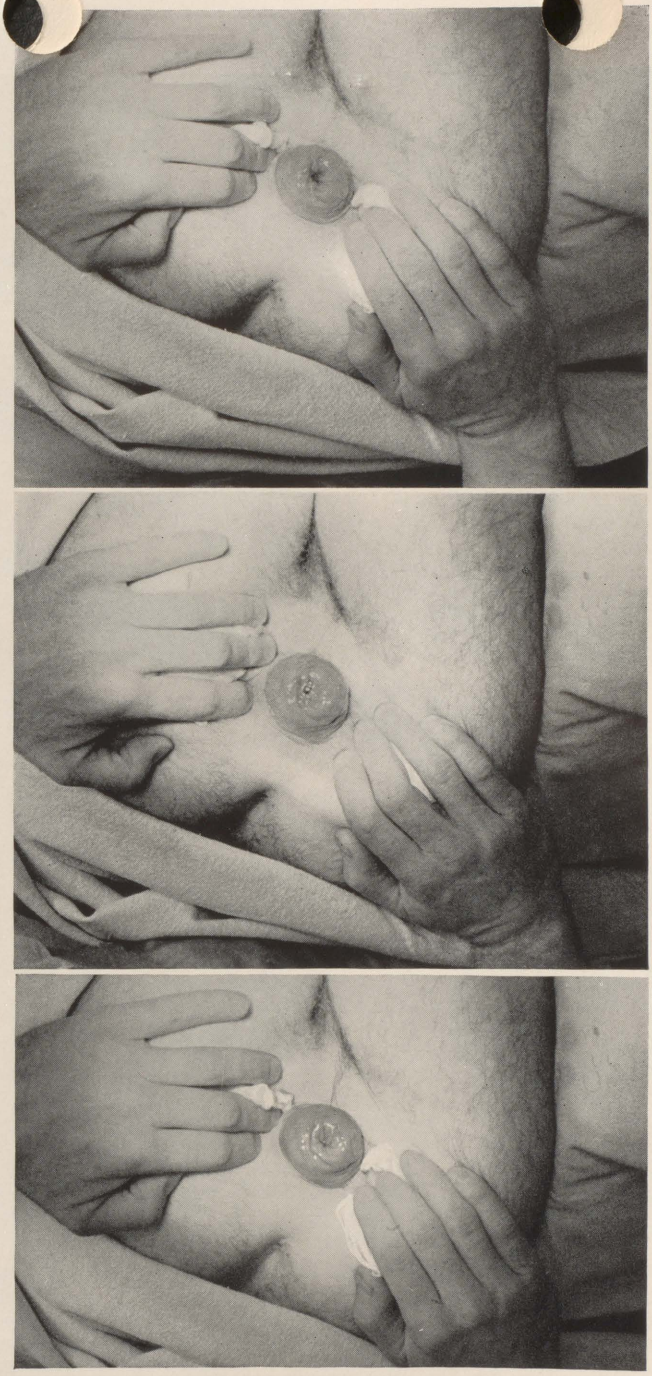
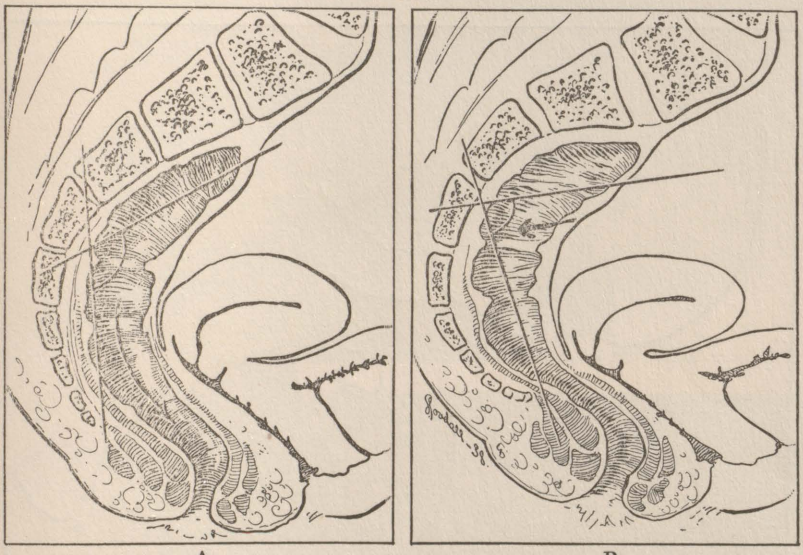


FIG. 2. Complete prolapse of the rectum. Treated by submucous and para-rectal injections followed by plication of the ano-rectal musculature described in the text.

jecting either water or normal saline submucosally. It will be observed with what ease the redundant mucous membrane may be floated off its submucosal bed. A loosely attached, redundant mucous membrane in the next stage becomes quite superflous and in concertina-like fashion rises up into the ampulla recti. It may now be easily seen that if the anal orifice permits, this redundant superfluous mucous membrane may be extruded and the condition known as mucous-membrane prolapse of the rectum becomes established. The extrusion



A B

FIG. 5. A. This is a diagrammatic representation of the flat type of sacrum found in many of the author's cases of rectal prolapse. The long axis of the rectum forms an obtuse angle with the long axis of the lower sacrum and coccyx. The bowel does not fit into the "sacral bowl" found normally. The arrow indicates the direction of gravity directed through the anal orifice, naturally predisposing to prolapse.

B. Showing the normal type of sacrum. An acute angle is formed, and gravity directs the long axis of the rectum almost in a right angle against the lower sacrum and coccyx thus preventing prolapse through the anal orifice.

of a redundant mucous membrane through the anal orifice depends more upon the anatomic condition of the anal tube than upon the degree of the redundancy. In normal individuals the anal canal (i.e., the distance from the skin of the integument to the ano-rectal line, a distance normally of approximately 2 inches posteriorly and 1 inch anteriorly) is long, narrow, slit-like and tubular. The anal canal moreover, at the ano-rectal junction (opposite the prostate in the male) as already pointed out, bends abruptly backward and its long axis

forms an obtuse angle with the long axis of the rectum. This can be verified in doing a digital examination in such individuals, when, in traversing the anal canal, the tip of the finger points towards the umbilicus and in females is especially evident as it follows the posterior vaginal wall. A sharp angle or ridge is thus encountered at the anorectal junction and in entering the rectum the finger turns abruptly backwards towards the spinal column. Such a tubular anal canal, with its long axis in a different plane from that of the rectum, acts as a

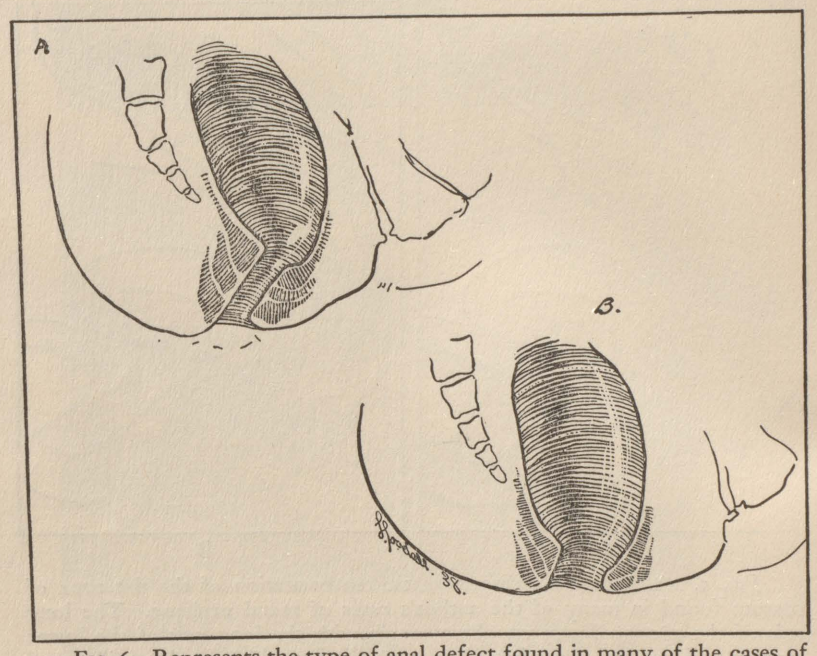


FIG. 6. Represents the type of anal defect found in many of the cases of rectal prolapse.
A—Represents the long tubular type of anal canal (Normal).
B—Represents the abnormal type predisposing to prolapse.

formidable barrier to the extrusion of any of the components of the bowel wall, such as a redundant superfluous mucosa. When this occurs, the whole of the lower rectum and ampulla recti on proctoscopic examination will be observed to be crowded and filled with this redundant, concertina-like mucosa. However, upon questioning the patient no history of prolapse or extrusion is recorded. The patient on the other hand will be conscious of a feeling of fullness as if a foreign body were present in the rectum. (The author would use the name of "concealed mucous-membrane prolapse" for this variety.)

A

B

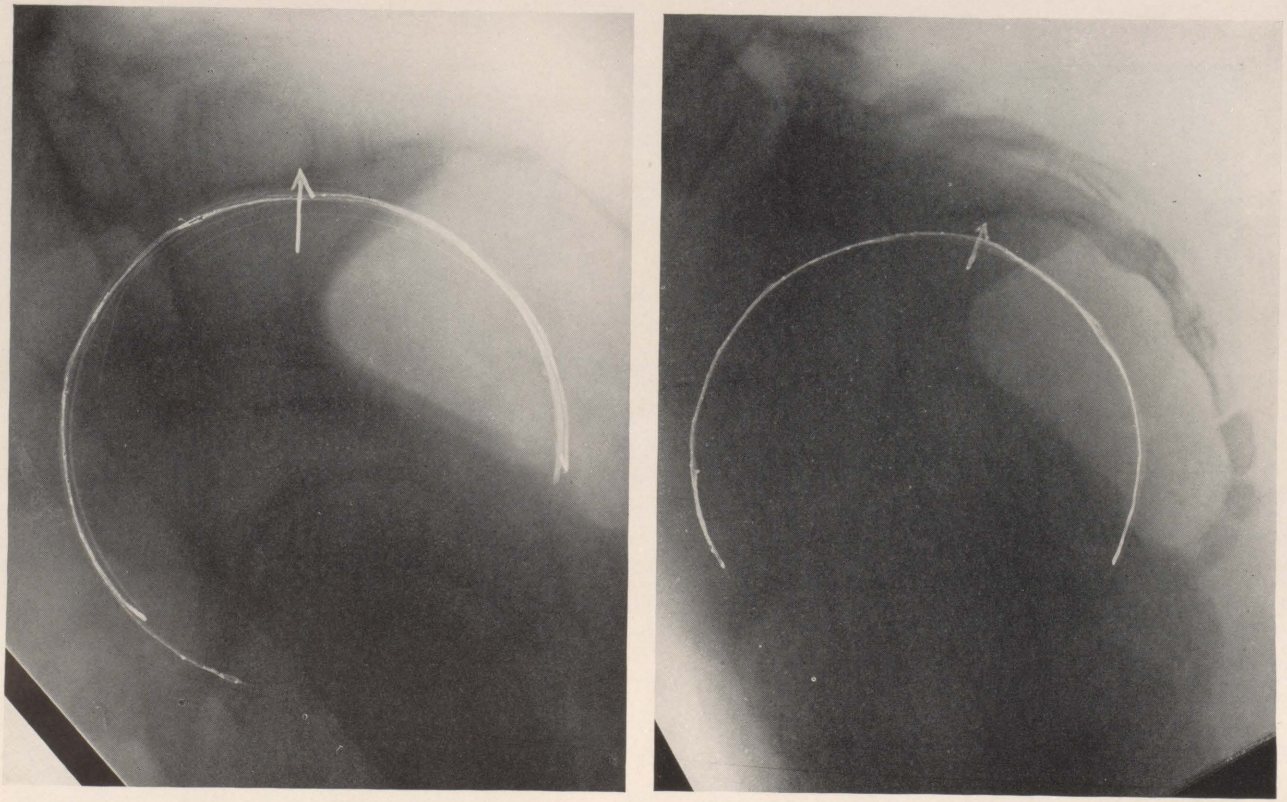


FIG. 3. A, B, and C. X-ray studies of the sacrum. The inner surface of this bone is quite flat as compared with the normal. This is more obvious upon digital examination through the rectum. The arrows indicate the flat area. The straight coccyx and anal deformity may or may not accompany this type of sacrum. The latter defects may be present singly as described.

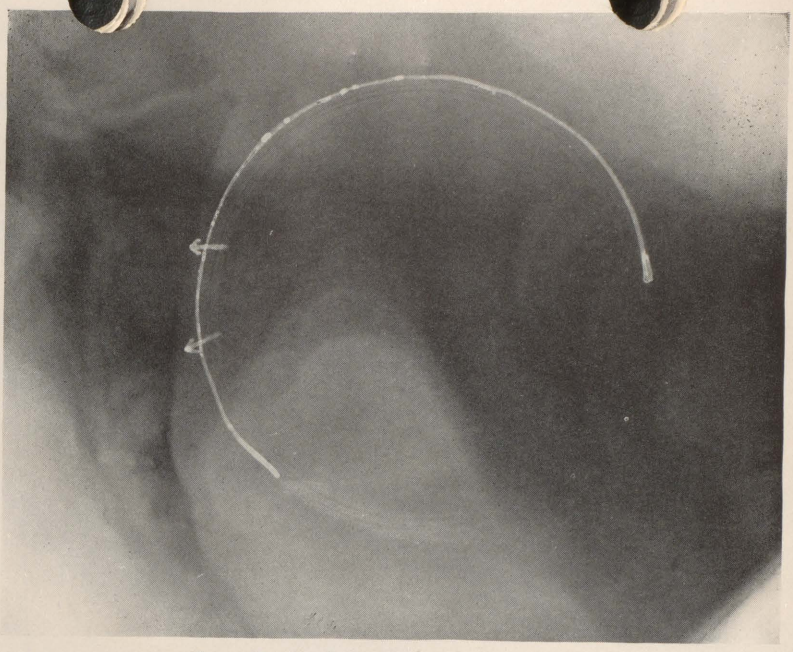


FIG. 3C. See legend on previous page.

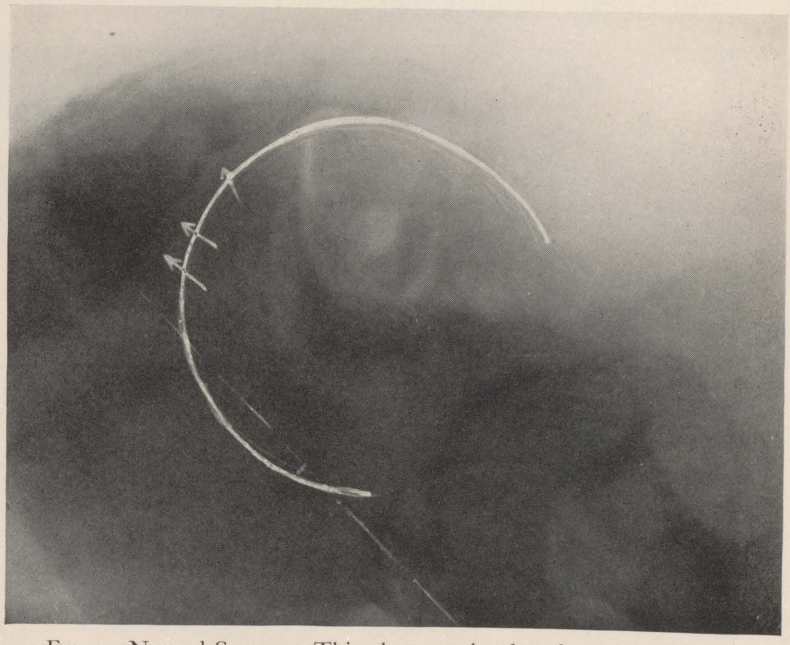


FIG. 4. Normal Sacrum. This photograph taken from a pelvis showing a well-hollowed sacrum, especially apparent on digital examination and stereoscopic study.

Backache will frequently be complained of in this "internal" non-extruding type of mucous-membrane prolapse. Constipation is a common occurrence since this redundancy fills the lower rectum and precedes the stool. Such individuals frequently develop a low-grade proctitis with anal fissures and pruritus. There may be a mucoid or muco-purulent discharge.

In the other type of individual, where the anal canal is not tubular and angulated but short, stout and patulous a mucosal redundancy will

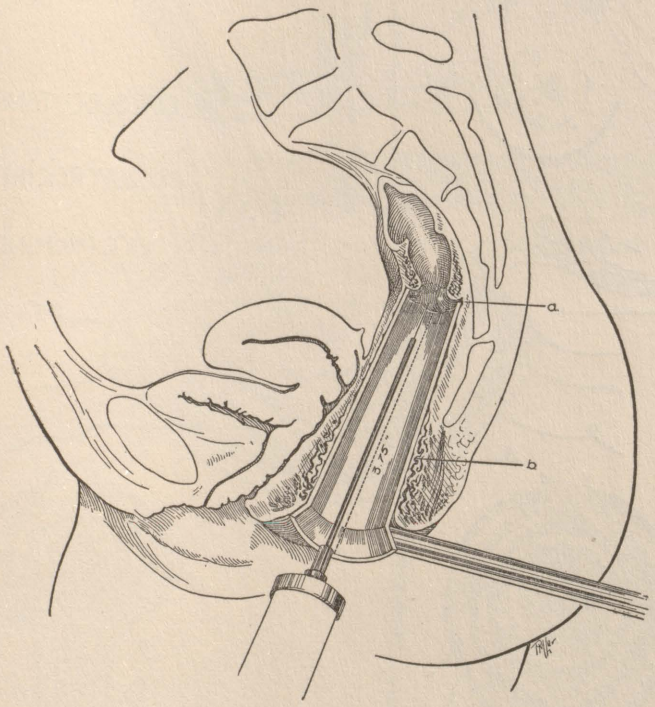


FIG. 8. Submucous injection of redundant superfluous mucous membrane with sclerosing solution.

be easily extruded. In the former type, digital examination is not easily carried out and the ano-rectal musculature grips the finger as a long, tubular, constricting structure. In the latter type, digital examination is easily carried out, and the anal canal is recognized as a loose orifice, easily traversed by the examining finger and not possessing the ano-rectal angulation already described.

Prolapse of the rectum in children may present itself as a mucous-membrane type or as a complete prolapse including the muscle coats.

The plane of the infant pelvis is rather vertical and the inner surface of the sacrum quite flat.[3] The coccyx possesses very little of a forward tilt and may lie almost in a straight line with the sacrum. The anal orifice, too, has a more posterior position than that seen in adults, and a line drawn down from the tip of the coccyx will be found to be

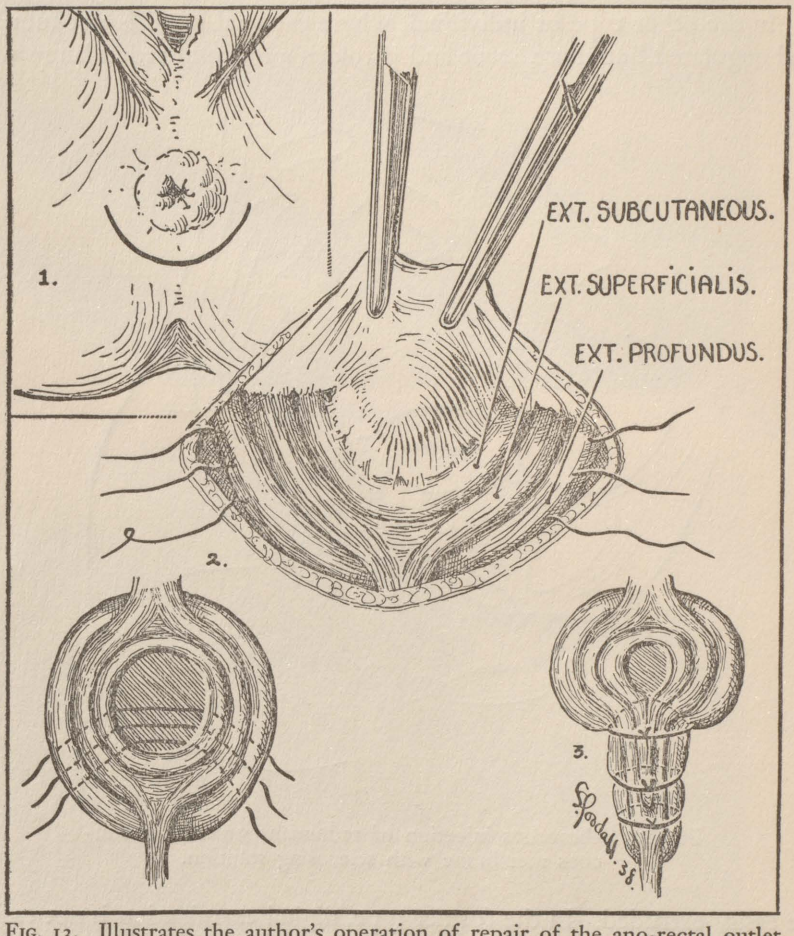


FIG. 13. Illustrates the author's operation of repair of the ano-rectal outlet described in the text.

almost in a straight line with the anal orifice. It will thus be seen that although children often present a mucous-membrane variety of rectal prolapse, conditions are favorable for the development of a complete type of descent. It is thus apparent that in infants undue loss of weight, diarrhoea and straining at stool are very often sufficient to

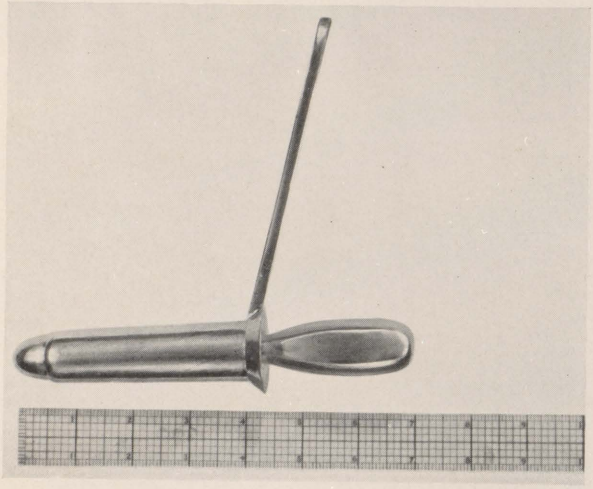


FIG. 7. Author's adult pattern of proctoscope.*

* Brass & Bronze Manufacturing Co., Limited, 269 Craig Street West, Montreal.

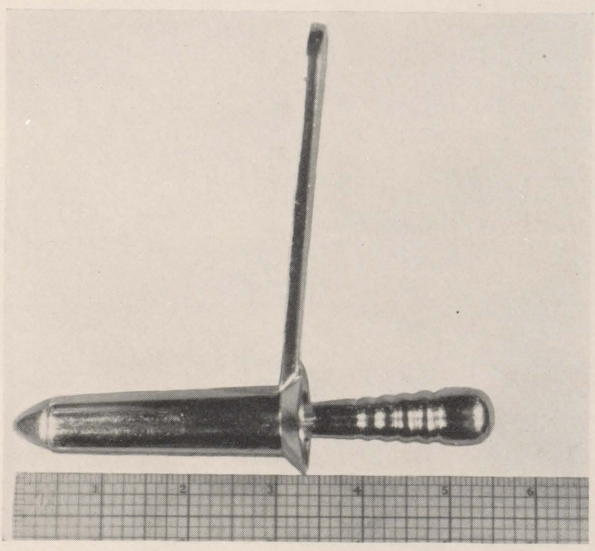


FIG. 9. Author's pattern of infant proctoscope* used in injecting the submucosal space.

* Brass & Bronze Manufacturing Co., Limited, 269 Craig Street West, Montreal.

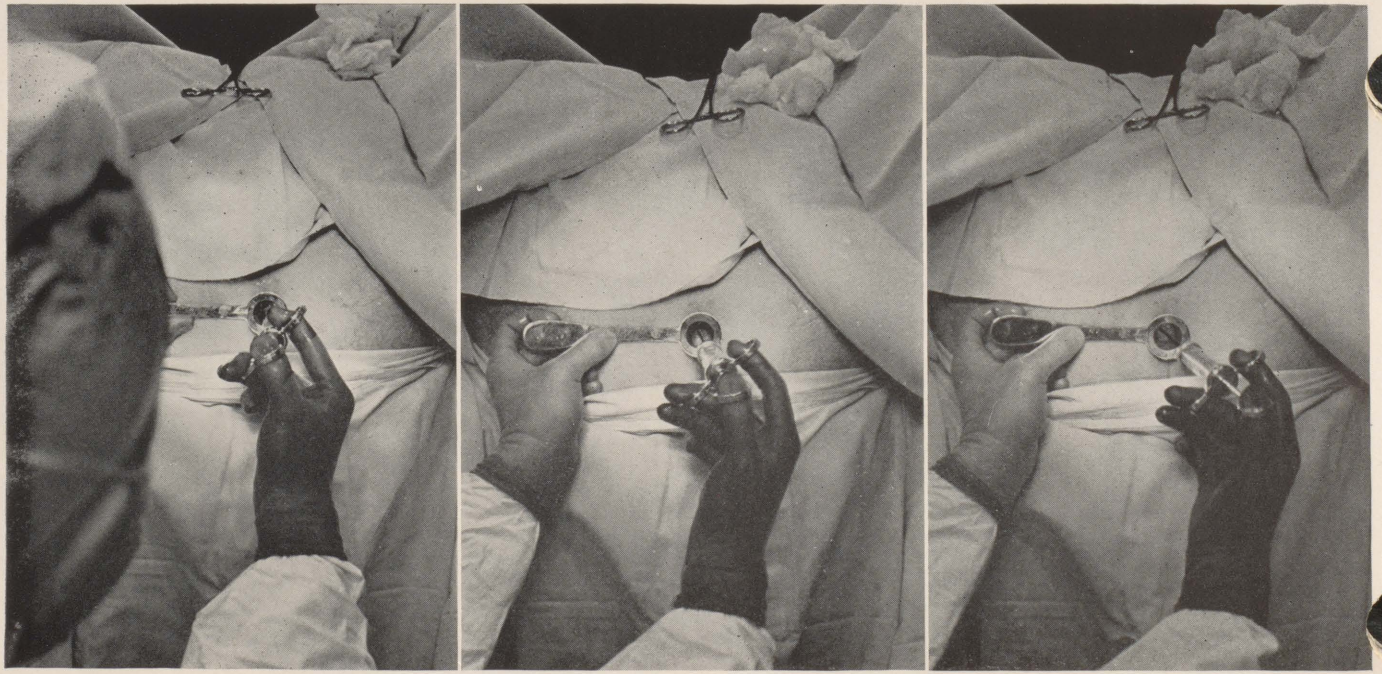


FIG. 10. Demonstrates the technic of submucous injection described in the text.

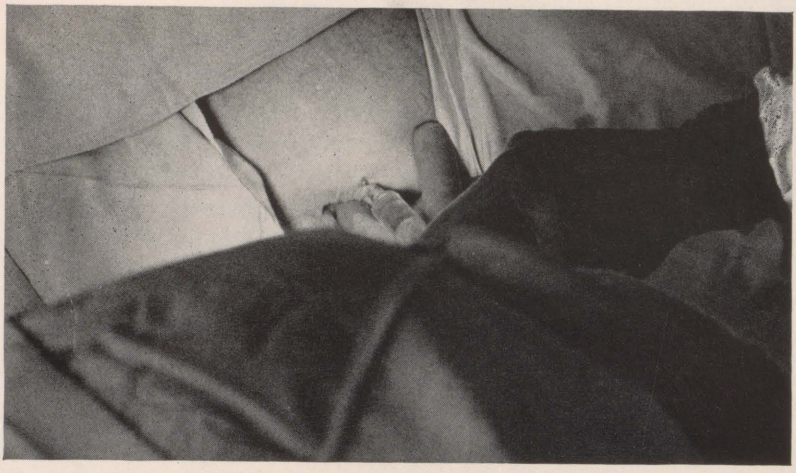


FIG. 11. Represents a pararectal injection with the finger in the anal canal guiding the direction of the needle.

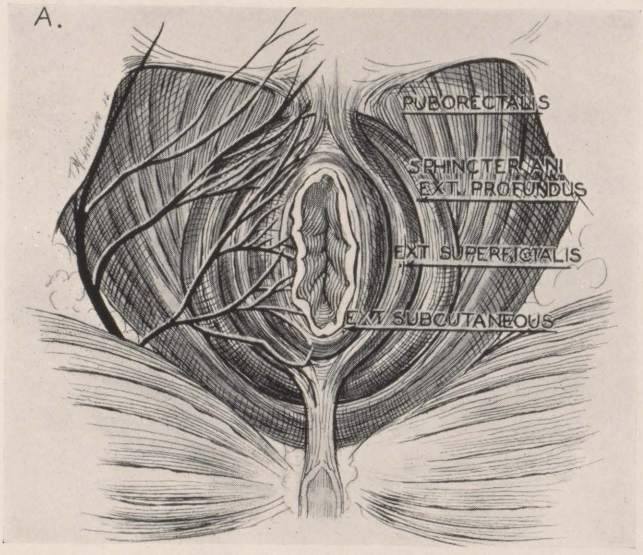
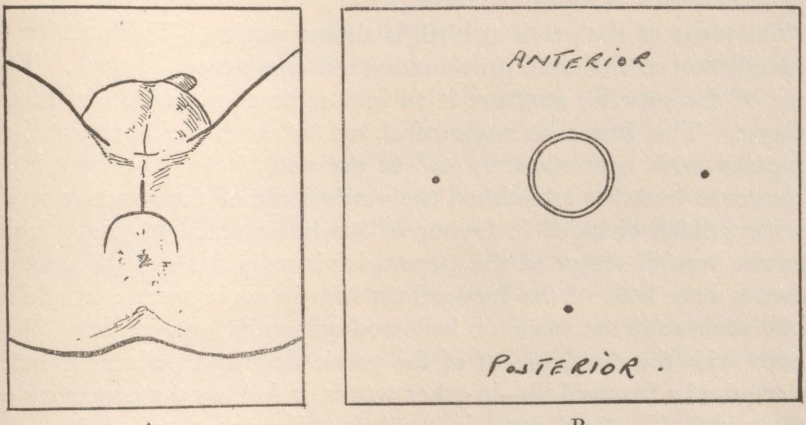


FIG. 12. Represents the musculature of the ano-rectal region viewed from below. The three portions of the sphincter ani externus muscle are shown in their respective planes. The inferior haemorrhoidal and perineal nerves are shown in position on one side. (Reproduced through the courtesy of the Amer. Jour. Digest. Dis. and Nutr., 3:775, 1936. Daniels, E. A.: Anal Fissure, Anal Spasm, and Anal Stenosis.)

initiate either an incomplete or a complete prolapse of the bowel, since the stage is set and conditions are favorable for such a disturbance.

Complete prolapse of the rectum is, in the author's opinion, not frequently preceded by the mucous-membrane variety, but more often than not commences at once as an extrusion of both mucous membrane and muscle coats of the bowel wall. A large redundant mucosal prolapse may eventually drag down the muscle coats of the rectum during the process of descent. However, in the author's cases of complete rectal prolapse, it appears that very few of these were preceded by a mucous-membrane type of extrusion. In this type of prolapse,



A B

FIG. 14. A—Represents the incision in a male patient as the initial step in performing the plastic procedure upon the levatores ani muscles described in the text.

B—Represents the three areas of needle punctures in performing the para-rectal injection described.

the whole of the rectum descends through the anal orifice. Reduction may be easily effected or, on the other hand, its return may become impossible. Obstruction of the blood supply through the prolapsed bowel occurs, edema ensues and in severe cases the lesion may present itself as a strangulation in the form of a large, bluish, edematous, deeply engorged mass with areas of necrosis on the surface of the lesion. The factors operative in initiating a complete descent of the whole rectum are mechanical in nature. One should first discuss what actually occurs to the rectum in complete prolapse and then try to tabulate certain of the conditions which would predispose to such a state. Complete prolapse may be defined as a loss of the normal degree of fixation of the rectum in the pelvis. The supporting structures of this organ become lax, stretched and inefficient and with conditions

favorable at the outlet, permit its descent. The peritoneum supporting this portion of the terminal bowel especially its lateral ligaments, the retro-rectal connective tissue, the meso-rectum carrying the haemorrhoidal vessels, the dense connective tissue between the rectum and the prostate and seminal vesicles in the male, and the posterior vaginal wall in the female, are all stretched and subluxated. The rectum now lies more or less unsupported in the true pelvis. Its excursion in almost any direction is now possible. Gravity now favours its downward descent and one has only the resistance offered by the pelvic floor and the anal tube to prevent the complete extrusion of this portion of the terminal bowel.

The plane of the pelvis at birth is almost vertical. This position is gradually lost as the child grows older and commences to walk. The plane of the superior aperture is an important consideration in rectal prolapse. This forms an angle of about 80° with the horizontal, as compared with approximately 60° in the adult.[3] In other words as adolescence becomes established the vertical tilt of the superior strait has been much changed in favour of the horizontal. In young children the ventral aspect of the sacrum is virtually flat and the coccyx possesses very little of the forward tilt seen in adolescence and adults. In the adult stage the sacrum is hallowed out on its inner surface. The coccyx with the development of the pelvic floor and perineal muscles has acquired a forward tilt—in other words, it follows the concavity of the sacrum. The anal canal in the infant lies completely below the level of the tip of the coccyx, and the anal orifice occupies a more posterior position. In other words a vertical line drawn downward from the tip of the coccyx will be found to pass through the anal orifice, whereas in the adult such a line generally will be distinctly behind it. The rectum in the infant is a straight tube possessing very little of the lateral and antero-posterior curvature seen in the adult bowel. It has a straight descent and does not, as in grown-ups, lie in and fill out the concavity formed by the inner surfaces of the sacrum and coccyx. In the adult, the long axis of the rectum therefore lies at a virtual right or an acute angle to the lower portion of the sacrum and to the coccyx and at the same time its posterior wall follows the concavity formed by the inner surface of the upper and middle thirds of the sacrum. The rectum, therefore, in trying to descend, would impinge almost at right angles against the lower portion of the sacrum and coccyx and recto-coccygeal ligament. In the infant a straight line drawn from the superior strait of the pelvis would virtually drop out of the anal orifice, and it will thus be seen that the long axis of the rectum in young children is almost parallel to the long axis of the

sacrum and coccyx. The anal canal in children is quite straight and continues directly downwards from the rectum, whereas in normal adults, as already pointed out, a rather sharp angulation backwards occurs at the ano-rectum (opposite the prostate in the male) and the examining finger, in the adult will recognize the anal canal as running in a forward direction. In grown-ups, this anatomic configuration of the anus in the presence of a well developed tubular canal acts as a resisting force in preventing prolapse.

Age of Onset. The author believes that complete prolapse of the rectum becomes initiated in early childhood and adolescence due to a retarded development of these adult characteristics of the pelvis and rectum. Stretching of the supporting structures of the rectum thus takes place over a long enough period of time to enable the prolapsing bowel to overcome the resistance offered by the anal outlet (usually a short vulnerable one) and the process of complete rectal prolapse has become established. In the author's series of cases of complete prolapse of the rectum examination has nearly always revealed a rather flat inner surface of the sacrum with a coccyx possessing very little forward tilt. In a high percentage of these patients the anal orifice was observed to be almost in a straight line with the tip of the coccyx and not definitely anterior to it as observed in normal adults. The anal canal too, was found to be short, vulnerable, easily traversed and quite lax.

The pelvic floor formed by the levatores ani muscles and their fasciae and especially that formed in front by the puborectales portion of this muscle is an important consideration. The pelvic floor acts as a sling in supporting the rectum especially anteriorly. In parturition rupture of this support will favour descent of the rectum, particularly so if the abnormalities described should be present.

The long, tubular type of anal canal already described in which the sphincteric muscles are well developed and quite powerful acts as a definite barrier to the extrusion and descent of the rectum. As already pointed out, certain individuals possess very little of this type of anal configuration, but have a rather short patulous, "sawed-off" anal orifice. This latter type of anal canal with or without the abnormal type of pelvis already studied definitely facilitates the descent of the rectum.

From this description it will be seen that certain anatomic and mechanical factors when present will favour and predispose to complete prolapse of the rectum. These conditions, above outlined, initiate a downward excursion of the terminal bowel, stretching of its supporting structures and its eventual extrusion through the anal orifice. It will thus be seen that a good pelvic floor in the presence of a long,

tubular, more or less rigid, anal canal will offset an infantile type of rectum and pelvis in preventing prolapse. It is for this reason that complete prolapse of the rectum will often spontaneously follow parturition, coccyxgectomy or fistulotomy where sphincter tissue has been sacrificed. Large protruding haemorrhoids or a mucous-membrane prolapse producing an eventual relaxation of the anal sphincters in the presence of the above predisposing mechanical factors may initiate a complete prolapse of the rectum. Complete prolapse thus following spontaneously upon any of these factors, however, must pre-suppose the presence of either the abnormal type of pelvis or anal tube already described. In the author's experience loss of weight and loss of the fatty tissues surrounding the rectum will not in itself favour prolapse of the bowel unless the pelvis and outlet are such as to favour extrusion of the rectum.

Examination of a case of complete rectal prolapse during the act of extrusion of the mass presents a characteristic appearance. This may be likened to the appearance of the uterus during the last stages of labor in which the lower uterine segment has become obliterated. The lowest cone-like segment of the uterus becomes part of the upper uterine segment and undistinguishable from it. In the same fashion the slit-like anal canal has become obliterated as shown in the diagram. One sees merely a bulging spheroidal structure impinging against the perianal tissues with only the thinned-out and stretched subcutaneous portion of the external sphincter visible under the skin, as the last barrier. A complete corona of dilated external haemorrhoidal veins may be seen between the skin and the fibres of this subcutaneous muscle.

TREATMENT

The treatment of prolapse of the rectum first implies that a careful study and diagnosis of the patient has been arrived at. It is essential that the operator determine whether the lesion is a mucous-membrane one or whether one is dealing with a complete prolapse of the rectum. An effort must be made to see the condition in the prolapsed state. One should then encounter no difficulty in determining whether the prolapse consists only of mucous membrane or includes the muscle coats as well. The bony structure of the pelvis must be carefully studied and the amount of relaxation of the anal orifice determined. If the patient is a woman who has borne children the presence of a ruptured perineum must be sought for. One should examine a case for any evidence of fistulotomy or other rectal operative wounds.

In studying my cases of rectal prolapse, I was struck by the peculiar type of anal canal already described which a high percentage of these

patients presented. There was this short "sawed-off" type of anal orifice in a vertical practically straight line with the coccyx (anus normally far to the front) easily traversed by the examining finger which seemed to "fall" at once into a roomy ampulla. This has already been compared to the obliterated lower uterine segment seen in the last stages of labor. In many of these patients the inner aspect of the sacrum on digital examination presented itself as more or less of a flat surface by comparison with the normal. X-ray studies in these patients, particularly the stereoscopic views, bore out this finding. The coccyx in my cases of rectal prolapse had very little forward tilt as found normally. In other words, it pointed directly downwards in a straight line with the long axis of the sacrum instead of having a forward curve in a hammock fashion. In two of my patients suffering from complete prolapse of the rectum, coccygectomy had been performed six months previous to the onset of symptoms. Of the etioligic factors above outlined found more or less consistently in my cases of rectal prolapse, it was my considered opinion that the mechanical anal defect already described was the more constant and the more important of the predisposing factors outlined.

One cannot reconstruct the pelvis nor remove the predisposing factors operative in producing rectal prolapse, but with the knowledge at hand concerning this condition one can carry out certain conservative but effective measures based upon the etiology outlined in order to correct or alleviate the condition. These measures have been very gratifying in the author's hands in a series of 22 cases of complete rectal prolapse and in well over 50 cases of mucous membrane prolapse (incomplete) treated during the past four years. The principal underlying treatment consists of fixing in the pelvis the descending portion of the bowel which may be only mucous membrane or include all the muscle coats. One attempts a "restitutio ad integras." In prolapse of the rectum, the supporting structures of the bowel which keep the uppermost part of the rectum from about the level of the third sacral vertebra fixed in the hollow of the sacrum have all become subluxated, stretched and inefficient. These supporting structures have been described.

The author's plan of treatment consists as follows:

A. Obliteration of the redundant superfluous mucous membrane by a sclerosing solution.
B. Fixation of the rectum in the true pelvis by surrounding this organ with an area of dense inflammatory scar tissue, employing a sclerosing solution.

C. Repair and reinforcement of the ano-rectal sphincteric musculature.
D. Perineorrhaphy operation in either males or females to supplement A, B, or C.

The author has thus far never had to resort to the radical operation known as recto-sigmoidectomy which, on the basis of this work, would seem to be the only radical operation suitable for the permanent cure of this condition, should the above conservative measures fail. In his series of patients the conservative measures to be described have been sufficient to either completely correct the disturbance in the majority of cases or to give sufficient relief to make the patient's life agreeable.

A. In dealing with either the incomplete or complete type of rectal prolapse, the writer carries out a thorough plan of injecting the redundant mucosa with a 5 per cent solution of phenol in sweet-almond oil in the submucosal plane. In many cases of complete rectal prolapse there may be very little or no mucosal redundancy in which variety the whole rectum descends at once without being preceded by a mucous-membrane prolapse. In this type as well, the author carries out his plan of producing some degree of submucous indurative reaction as the first step in dealing with the lesion. A long tubular proctoscope (Fig. 7, author's pattern)* is employed in carrying out the submucous injection. From 10 to 30 cc. of 5 per cent phenol in sweet-almond oil is injected submucosally employing a St. Mark's Hospital type of Luer Lock syringe. The whole of the redundant mucous membrane is thus floated off its submucous bed. (Figs. 8, 10.) This technique is described in detail under reference No. 1. At the completion of this procedure the whole of the ampulla recti is packed with vaseline gauze through the proctoscope and the instrument slowly withdrawn. This packing is left in from 24 to 48 hours and assists in effecting a submucous indurative reaction and at the same time assists in fixing the redundant mucous membrane to the submucosa. This procedure may be all that is required in the mucous-membrane type of prolapse of the rectum but on the other hand will merely be the first step in dealing with the complete variety. In infants suffering from mucous-membrane prolapse of the rectum the author has employed this technique,[3] employing his small infant proctoscope* (Fig. 9) and using from 5 to 15 cc. of 5 per cent phenol in sweet-almond oil. One may have to repeat this treatment after a period of weeks or months. No anaesthesia is required for the submucous injection.

B. In complete prolapse of the rectum one carries out the next step[4] in dealing with the lesion. This consists of a para-rectal injection of

* Brass and Bronze Manufacturing Co., 269 Craig St. West, Montreal.

quinine sulphate in dilute sulphuric acid. (Fig. 11.) (Quinine sulphate 12 gr. dilute sulphuric acid 30 minims, water 30 minims, the whole sterilized by boiling.) This measure is carried out under low spinal anaesthesia, within a week following the submucous procedure described under A. A long, 4-inch, 18- to 20-gauge needle is employed and three points are selected—two lateral and one posterior, each about an inch from the subcutaneous portion of the external sphincter. Between 9 and 12 cc. of this solution is employed in carrying out the para-rectal injection. The solution is injected laterally on each side of the rectum, both above and below the levatores ani. Posteriorly the solution is injected into the retro-rectal fascia between the rectum and the sacrum. Between 3 and 4 cc. is injected in each plane. On rare occasions, in fat robust individuals, the author has employed as much as 5 cc. in each plane. A firm binder with a thick perineal support is placed over the perineum and the patient returned to his room. Within several days when the finger is inserted into the rectum a fairly marked para-rectal reaction will be noted. This para-rectal technique first employed by Swinford Edwards and more recently by Mr. Gabriel[4] of St. Mark's Hospital, London, has proved very valuable as a step in the author's plan of dealing with complete prolapse of the rectum. One cannot employ a very large quantity of this solution because of its toxicity but no untoward effects have thus far been observed.

C. This next step is carried out either at once following the para-rectal injection but is better deferred for several weeks to a month to see whether steps A and B have not proved sufficient to correct the lesion. This operative procedure is indicated when there is marked relaxation of the ano-rectal outlet as the result of stretching by the protruding bowel. This operative procedure is based upon the anatomic studies of Milligan and Morgan on the anatomy of the ano-rectal musculature.[5] This procedure is carried out upon the sphincter ani externus muscle by first dissecting out its three component parts, as pointed out by the above authors. (Fig. 12.) The external sphincter, as shown in the diagram, extends up posteriorly around the ano rectum for a full distance of two inches. It would be impossible to describe the detailed anatomy of the external sphincter and the pubo-rectalis portion of the levator ani in this paper. The reader is referred to the work of Milligan and Morgan[5] and to the work on the anal canal[6] published by the author of this paper. A circular incision between the coccyx and the anus is made and one dissects down upon this area exposing the component parts of the external sphincter muscle as follows: First the subcutaneous portion which runs circularly around the anal

canal (beneath this one can see the longitudinal muscle of the bowel), one next comes upon the superficial portion of the external sphincter which runs backwards to find its insertion in the coccyx. Deep to this one finds the fibres of the sphincter ani externus profundus which fuses with the pubo-rectalis portion of the levator ani and the longitudinal muscle of the bowel to form the ano-rectal ring. In the author's plan of treatment the subcutaneous portion of the sphincter is folded over employing through and through sutures of No. 1 twenty-day chromicized catgut and next carried deeply through the superficial sphincter, into and picking up the profundus portion of the muscle in the same fashion. Two or three of these sutures are employed in this fashion, thus plicating this musculature. With the finger in the anal canal an actual tightening up of the orifice will be observed and effected. The skin is then closed with fine linen and no drainage employed. (Fig. 13.)

D. The perineorrhaphy operation in either male or female to supplement either A, B or C is carried out as a last stage when the above measures are not effective, either alone or collectively. In the female the ordinary type of perineorrhaphy, pulling up the levatores ani and tightening these, thus building up a firmer perineal body, is carried out. Such a procedure can and has been done even in nulliparae.

In the male the dissection is carried out between the bulb of the urethra and the rectal wall through a T shaped incision over the perineum as shown in the diagram. (Fig. 14.) The levatores ani are exposed and tightened by through and through sutures or over plication employed No. 1 chromic catgut. It is imperative when carrying out this perineorrhaphy that the suture be carried through the outer longitudinal muscle of the bowel wall, thus pulling up and holding the rectum as well as re-enforcing the levatores ani muscles. After the last two procedures the patient is kept on a very low residue diet and the bowels confined for a full week. Walking is not permitted for three weeks.

The author in his series of cases has never had to carry out the operation of recto-sigmoidectomy, since practically all of his cases were either greatly improved or cured by the above measures described. Recto-sigmoidectomy, which is a very radical procedure, has a definite place in dealing with the intractable type of prolapse. Fortunately such a case has not come to hand and the author feels that the conservative measures outlined in this paper will deal with the average case of prolapse of the rectum.

SUMMARY

A study of rectal prolapse has been carried out by the author in a series of cases taken from hospital and private practice. The etiology of this lesion has been investigated and in the author's experience certain factors have been consistently present. These have been described. A conservative plan of treatment has been outlined.

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GENERALLERGY AND METRALLERGY

J. R. GOODALL, M.D.
and
R. M. H. POWER, M.D.
Montreal, Que.
From the Wards and Research Laboratory of St. Mary's Hospital

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GENERALLERGY AND METRALLERGY*

J. R. Goodall, M.D., and R. M. H. Power, M.D., Montreal, Que.
(From the Wards and Research Laboratory of St. Mary's Hospital)

THESE two new composite terms are used to denote allergy of the genital system generally, and of the uterus in particular. Their derivation (from the old Greek verb, γενω, or from the more recent γίγνομαι, γενήσομαι, to bring into or beget; and μήτρα, womb, in combination with ἄλλος, other, altered, ἔργον, reactivity) signifies an altered or different reactivity of tissues of endowed individuals to substances that do not affect ordinary normals.

The substances that evoke these altered reactions are allergens, and the substances which they generate in the affected cells are allergins. These are poured out to neutralize the allergens when the latter come into contact with the specific sensitized cells. Allergens are widely distributed throughout the material world, and may be gaseous, liquid, or solid in character. They reach the innermost recesses of the individual, by absorption from mucous membranes, or by mere contact. Cells that resent the presence of allergens, develop allergins in their substance and thereby become sensitized to that specific allergen, and when this specific allergen is absorbed in quantities larger than the allergins of the blood stream can immediately neutralize, the excess allergen comes into contact with the sensitized cells, and an allergin is secreted which, by its irritating influence, causes local irritation and extravasation of plasma, by altering the normal function of the capillary lining, causing it to become excessively permeable to either plasma alone, or to all the components of the blood.

Allergins are the defensive mechanism of a highly sensitive person, much in the same way as many terrestrial animals and fish emit a defensive substance when danger is at hand.

The knowledge of the effect of the allergens upon the organs of generation is new. So little is at present known about it, that this study, we hope, will evoke a new interest. For us it has cleared many knotty problems. Pelvic allergy is full of interest and its victims are as numerous as those affected with the more easily recognizable forms of allergy. When the symptoms and pathology are described, it will doubtless create the surprise that it was not thought of before. For

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a better understanding of the subsequent description of pelvic affections of this nature, a short retrospect of the obvious manifestations will be of considerable help. Allergy expresses itself in one of two general ways. First, as an extravasatory disease, a protective reaction of which the common example is, of course, hay fever; and second, as an irritant to involuntary muscle causing spasm, of which again the classical example is asthma. There are many other diseases that are caused by one or the other of these two forms of expression, the commonest being antrum disease, migraine, urticaria, eczema, intertrigo, prurigo, local and general edema, angioneurotic edema, and neurasthenia. In this incomplete list omissions are conspicuous, but we have enumerated only the more obvious symptomatic developments.

One of the chief reasons that so little is known about allergy, is to be found in the fact that pathologists see nothing of it. Its chief manifestations are edema or spasm, both of which are not discernible at autopsy as a pathologic study.

But we feel that it is only a matter of time before a technic is evolved that will demonstrate the vascular changes which bring about the pathologic hyperpermeability, and the changes locally in the neuromuscular combination which cause continuous spasm under the influence of the allergins. Is the pathology primarily centered in the capillaries and the musculoneural endings, or is it a disease of the autonomic nervous system that brings about these changes in the affected tissues? There has been some thought spent upon these two theories, but it will probably be a long time before it is fully determined which is primary and which is secondary. Allergics are numerous, constituting from 28 to 60 per cent of individuals. There are those in this computation who are continually allergic, and constitute chronics, and there are those who are periodically allergic. These constitute the vast majority, and are chiefly those whose cases have been misunderstood because they are more difficult to detect.

In all the allergies there is a subsoil of hereditary susceptibility which may be exposed at all times as in chronic cases, or may crop up to the surface only when the oversoil is removed. The surface soil may vary in thickness, not only in families, but also in different members of the same family.

The symptomatic expression of allergy may also vary in the same individual at different ages. The allergy may shift its chief attack from one system to another, under the influence of age and environment, using that term in its broadest sense. Males with an hereditary subsoil are much more likely to show symptoms in the years of childhood, but females greatly outnumber them after the age of puberty, thereby showing the great influence exercised by endocrine imbalances upon the allergic substratum. This is important to grasp fully, other-

wise endocrinology and allergy become inextricably interwoven in our appreciation of a patient's symptomatology. It is important to remember, that in such cases the endocrinologic imbalance is the adventitious or intermediate cause, but the allergic hypersensitivity is the constant factor always present, every ready to manifest itself when the general balance is upset.

In order to bring the matter more intimately home to our readers, we would like to broaden the subject at times, occasionally referring to some unusual, unrecognized, expressions of allergy in women, as regards their systems other than the genitals, because these disgressions help to throw a great deal of light upon the more recondite manifestations of the same disease in the pelvic organs.

We have found so far, that allergy, of whatever origin, manifests itself in the pelvic system in the following symptoms, signs and pathology:

1. General pelvic edema and free fluid in pelvic cavity
2. Chronic endometrial edema and hyperplasia
3. Acute endometrial edema associated with the phases of the menstrual cycle
4. Menorrhagia
5. Acute menorrhagia
6. Metrorrhagia
7. Dysmenorrhea
8. Leucorrhea
9. Allergic vaginitis and vulvitis.

We wish to emphasize the fact that allergy is a general disease. It is due to a foreign substance in the blood, to which allergic hypersensitive individuals show a definite systemic tissue resentment, expressed in extravasation, and above all, a fact emphasized by all authorities on allergy, no system is immune to its influence.

In 1914, Osler, when considering the subjects of purpura, asthma, edema of the glottis, hay fever and similar diseases, wrote "some day some anaphylactic key will unlock the door to this dark chamber." How true his prophecy! Only nowadays we use the term anaphylaxis for animals, and allergy for human beings. It is but a refinement of terminology, though there are certain distinct differences in the two diseases, due to differences in aeons of evolution.

For several years, Goodall was tremendously impressed when opening the abdomen, to find a diffuse edema of all the pelvic organs, a thickening of the peritoneum, and free fluid in the pelvic cavity. Many such cases have since been noted. The peritoneum is edematous, there are bullae upon the surface of the uterus, and widely dilated lymph spaces in the broad ligament; thickening of the bladder, toneless, edematous, swollen pale tubes; enlarged cysts of Morgagni and large edematous ovaries, sometimes without evidences of recent ovulation cycles, sometimes simple cystic hypersecretions. The uterus is larger

than normal, and extremely soft, like an early pregnancy. Eight such cases were described before their true nature was detected. Let us describe one of these very pronounced recent cases in Dr. Goodall's service. Of course, it must be well understood that the signs described above, vary in degree in different individuals. At one extreme is the case about to be described, at the other extreme the signs may fade imperceptibly into the normal, and minor degrees of the condition might readily pass undetected.

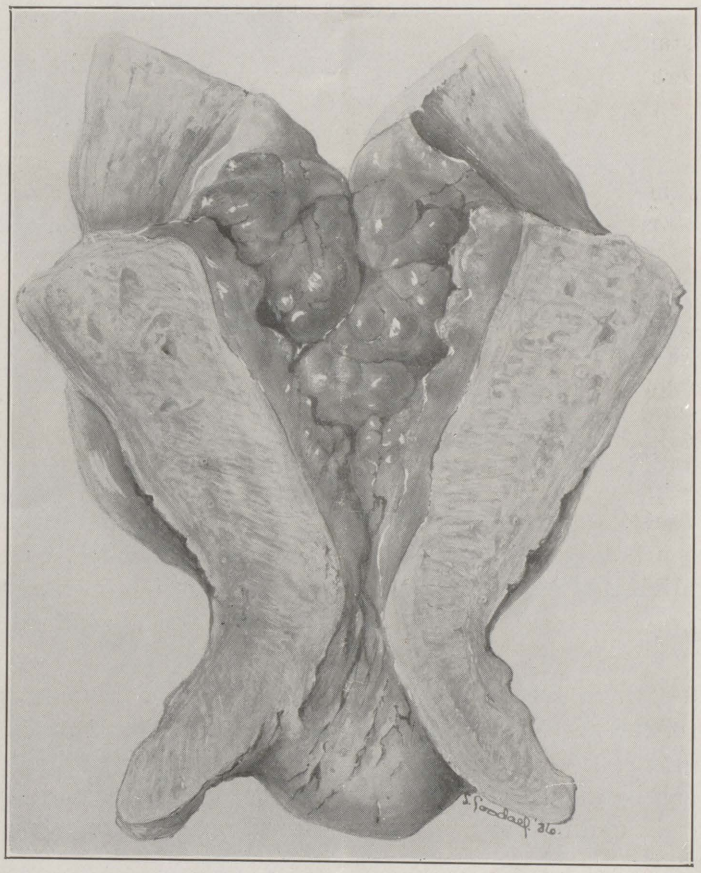

Fig. 1.—Showing swollen, bulbous mucosa and hypertrophy of muscularis.

The patient, Mrs. J., came complaining of menorrhagia. Her periods had always been prolonged, lasting, for two years after puberty, for seven days, and in the past two years lasting eleven days, during which time she was confined to bed, for the major part. The quantity was always copious. She had a constant watery leucorrhea in the interval. She is a very intelligent woman, with an extraordinarily clear marbled skin, and abnormally pale-blue sclerotics. Something of the Lorrain type, without necessarily being "petite." She had a fairly marked cystocele and rectocele and a lack of tone of all the tissues. This is emphasized, because we think it an expression of overrefinement. It is a singular fact, that numerous computations among school children have shown that allergic children, or children of

allergic parents, have a mental capacity far beyond their classmates. Her uterus was larger than normal, but symmetrical. Operation of repair and hysterectomy was advised. She was transfused and a wheal developed at the site of the puncture. No other untoward symptoms. The patient bled profusely during the repair, and when the abdomen was opened, there was a general edema of the pelvic organs, free fluid, etc., just as described above, and to Dr. Goodall's surprise, the uterus had all the characteristics of a two and one-half to three months' pregnancy. There was not the slightest doubt in his mind and in that of his assistant, that they were dealing with a pregnancy, though the patient had menstruated regularly, the last period

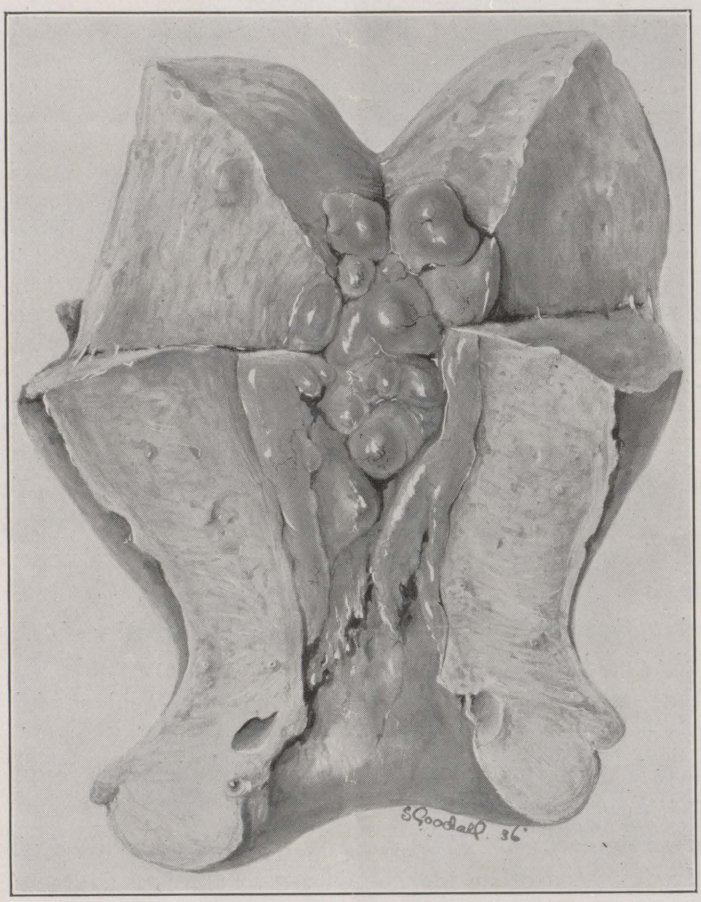

Fig. 2.—Mucosa and muscularis involved as in Fig. 1. The edematous mucosa hangs as a veil at the internal os. Cervix hypertrophied and edematous.

being ten days previous to the operation. Owing to her past history, which will be detailed below, it was decided to do a total hysterectomy. When this was completed, the uterus was opened. The muscularis was like that of a pregnancy, greatly hypertrophied, but there was no pregnancy. Instead, the uterine mucosa was from an inch thick at the fundus to lesser degrees toward the internal os (Fig. 1). It resembled, in color, the pulp of a grapefruit, and was thrown into folds or convolutions like the brain, a picture, in every detail, resembling the edematous nasal and antral mucosa, under the changes characteristic of hay fever (Fig. 2). The true nature of this condition suddenly dawned on us, and in retrospect the whole of the past cases fell in a clear perspective.

The retrospect of her history in the light of this knowledge now became interesting. She has a markedly allergic family history, of asthma, bronchitis and eczema, and one of her three children is allergic to egg albumen. She had frequent nosebleeds as a child, that caused her parents great alarm, and so severe on two occasions as to cause the family physician to remain all night. These nosebleeds ceased when menstruation came on, and had not recurred since. She had retroplacental hemorrhage with one child and bled extremely profusely at that, and her other deliveries, twice to the degree as to cause great alarm for her life. In a very smooth recovery on the fifth day after the operation detailed above, she was pulling up the sheet so as to smooth it out, when she barely touched her nose. Epistaxis set in, and it was so profuse that she had to have her nostrils plugged. This ceased, and three days later she developed vaginal bleeding, at first slight, later more copious, until her condition became critical. There was no fever, and no infection. She was taken to the operating room, the clots were removed, and the vagina packed gently, and the patient was again transfused. She again developed a wheal at the site of puncture. Her recovery was uninterrupted afterward. The drawing made immediately after operation will clearly illustrate hypertrophy (Fig. 1). Microscopically, there was great thickness of the mucosa, and a diffuse edema, so marked as to separate the connective tissue cells as if by artefact. But it is not of this nature, because it is universal, involving the whole thickness of the uterus, but decidedly more pronounced near the surface layers of the mucosa. The microscopic drawings beautifully show the different degrees in the various depths of the mucosal tissues.

There was in this case, owing to the chronic irritation occasioned by the allergin and the consequent extravasation of plasma, a chronic hypertrophy of all the tissues of the uterus and a hyperplasia added to the great thickness occasioned by the edema. The microscopic picture is strikingly like that of the antrum of Highmore under similar causal agents.

Knowing that cases of allergy are largely hypothyroids, and are frequently deficient in gastric HCl, these tests were made, and it was found that her basal rate was –15, and she was achlorhydric before and after a test meal. Her white cells showed a distinct shift to the right in a lympho- and monocytosis, all of which are frequent concomitants of, or causative agents in, allergy.

In the acute cases of pelvic allergy, the uterine mucosa alone may be involved, or the other uterine tissues to a lesser degree (Fig. 2). The major expression of the edema is always near the surface of the mucosa; the deeper tissues are more compact. In many instances one part of the mucosa is edematous, while another part is hemorrhagic. The mucosa is greatly thickened and may be associated with cyclic phases of menstruation. In many of these instances the condition of the cervix may give one a clue to the nature of the disease of the mucosa by a diffuse edema of the cervix, in which circumstances the cervix is swollen, usually one lip more than the other, semitransparent and boggy, signs characteristic of an advanced edema. Such cervices, if cauterized, do not heal kindly.

Many of these patients, like others similarly afflicted, only in systems other than the pelvis, are allergic only at the menstrual period. This is where the endocrine imbalance and allergy become associated. It is a well-known fact that most allergies are worse at the menstrual periods, and the tremendous increase of allergic cases in females, at and after puberty, only substantiates this association.

Allergics are notably hypothyroid and hypoovarian and the natural inference is that they sink a little lower in their endocrine imbalance at menstruation, and the constant subsoil of hereditary allergic hypersensitivity comes to the surface. Menstruation does cause an increased thyroid and ovarian demand, and if these cannot be responded to, the general tissue well-being is correspondingly lowered. In these instances of sporadic allergy, it is not necessary to treat the allergy; it is only necessary to submerge it by thickening the surface soil, by administration of thyroid, and if need be, ovarian opotherapy.

Chronic menorrhagia and metrorrhagia are frequently of allergic origin. Many of these cases have come under observation. The difference between the plasma extravasatory types and the hemorrhagic types is one of degree, rather than of difference. However, one finds three types which may fade imperceptibly one into the other. The first is the extreme plasma exudative type as described at some length above. The second is one which shows less edema and more diapedetic extravasation; and the third shows no edema, but a thin atrophic mucosa with a bright red extravasated surface. The differences parallel similar allergic states elsewhere in the body.

Let us outline one of the latter types. Goodall has had to revise his dicta upon the subject of hemorrhage in cases of that disease variously described as chronic metritis, chronic fibrosis uteri, and chronic subinvolution. This disease of the uterus usually precedes the onset of menorrhagia and metrorrhagia by many years. But suddenly we find these signs appearing without apparent reason. To arrest the hemorrhage, operative procedures or sterilizing methods have frequently to be resorted to, and Dr. William Fletcher Shaw once stated, "These procedures are expressions of defeat." There is, of course, a reason for the onset of hemorrhages, but in most cases of the chronic fibrosis and in many instances of fibroids, and in still more instances of total absence of local pathology, the cause is a general allergy. Many of these are spoken of as idiopathic, myopathic, and endocrine. The two first terms mean nothing. The latter may operate in a large number of cases, either alone or as an adjuvant, in exposing an allergic, heretofore dormant, predisposition.

Let us return to our case. Mrs. L., thirty-six years of age. Mother of three children. Has always had profuse menstruation. Three years ago developed metrorrhagia. Was treated in the radium institute with three series of deep x-ray of

14 sittings, 42 in all. This arrested menstruation for six months. Metrorrhagia set in again and she was subjected to 14 more sittings of deep x-ray, without any appreciable effect upon the blood loss. She then came to St. Mary's, a thin little woman, complaining of attacks of migraine and pain about the chest. There was a family history of allergy, and she had deprived herself of bread and all substances made of wheat for many months. On physical examination, the cervix was almost infantile, and the uterus was hyperinvoluted, and hard. Total hysterectomy was performed after transfusion. The uterine mucosa was blood red everywhere, and atrophic. Five days after operation she had two nosebleeds, copious, followed by several others in the next few days. Neither this patient nor the previous one showed any epistaxis while on reduced diet. We later told her to eat some bread. She shortly afterward became chilly, with cold extremities, and a cold perspiration and a great acceleration of the heart rate, and precordial pain. These symptoms were reproduced at will by the ingestion of bread, and the attacks of epistaxis were more or less synchronous.

Many of the cases of pelvic allergy become so, only on occasions when the general health is lowered by menstruation, worry, fatigue, exhaustion, after periods of mental or physical stress, and after any infection. Most of these are interpreted in terms of the conditions enumerated, and treatment directed to the underlying cause is not necessary. Hence the actual cause is seldom detected, one sees only the intermediate. Many patients become allergic in one or another system after operation, a condition that is seldom recognized, and therefore seldom properly interpreted.

Allergic dysmenorrhea is a fairly common condition. It falls into the group of the muscular spasms. Allergic dysmenorrhea is characterized by laborlike pains, but usually more continuous during the first hours or day of the flow. It has nothing characteristic to distinguish it from the ordinary type of dysmenorrhea in young girls. But it should be suspected in such cases, and an inquiry made into the family history for evidence of stigmas of allergy. One has always a means at hand to prove the etiology. By injecting from two to five minims of 1-1,000 solution of adrenaline, the case reacts promptly if of allergic origin. Unfortunately, the relief is only temporary, but the injection may be repeated in small doses. Another and very old-fashioned remedy, the true significance of which was perhaps never suspected, consists in giving a large dose of castor oil two or three days before the expected period, and then recommending that the patient remain on a milk, or greatly reduced diet, until menstruation is over, or at least well established. If in such cases the allergy was of alimentary origin, and the offending substance was eliminated, the patient was greatly improved. If the allergy was not of alimentary origin, or of some dietetic substance still included in the reduced dietary, then improvement did not follow. The usual explanation of the frequent improvement brought about by this treatment, was that it decongested, and was antiphlogistic.

Let us illustrate a few cases. A young girl, free from dysmenorrhea except periodically, and that rarely, went to work in a flower shop. From that time on her dysmenorrhea was severe and incapacitating. The relationship of this occupational cause to the sudden onset of the symptom having been established, she relinquished her position for another type of work, and was completely relieved.

Another patient suffered excruciatingly with menstrual dysmenorrhea. This symptom had come on after a rather severe infection in the nature of a crop of boils, which left her anemic and fatigued. Large doses of ammonium citrate of iron gave almost magical freedom from dysmenorrhea. Cases of this nature are very numerous.

A young girl of our circle of acquaintance developed severe dysmenorrhea after an operation for an acute appendicitis. The dysmenorrhea was always associated with bowel tenesmus and upset stomach. Previous to her appendicitis she was unconscious of any discomfort at menstruation. Later she developed a severe nasal infection and became definitely and periodically asthmatic. Her basal rate was found to be -12. When placed upon thyroid and iron tonics, her general condition improved and her dysmenorrhea and asthma became completely covered, after having been discovered for a period of about nine months.

Another case is of more than passing interest. The patient is so incapacitated by her menstrual dysmenorrhea that she will not go from home at that period, for fear of disturbing her entourage. She has been in the hands of many prominent gynecologists of this city, and treatment by Goodall proved equally futile. The girl was likely to become an alcoholic and morphine habitué, because she took gin in large quantities and morphine had to be exhibited several times to give relief, and stop her screams. She was given a hypo of adrenaline, and the relief was almost instantaneous, except for a residual pain following cramps. She is now under observation. Her thyroid is -22. She has no free hydrochloric acid. Her family history is replete with allergic roots, and hope is high that she will ultimately be cured by finding the allergen or allergens that are causing the cramps. Meanwhile she is on thyroid therapy, combined with dilute hydrochloric acid and pepsin.

Enough cases have been described to demonstrate the type and procedure adopted.

Leucorrhea of allergic origin is more difficult to establish convincingly. However, in a paper written two years ago by Dr. Goodall, which appears in the *Journal of Gynecology and Obstetrics of the British Empire* for October, 1936, 200 cases of mucous colitis are described, with all the symptoms arising out of referred pain in such cases. A very large percentage of spastic colon in mucous colitis are allergic in origin, and in that paper several nurses volunteered the information, that they had periodic attacks of leucorrhea and that these always synchronized with exacerbations of their colitic symptoms, and with improvement of the symptoms of colitis under rigid diet, the leucorrhea disappeared completely.

Another form of leucorrhea is met with in the next category of cases. Those of allergic vaginitis and intertriginous vulvitis. These patients complain most bitterly of pruritis vulvae, and irritating acrid discharge. On examination, an extravasatory intertrigo is found about the vulva, in the cleft of the buttock, in the anal orifice, and in the

folds of the inguinal area. The tissues in the more chronic cases, crack and expose the tender corium. Scratching often leads to infection and a complicating impetigo. In many instances, signs of kraurosis vulvae are already present. The vagina is reddened, pruritic and burning. It is edematous, the natural rugae are increased, the surfaces are desquamative, and the underlying surface looks raw and angry. In some cases, patients have of their own accord, inserted cotton in the vagina and vulva to separate the affected and contiguous surfaces. The family history is usually affirmative, and the patient may have had other signs of allergy in other parts of the body. Allergic vulvitis, due to glycosuria, falls into this category of allergic vulvitis and vaginitis. This condition is absolutely indistinguishable from the description given above. The popular idea among medical men that diabetic vulvitis and vaginitis is due to local decomposition of the glucose of the urine, is entirely wrong, for no matter how clean a woman, the symptoms persist, and some of the worst cases of diabetic vulvar allergy which we have seen have been in cases where sugar was never found in the urine, but a high blood sugar was present, the overflow into the urine being prevented by a high renal threshold. Reduction in the blood sugar content invariably leads to prompt relief. This is a vulvar allergy in which the allergen is glucose, which exists in such quantity in the blood that it sets up an allergin in the affected tissues.

In other instances, other allergens are the cause. In one instance, the patient at menopause developed an allergic eczema of the auditory canal and suffered frightfully from intolerable itching. She later developed prurigo, and still later an insufferable vaginitis and eczema of the vulva. Her sugar content was normal. She was a –12 rate, no free hydrochloric acid, but a distinct eosinophilia. Reductions of her diet to boiled skimmed milk, and the exhibition of thyroid and hydrochloric acid, brought about a rapid amelioration of symptoms. Later it was found that she had become allergic to potatoes, and when these were excluded from her diet her continued enjoyment of life was restored.

May we digress here, to consider a few relevant cases of allergy in gynecology, and incidentally these remarks must apply to men, though perhaps less frequently, or in a mitigated form. In our wards at present is an elderly single woman who came in exhibiting great pallor, orthopnea, general anasarca, uterine hemorrhage, and severe dyspepsia and precordial pain. Headaches were severe. She had lost all the hair of the scalp and of the normally hirsute areas. She at once impressed one as being in the last steps of Bright's disease. However, on examination, the heart was negative, and the lungs showed some rhonchi and râles. The urine was absolutely normal, as was also the blood chemistry. There was an advanced degree of anemia. Her basal rate was minus 12. She was transfused and given iron. She improved rapidly. The edema and respiratory distress disappeared completely, and

her headaches gradually were alleviated. We then undertook to do a hysterectomy for fibroids. She went through the operation perfectly and was remarkably well for five days, when she developed a left leg thrombosis and temperature. Almost at once she became allergic again, with inability to take food; precordial pain, return of the headaches and a feeling of weakness, and generally wretched. She improved promptly again under a 10 per cent glucose intravenous injection and twenty-four hours later, another blood transfusion. This case illustrates beautifully the double onset of allergy under first, a condition of lowered vitality, and later, after a post-operative infection. These cases are extremely numerous, and we are convinced, after a careful examination of the fibroids, that the uterine bleeding in this case was allergic, and did not proceed from the growths. The fibroids and their surfaces were almost unaffected, but the rest of the uterine mucosa was hemorrhagic and infiltrated with eosinophiles. Another patient just discharged from the wards of St. Mary's affords a clear vision of another common gynecologic association of allergy and colitis, with referred symptoms.

Mrs. L., an elderly lady of sixty-five years, but of much older appearance, came into the ward complaining of precordial pain, epigastric pain, cold, numb feet, and headache. But her chief complaint was pain in both lower quadrants. The pelvis was negative. X-ray of gallbladder and barium series were negative. The diagnosis of acute exacerbation of a chronic colitis was suspected from the first, later confirmed. It was also suspected that the precordial pain and headache and numbness of extremities were allergic. She improved tremendously under rest and treatment. But in a sudden bout of exaggerated colitis, she developed acute precordial pain, simulating angina, followed by a severe attack of allergic asthma. Her condition was most distressing. The house surgeon, Dr. Macphail, aware of our diagnosis, immediately gave her injection of adrenaline, with prompt relief.

It has been our experience that many of the cases of weakness, fatigue, and lassitude which come periodically over women who, at other times, are inordinately filled with energy, are due to temporary allergic states brought about by exhaustion or other contributory factors. And sudden changes of disposition are attributable to similar causes. We feel certain that many who bear the stigma of neurasthenics fall into this category. In fact, it is well recognized that allergic individuals change their character completely when under the influence of an allergen.

Apropos this subject. Mrs. H. was in one of the hospitals of this city some four years ago, suffering from what was considered cardiac asthma. She was under the observation of some of the best cardiologists for a period of six weeks, without any appreciable improvement. She was then advised to go to Atlantic City, which she did, and promptly grew worse. She was brought back to Montreal in an ambulance, and it was thought futile to seek further treatment. However, uterine hemorrhage set in, Dr. Goodall was called. She had passed her menopause. Patient was sitting up in bed, grasping the sides to fix her chest. Her chest was overexpanded, she was markedly cyanosed. A most pitiful picture. Pelvis negative. However, examination of the abdomen revealed a huge colon, the size of my upper arm. The bowels had been moving daily. Dr. Campbell P. Howard was called in consultation. Castor oil in large quantities was administered, and high colonic lavage instituted. The amount of fetid material that was brought away was incredible. The asthma was promptly relieved, and the patient has been able to play golf and drive

her car without recurrence, for the last four years. Recently she began to slip again. She is now becoming so cranky and so neurasthenic that she is almost impossible of association. This is but another manifestation of her allergic state, the true nature of which it is impossible to determine, and being so chronic, would probably not respond to any form of treatment. Moreover, her advancing years makes rehabilitation more difficult, so that the outlook is poor. She is looked upon by all who contact her as one of the worst of neurasthenics, whereas she is but a poor allergic, with a very bad family tree rooting in a bad subsoil.

These cases deserve our most inquiring, unexpressed sympathy and study. Where in such cases the allergic sensitivity becomes active, owing to general ill-health and advancing years, the outlook is far from encouraging, and will tax our ingenuity to its limits. Most of us surgeons of experience find hours too filled to give these cases the study they require, and they end up with a loss of faith in the profession usually.

From a gynecologic and obstetric interest we cannot refrain from detailing two of the most interesting cases.

The first is a woman, thirty-four years of age, upon whom Dr. Goodall performed a cesarean section three times. She has a bad family history of allergic sensitivity, and she, herself, has suffered from migraine for years. Migraine, it is well known, is due to an allergic edema of the brain. She is allergic to certain kinds of fish, and one of her children is allergic to milk. When she was five months pregnant, she ate some shellfish at her evening meal, and shortly afterward, she had the "migrins." That night, vomiting set in, and the next morning castor oil was administered, upon the assumption that it was a case of ptomaine. That afternoon her headache increased; she became stuporose, facial paresis and paresis of the upper extremities set in. By evening she was completely comatose, and propulsive vomiting, widespread paresis, and changes in the optic discs had developed, all indications of intracranial pressure. Doctors Goodall, Scriver and Cone, gynecologist, internist and neurologist respectively, concluded that this was a case of choroidal hemorrhage, probably associated with toxicity of pregnancy, on the assumption that all pregnant women are toxic, though there were no signs of toxicity or diabetes. Her spinal fluid was under high pressure but was free from blood. It was decided that it was advisable to interrupt the pregnancy. This was done by cesarean section. The case was very hemorrhagic at operation. She promptly improved, was lucid in twenty-four hours, and nonparetic in forty-eight. The inference was, of course, that the condition was due to the pregnancy. Its true significance did not develop until three months later, when after a meal of mackerel, she developed the same symptoms, paresis, vomiting, diplopia, and screaming headache. She was immediately taken into the hospital, given a gastric and colonic lavage and 3 minims of adrenaline. The symptoms subsided as if by magic, and recovery was complete. This was undoubtedly a case of allergy of the brain, not a choroidal hemorrhage, but a choroidal edema. Hence its rapid disappearance under treatment. Let us repeat, there is no system in the body that is exempt from the effects of allergins. It is intensely selective in its incidence, though not infrequently it is composite in the one individual by attacking two or more systems simultaneously, or in sequence.

One other case: A young woman had suffered a severe toxemia of pregnancy, complicated by numerous convulsions. The baby was lost in delivery, and the mother's puerperal recovery was slow and eventful. She was sent home in the ambulance, three weeks postpartum, when she promptly developed a most severe

retrosternal pain, with a degree of temperature. Low grades of temperature are not uncommon in allergic patients. Goodall was called in consultation, and after examination, pulmonary infarct was suspected, but the picture did not fit the diagnosis. However, *faute de mieux,* it was maintained. Patient had several of these attacks, all similar, without any objective signs. She took very little food, vomited a great deal, felt that food made her worse, and brought on the attacks, so that by abstention she was reduced to a state of severe malnutrition. Suddenly something clicked (in the surgeon's brain, we mean). It is allergy. She was sent to another hospital, the basal metabolic rate was found to be -18, no free hydrochloric acid in the stomach, and a grave hypochromic anemia was detected. With her next spasm she was given 3 minims of adrenaline, with immediate results. She was then put on thyroid, with pepsin and hydrochloric acid, and her symptoms promptly subsided and her nutrition was quickly restored. But perhaps the greatest change was in her mental state. From a depressed, apathetic, hypochondriac, she soon recovered her pristine buoyancy and *joie de vivre.*

This is another instance of the importance of raising the general health to overcome a specific cause. To those who do a great deal of gynecologic surgery, the composite picture of pelvic allergy, complicated by pelvic inflammatory disease, will readily recur. Of course, one does not operate upon pelvic inflammatory disease nowadays except in occasional cases. When these two diseases co-exist, we find a diffuse inflammatory pelvis, over which large and small lakes of clear yellow subperitoneal or subadhesional lymph exist. The lymphatics of the pelvis are widely dilated, and many of them have a clear coagulum in them. The blood vessels of the broad ligaments are engorged, and adhesions, when broken through, are inordinately oozy. The lymph collections may vary in size from millet seeds to that of an egg. The uterus is large and soft, and not infrequently the outer third of its thickness is under a white coagulation of all its tissues. The ovaries are edematous and usually, though not constantly, filled with small cystic follicles in which the egg may or may not have surrendered.

We believe that many conditions at present not generally attributed to allergy are allergic basically, such as pseudoanginal attacks, precordial pain, many cases of colitis, acidosis, sudden death in children and adults without pathologic findings, for the conditions of edema and spasm, the chief expressions of allergy, are unrecognizable at autopsy, or are looked upon as postmortem fluid gravitation. The periodic overabsorption of unsplit proteins may depend upon many extraneous causes enumerated above, so that the normal neutralizers in the blood are used up and the excess of allergens evoke the allergins of sensitized cells, cardiac or other. We would include many instances of neurasthenia with headache radiating down the back of the neck. We think also that those cases of spasm of the common or cystic bile duct, simulating cholecystitis or gallstones, are of this nature, as are also the muscular spasms of pregnancy. It is intriguing also, to think that erythroblastosis in the infant may be hereditary from one or both parents, or due to placental transfusion from the mother. There is

much to substantiate this. We would include from observation, many cases of lassitude and fatigue, and general feeling of malaise, frequently associated with general skin irritation after the day's work, such that clothes must be removed. These, we think, are temporary allergies due to prolonged effort, mental or physical, in which specific treatment, owing to its composite nature, is of little avail, but change of environment and surcease from mental bombardment and physical effort will recover the subsoil and restore equilibrium.

Treatment in the acute cases is often surprisingly gratifying, often a complete cure. In the chronic cases, it is often not even a mitigant. Nevertheless, the satisfaction to the physician and surgeon is the knowledge that he is dealing with a clinical entity. Unfortunately, it is an entity which, in most cases, is hidden from the pathologist.

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ARRESTED DEVELOPMENT OF THE RECTUM*

(Report of a Case and Review of the Embryology and Anatomy Concerned)

By E. A. Daniels, M.Sc., M.D.

Montreal

ARRESTED development of the rectum is an extremely rare congenital defect. Such an anomaly, according to Starr (quoted by Lockhart-Mummery[1]), is found only in a very small percentage of all births, and he states that it may be observed approximately only once in every ten thousand deliveries. It is reasonable to suppose however, that this figure is probably too high, since such cases are very rarely encountered by active hospitals and clinics. The author has recently observed and operated upon a newborn infant with such a congenital defect. The rarity of this lesion should make an anomaly of this type of sufficient interest to report. Although rare it is nevertheless necessary to understand the fundamentals of such congenital defects. Such a condition presents itself as a rule as a surgical emergency, and one must be prepared to carry out an appropriate plan of treatment. The danger to the child's life is imminent, and surgical intervention in a newborn infant is a grave undertaking. A knowledge of the evolution of the development of the terminal bowel and its anatomy in the newborn therefore becomes imperative.

Embryology and Anatomy Concerned

In the early stages of development in the human embryo the allantois communicates and is continuous with the hind-gut, and anteriorly enters into the formation of the body-stalk. A U-shaped angulation is thus formed at the point where the allantois joins with the hind-gut. The apex of this angulation becomes increasingly dilated downwards to form a pouch situated at the posterior end of the embryo, known as the post-allantoic gut. This is really a process of downward prolongation and sacculation of the hind-gut into which opens the allantois in front. The downward prolongation of the hind-gut (post-allantoic gut) continues to grow downwards and backwards past its anterior or cloacal opening, continuous with the growth of the hind portion of the body of the fetus. The communication between the hind-gut and the allantois in front soon becomes sealed off in the process of development. At a later stage the hind-gut and its posterior sacculation, already described, are completely separated from the allantois in front and are thus without any communication with the body or allantoic stalk. In the male the bladder, prostate and urethra are developed at the lower portion of the allantois, whereas the Müllerian ducts are developed from the upper portion of this structure. In the female the uterus and vestibule of the vagina are developed between the hind-gut and the allantois. Fig. 1 shows the above in diagrammatic fashion. It will be noted that the communication between the hind-gut and the allantois ultimately becomes a firm band of tissue, referred to as the obliterated communication between the hind-gut and allantois (Fig. 1). Should this communication fail to close, that is, when persistent, this occurs at a definite point as follows: in the male, at the prostatic urethra; and in the female at the posterior vaginal wall just below the cervix. Lockhart-Mummery states that the site of this communication is thus constant at the two above-mentioned points in male or female because of the constancy of the embryological picture. He points out that this does not follow any haphazard scheme, and for this reason a communication between the hind-gut and allantois is generally into the prostatic urethra in the male and at the top of the posterior vaginal wall in the female. In arrested development of the rectum the post-allantoic gut, that is, the posterior prolongation of the hind-gut, is entirely missing. The point of junction of the hind-gut and the post-allantoic gut occurs at the point of the reflection of the peritoneum from the anterior surface of the rectum. Consequently, in cases of imperforate rectum the blind termination of the hind-gut will be found opposite the recto-vesical pouch of peritoneum in the male and the recto-vaginal pouch of peritoneum in the female. Such were

* From the Woman's General Hospital, Montreal.

the findings in the anomaly to be presently reported.

The next step in the development of the ano-rectum is as follows. A slight depression or dimple appears on the body wall between the coccyx and the mid-point of the perineum, at a point parallel with the long axis of the rectum (post-allantoic gut). This is known as the proctodeum, which is simply an ectodermal depression in the area which will go to form the future anus. This is shown in the illustration. A membrane is thus formed, known as the proctodeal membrane, which soon breaks down or is absorbed, forming a communication with the post-allantoic gut. It can be thus seen that the rectum and anus are developed from three distinct structures: (*a*) the hind-gut; (*b*) the sacculation developed by the downward prolongation of the hind-gut, already referred to as the post-allantoic gut, and (*c*) the proctodeal depression. The point of junction between the proctodeum and the hind-gut, that is, the point where the proctodeal membrane has broken down, is seen in the adult rectum as the dentate or pectinate line. This dentate or pectinate line marks the point of junction between the squamous epithelium of the body wall and the columnar epithelium of the hind-gut.

One can visualize the type of congenital defect that might be observed if the above steps in the evolution of the development of the ano-rectum are understood, namely: (1) Failure of the downward development of the post-allantoic gut. The blind pouch marking the termination of the hind-gut is therefore found opposite the recto-vesical or recto-vaginal peritoneal reflection: (*a*) without a fistulous communication to the prostatic urethra in the male or posterior vagina in the female; (*b*) with a fistulous communication to the prostatic urethra or posterior vagina. The defect to be presently described belonged to 1(*a*). (2) Defects resulting from imperfect obliteration of the proctodeal membrane. Such a defect will therefore be found low-down at the dentate line.

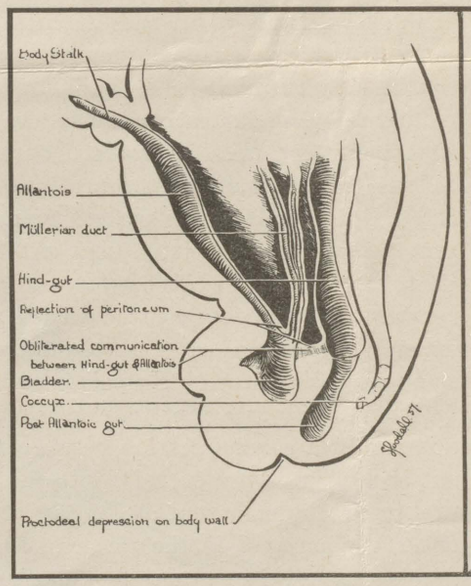

Fig. 1

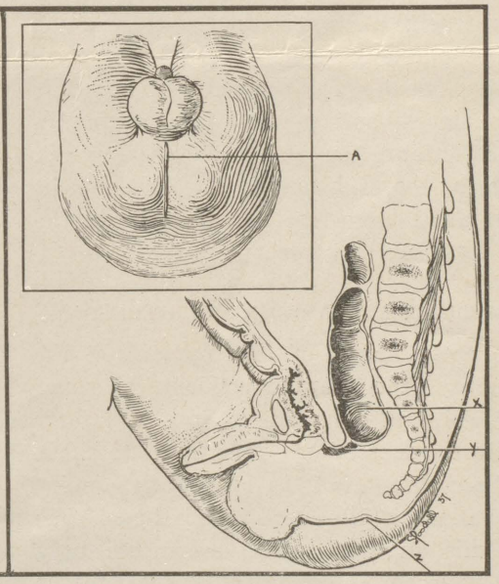

Fig. 2

Fig. 1.—The above illustrates the various steps in the evolution of the development of the rectum. It is seen that the future rectum is developed as a downward prolongation from the hind-gut, known as the post-allantoic gut.

Fig. 2.—The inset shows the line of incision from just behind the scrotum to the coccyx.

X.—In the large illustration, demonstrates where the hind-gut had become arrested in its development, opposite the recto-vesical pouch of peritoneum. The post-allantoic gut is absent.

Y.—Demonstrates the obliterated communication between the prostatic urethra and bladder (allantois), and hind-gut.

Z.—Represents the area where the proctodeal dimple was present, surrounded by sphincteric tissue. This was the site of the anal canal and was used for this purpose at operation.

TREATMENT

One is dealing with a newborn infant in the presence of a surgical emergency. It is wise to wait for several hours in order that the child may recover from the trauma incident to its passage through the birth canal. It is reasonable to suppose that this period should not exceed twelve hours, since there is no outlet for meconium.

The pre-operative care consists of warmth and 5 to 10 per cent saline glucose given interstitially in quantities of 25 to 50 c.c. every hour or two until the time of operation. It is quite difficult to administer any of the usual anæsthetics. The author in his case employed a pledget of gauze dipped in brandy and given to the child by mouth to suck. This was found quite adequate. In female children arrested development of the rectum is very often accompanied by a fistulous communication between the hind-gut and the posterior vaginal wall which provides an outlet for meconium and fæces. This defect is therefore not an emergency, and when such a communication is found it is wise not to disturb the infant until the age of ten years or more is reached. At that time a modified perineorrhaphy, closure of the fistula, and implantation of the blind end of the hind-gut into the proctodeal area can be done. In male children born with absence of the rectum in which there is a fistulous communication between the hind-gut and the prostatic urethra, then the condition becomes incompatible with life, firstly, because such an opening is not sufficient for the free passage of meconium and fæces, and, secondly, because of the imminent danger of urinary infection. Such a defect must be treated as a surgical emergency by both closure of the fistulous tract, and by providing an outlet for meconium and fæces.

Whenever possible the blind end of the hind-gut should be found and brought down to the proctodeal area before opening the gut. Lockhart-Mummery and other workers in this field believe that it should never or very seldom be necessary to perform a colostomy; and that in nearly all instances it should be possible to find the blind end of the hind-gut at the point of its arrested development, and by carefully practised surgery to bring this down comfortably and implant it into the proctodeal area. A long operation should not be indulged in, as this endangers the child's life. The rarity of the lesion does not afford the surgeon a very wide experience in dealing with these cases. It is therefore necessary to have a knowledge of certain landmarks which will be consistently found in the anomaly. One must also be guided by the experience of other workers in the field who have had enough cases of this kind to enable them to outline a general plan of treatment.

The perineum should be opened by a mid-line incision extending through the blind proctodeal dimple from just behind the scrotum or fourchette to the coccyx. The dissection must be kept as much as possible in the hollow of the sacrum, as otherwise there is considerable danger of wounding the genito-urinary organs. Blind probing through a perineal stab in the hope of finding the bowel pouch is regarded as a dangerous method of treatment. The peritoneum need not be entered if dissection is confined to the hollow of the sacrum. As already pointed out by Lockhart-Mummery, the blind end of the hind-gut will nearly always be found opposite the recto-vesical or recto-vaginal pouch of peritoneum. One can identify this by first encountering the obliterated communication between the hind-gut and allantois as a heavy band of tissue running forwards. This may be divided to facilitate the dissection and to determine whether it contains a fistulous tract. Forcing the child to strain or cry will enable the operator to actually palpate the bulging termination of the gut and its distended meconium content. The cellular tissue all around this blind pouch should be freed and effective mobilization of the hind-gut carried out. The proctodeal area, as already pointed out, develops quite distinctly from the hind-gut as a depression on the body wall, and will as a rule be found to contain good muscular tissue. This was found in the author's case. After freeing the hind-gut it is brought down without tension to the proctodeal area and opened. The divided bowel lumen is then sutured all around with fine linen thread to what appears as the anal area. It must be remembered that the anal orifice in the infant lies almost in a straight line parallel with the tip of the coccyx,[2] that is, more posterior than in the adult. This should guide one in determining the area of the future anus. During the operation the child

should be surrounded by warm blankets and shock thus minimized. Post-operative care consists of warmth, fluids and glucose interstitially, and a full quota of breast milk by mouth. The pædiatrician's advice should be available at all times.

CASE REPORT

A primipara, aged 23, in normal health. Pre-natal course uneventful. Pelvic measurements normal; labour normal. The baby was born at midnight, April 26, 1937. Birth weight 5 pounds, 6 ounces. The nurse first noticed that the rectal temperature could not be taken because of the absence of an anal orifice. This was brought to the attention of the attending obstetrician. The following morning at ten o'clock the author examined the child. There was a very small depression or dimple in the region where the orifice should have been. This was surrounded by a small amount of pigment. A perineal bulge could not be elicited by pressure on the abdomen or by forcing the child to cry. It was then decided that the blind end of the hind-gut had become arrested high up in the pelvis.

The infant was prepared for operation, and this was carried out at eleven o'clock—11 hours after birth. Brandy was administered as an anæsthetic and the infant was surrounded with hot water bottles. The lithotomy position was employed. The incision was made from a point just behind the scrotum to the coccyx, as shown in diagram A. The dissection was continued posteriorly and trauma to the prostatic urethra, seminal vesicles, bladder and rectovesical pouch of the peritoneum was carefully avoided. The obliterated communication between the allantois and hind-gut, as shown in diagram Y, was encountered just below the recto-vesical pouch of peritoneum. No fistulous communication existed and this band of tissue was divided. The blind end of the hind-gut was found, as shown in the diagram X, opposite the peritoneal reflection and in the hollow of the sacrum. The gut was freed anteriorly without disturbing the peritoneum, and was dissected free posteriorly and laterally. Mobilization of the segment of the bowel was effectively carried out so that it could be easily brought down without tension to the proctodeal area, shown as Z in the diagram. The blind end of the hind-gut was thus brought down to the proctodeal area through which ran the skin incision as shown in the inset A. This was anchored by a stay suture of fine linen and the blind pouch incised. Meconium at once freely exuded. The mucosal periphery of the opened hind-gut was carefully sutured all around the area which was regarded as the anal site, Z. The skin incision was then closed without drainage by deep through and through discontinuous sutures of fine linen.

The child was then returned to his crib surrounded by hot water bottles. Five per cent saline glucose was administered interstitially at intervals, and feedings of small quantities of breast milk commenced at once. (The mother could not nurse, but breast milk was secured). The post-operative course was uneventful. The sutures were removed on the tenth day, the wound healed without infection, and the bowel movements were satisfactory and bulky. The stools were both formed and mushy, and there appeared to be good sphincteric control. The infant left the hospital on May 21, 1937, weighing 6 pounds, 14 ounces, and appeared to be in robust condition. Weight November 2, 1937, was 14 pounds 6 ounces. Examination revealed good sphincteric control. The anal orifice easily admitted a No. 22 F. catheter. The child was on a normal diet and appeared robust and well.

There is no reason to suppose that the child's future will be seriously affected by this congenital anomaly, since the operative procedure appeared to have been successfully carried out. At operation sphincteric tissue was quite discernible in the area of the proctodeal dimple. This was carefully sutured all around to the mucous membrane.

SUMMARY

Arrested development of the rectum when encountered in a newborn infant is a surgical emergency, which should be dealt with in a definite manner. Although a formidable surgical procedure, it becomes necessary to carry it out in order to preserve the child's life. Such interference can give a happy result if performed at an early period and under proper conditions.

The author wishes to express his thanks to Dr. F. E. Thompson, for permitting him to take charge of this patient and for his kind collaboration; to Dr. M. Scherzer, the attending pædiatrician, for his conscientious care and interest. The writer is also very grateful to Dr. J. R. Fraser, of the Department of Obstetrics, Royal Victoria Hospital, whose department was good enough to supply us with a liberal quantity of breast milk. This in no small measure contributed to the successful outcome of the case. Thanks are due to Miss Shirley Goodall for the excellent illustrations.

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Personal Experiences with the Manchester Operation

A. B. NASH, M.D.
VICTORIA, BRITISH COLUMBIA

THE history and underlying principles of the Manchester operation for the relief of genital prolapse have been frequently and adequately described and are all too well known to all of you to require any recapitulation on my part. On the other hand, a careful analysis of the difficulties, failures and successes of any operator with a reasonable experience in the application of the method are, I think, likely to be of interest and perhaps of value to others interested in the relief of this most common and distressing gynecological complaint.

In attempting to assess my own experiences with this procedure, I have carefully surveyed the results of 50 cases from my private practice, whose selection has been based solely upon my ability to pursue a careful follow-up, the shortest period of observation in the series being six months, the longest 84 months and the average of the whole, 33 months. The average patient's age at the time of operation was 53.5 years, the youngest patient being 29 years and the eldest 73 years.

Whereas the fundamental principles underlying this operation permit of no individual modification, the methods of applying those principles and of fulfilling their requirements undoubtedly undergo considerable variation in detail in the hands of various operators, some being, I have no doubt, for various and sundry reasons, more successfully and easily applicable than others.

For my own particular technic, I make, of course, no claim to originality. It represents a composite of the technic of many experienced and skilled operators whom I have observed and some of whom I have assisted. Numbered among these are Fletcher Shaw of Manchester and Chipman and Fraser of McGill, in whose Clinic the same principles have been employed continuously for, I think, as long, if not longer, than elsewhere on this continent.

Before proceeding to a description of operative technic, I wish to emphasize the paramount importance of careful preoperative preparation. Without reference to detail, the following principles are observed: (1) Marked cervicitis, especially when polypi are present, is first treated by cauterization and removal of polypi, at least six weeks elapsing thereafter before operation is undertaken. (2) Ulceration of the cervix accompanying complete procidentia is healed by bed rest and antiseptic tamponade. (3) When atrophic vaginitis is recognized, it is first overcome by local estrogenic therapy. (4) Urinary tract infection is treated until cured, the criterion of such being the absence of pyuria in at least four weekly cath-

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eter specimens. (5) A glucose tolerance test is performed to exclude the presence of actual or potential diabetes. (6) The usual precautions are observed to exclude general physical contraindication to any operative procedure and to insure an adequate preoperative intake of fluid and glucose. (7) Forty-eight hours of preoperative rest in bed is insisted upon.

Whereas the majority of the cases in this series have been performed under light cyclopropane anesthesia, both low spinal and local anesthesia are favored, the common objection thereto being only on the part of the patient.

Actual operative procedure is depicted in the accompanying illustrations which I will describe.

OPERATION

A preliminary dilatation of the cervix and curettage of the uterus are performed as a routine.

As illustrated in Figure 1, the labia minora have been retracted by suture to the skin of the thighs. The cervix is pulled down and out as far as possible with a double-toothed tenaculum. An elliptical transverse incision, with concavity upwards, is made on the anterior surface of the cervix, just below the point of reflection of the cervical and vaginal mucous membranes. This is actually carried further in each lateral direction than here depicted—out to the so-called "Fothergill's points," indicated in the drawing by forceps.

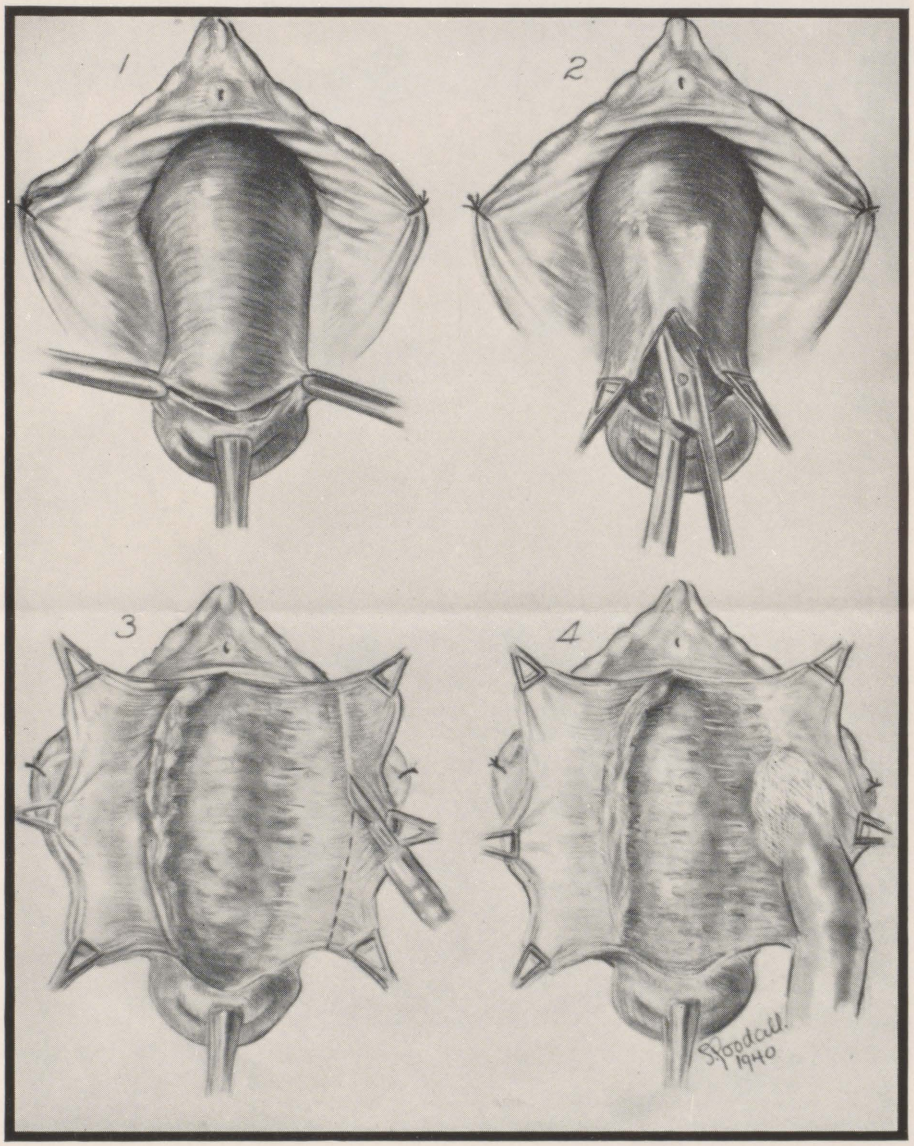
In Figure 2, a pair of curved scissors is introduced beneath the anterior vaginal mucous membrane as shown and, in this manner, the vaginal wall is dissected in the midline, from the pubocervical fascia and bladder. The extent to which this is carried forward is dependent upon the extent of the prolapse affecting the anterior vaginal wall. As this dissection progresses, triangular forceps are applied to the cut edges of the vaginal wall, and the continuing dissection is rendered easier by downward traction upon these forceps.

As illustrated in Figures 3 and 4, after incision of the dissected vaginal wall in the midline, the attached pubocervical fascia is freed laterally from each side by sharp dissection with a No. 15 Bard-Parker eye blade, and by gauze dissection to an extent determined by the redundancy of the vaginal wall.

Figure 5 illustrates how the bladder attachment to the cervix is freed in the midline by a few snips with the scissors, and the bladder is then pushed upward and forward by gauze dissection. As a rule, this is carried practically as far as the vesico-uterine pouch. Particular care is taken to free the bladder from its lateral attachments to the cervix and parametrium.

As shown in Figures 6 and 7, interrupted sutures of No. 1 chromic catgut are now inserted in the pubocervical fascia on each side as far laterally as is possible, without producing undue tension, and uniting the torn edges of the fascia in the midline. In cases with stress incontinence, this oppor-

Manchester Operation—Nash



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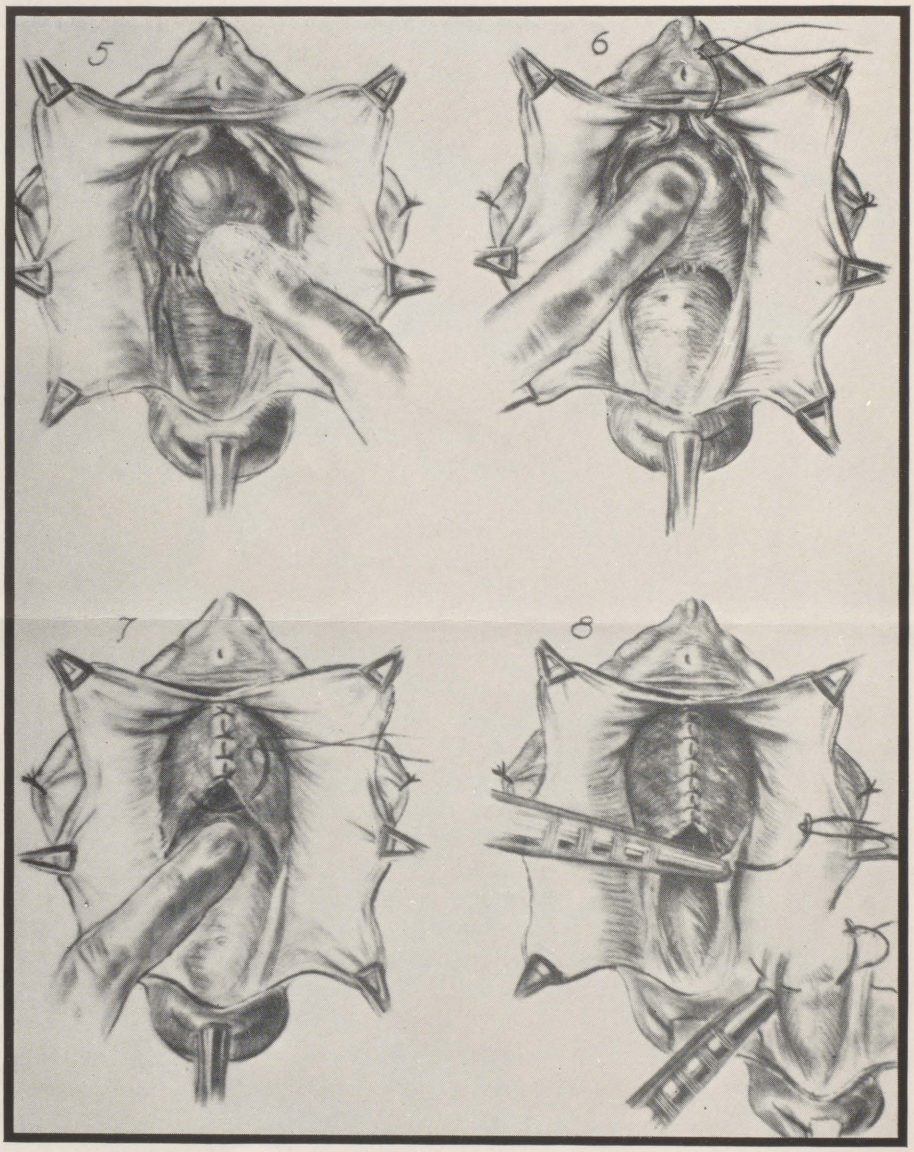

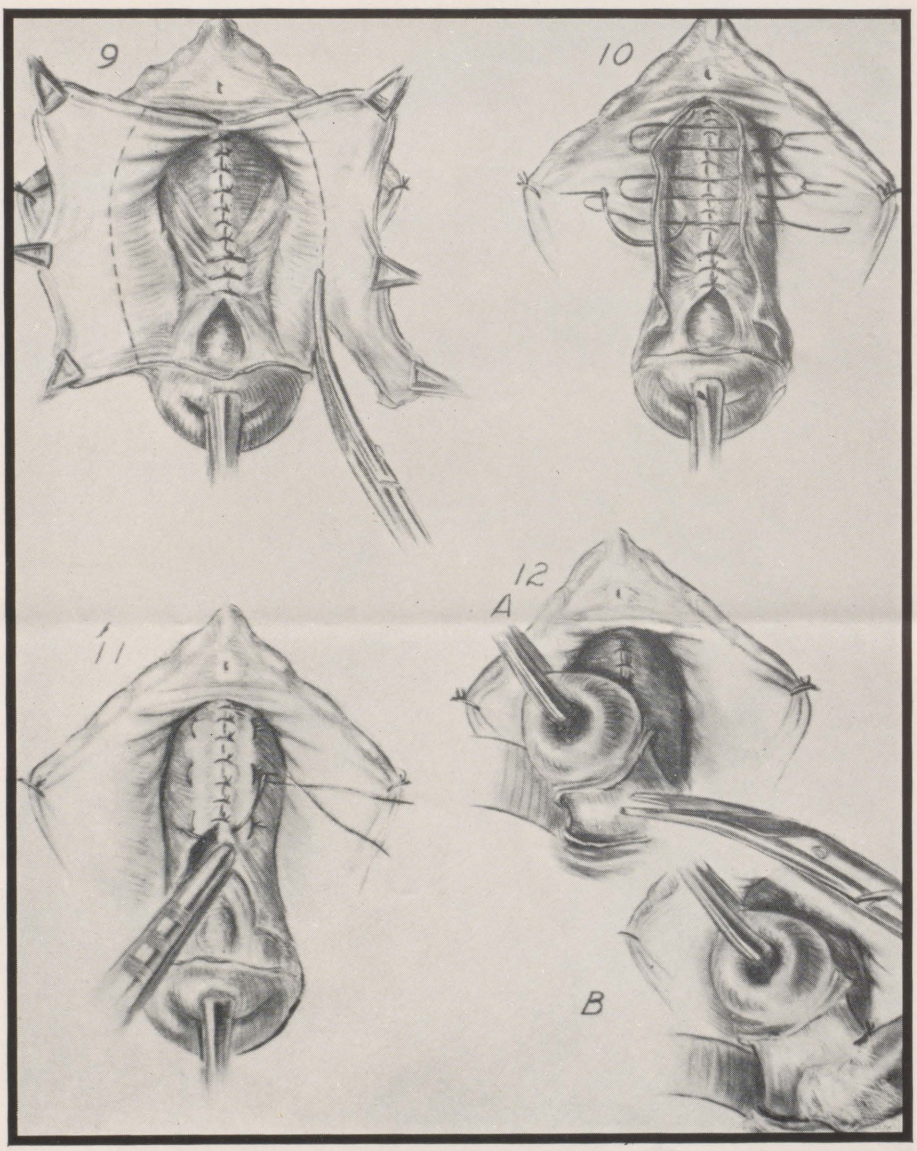
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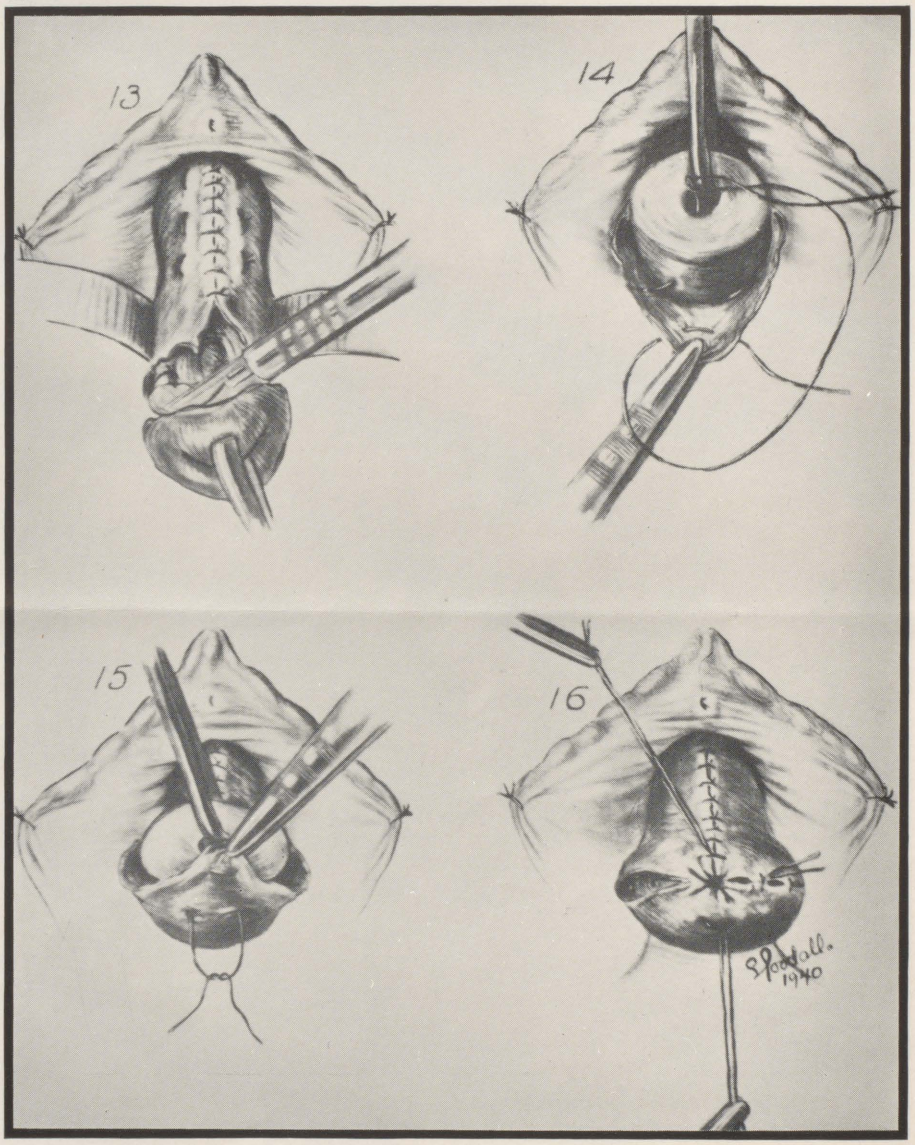
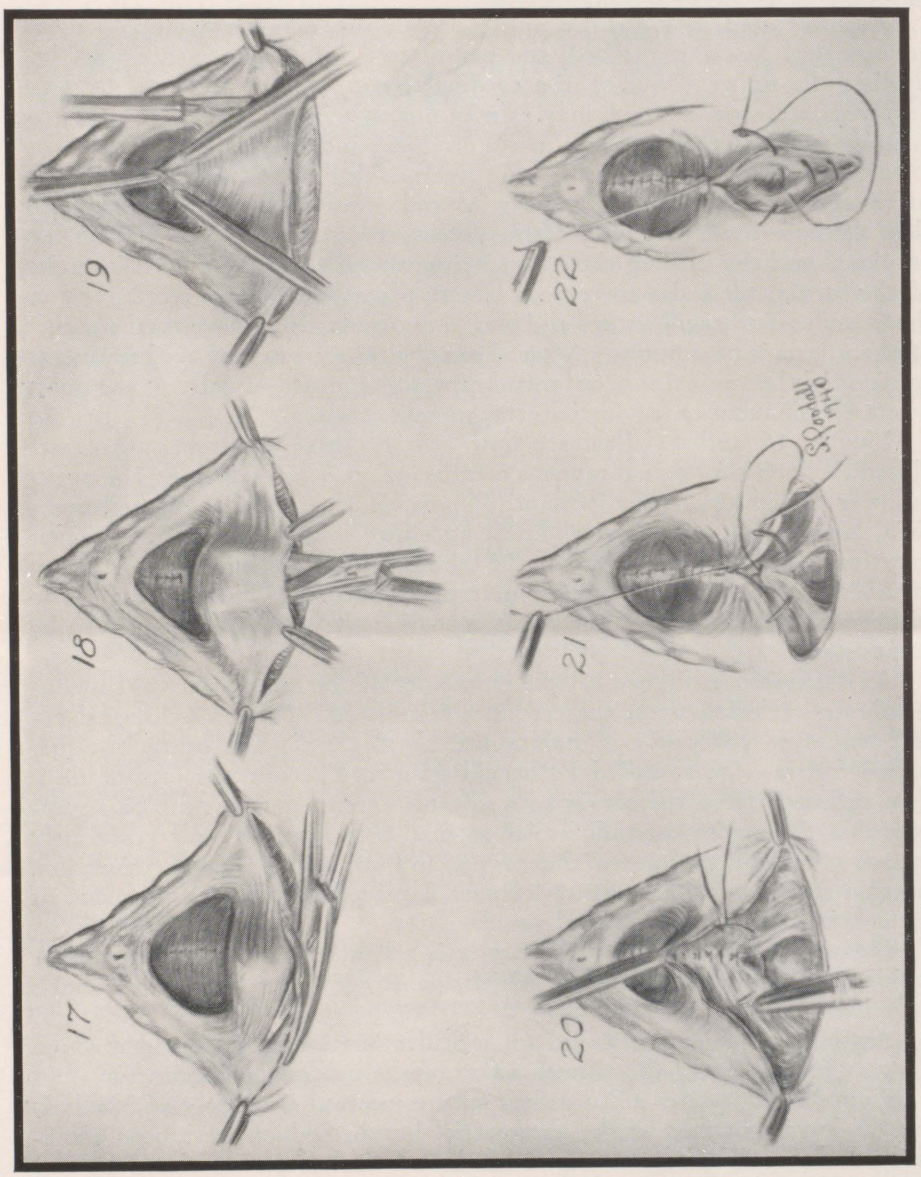
Manchester Operation—Nash





Figures 17, 18, 19, 20, 21, 22

tunity is taken to reinforce the bladder sphincter mechanism by carefully overfolding the floor of the urethra.

As illustrated in Figures 8 and 9, when the lowest extremity of the pubocervical fascia is reached, the parametrial bundle on either side, including the upper fibres of the cardinal ligament, is picked up by two deeply passed sutures which may or may not, as wished, include the vaginal mucous membrane and which in the midline, includes a bite, first in the lower or posterior extremity of the pubocervical fascia and then in the supravaginal cervix, before passing to the other side. These effectively close the lowermost extremity of the defect, through which the bladder has herniated and, by pulling the parametrium on either side forward in front of the cervix, tend also to correct retrodisplacement. They correspond to the so-called Fothergill suture and their importance in the eventual support of the uterus is paramount. After these sutures are inserted the redundant portions of the vaginal mucous membrane are trimmed, usually to the point where the denudation of fascial attachments ceases.

Figures 10 and 11 illustrate how two or three mattress sutures are placed through the vaginal mucous membrane on either side from the upper to lower extremities of the incision. If possible, these should include superficial bites in the pubocervical fascia on either side of the midline. Their insertion is followed by suture of the trimmed edges of the vaginal mucous membrane and, for this, I prefer interrupted sutures. These are carried to within a short distance of the point at which the cervical portio is to be amputated.

As illustrated in Figure 12, the remainder of the incision of the mucous membrane reflected upon the cervix is completed. The semi-elliptical effect employed anteriorly is maintained, the highest point being in either lateral fornix—the so-called Fothergill points. The mucous membrane is then reflected from the cervix to a suitable extent, maintaining to an appreciable degree its musculofascial attachments laterally and posteriorly by scissor clipping close to the cervix and subsequent gauze dissection. When this is complete a suture ligature is placed deeply on either side of the cervix to ligate the cervical vessels.

Figure 13 illustrates how the cervix is amputated by simple transverse incision, just below the level at which, on either side, the cervical vessels have been ligated.

As shown in Figures 14 and 15, a Sturmdorf suture is placed through the cervical canal and the posterior flap of the mucous membrane.

Figure 16 illustrates a Sturmdorf suture inserted anteriorly and including the lower portions of the parametrial bundles which have been previously sutured. The lateral angles of the cervical flaps are closed by one or two chromic sutures and by one silkworm gut suture on either side, deeply placed into the amputated cervix. The ends of these are left long to facilitate their subsequent removal and to provide adequate traction upon the vaginal vault in the treatment of possible secondary hemorrhage. I have at least on one occasion (Case No. 32) had reason to be thankful for their presence.

As shown in Figures 17 to 22, a posterior colporrhaphy and perineorraphy are performed in the usual manner. In the presence of posterior vault prolapse, or of considerable rectocele, a point is made of carrying this reparative procedure as high as possible, usually to the level of the cervix. A continuous blanket suture is used in the posterior vaginal wall and is discontinued at the mucocutaneous junction, to be tied to the upper end of a subcuticular suture commenced at the lower extremity of the perineal wound.

At the conclusion of the anterior wall and vault repair, a one inch gauze drain, impregnated with acriflavine emulsion is inserted into the cervical canal and its free end left out of the vagina to be tied, after the whole operation is completed, to a two inch acriflavine gauze, which is packed snugly, but not tightly, into the vaginal canal, to insure separation of the two vaginal walls and to aid in preventing submucous oozing. These are easily removed together in 48 hours.

As a rule, a Malecot self-retaining catheter is placed in the bladder and drainage maintained for five days, after which time I have never known a patient to require further catheterization.

Further postoperative care is almost negligible, except that it is my routine practice, should there occur any appreciable amount of vaginal discharge, especially if this should be either foul or sanguinous, to irrigate the vagina gently each day with a mild antiseptic solution, through a soft rubber catheter, which is inserted to the vaginal vault. In this manner it is felt that it is possible to remove debris, exudate and blood clot which might favor the establishment or extension of an infective process and thereby lead to possible septic necrosis and secondary hemorrhage. The latter, I regard as one of the major hazards of the postoperative period.

Patients are required to remain in bed 21 days postoperatively and at that time a gentle speculum and rectovaginal bimanual examination is performed. At this time, also, the two silkworm gut sutures are removed from the cervix.

All patients are given a routine set of exercises to perform after leaving hospital and are warned against undertaking any sustained effort and lifting for three months. Thereafter, they are permitted any activity.

In this series of 50 cases which I am reporting, I have analysed the main features as to symptoms, nature and extent of prolapse and the results in Tables I to IX as follows:

TABLE I

PREDOMINANT TYPE OF PROLAPSE

	No.	%
Vault	30	60
Anterior	16	32
Posterior	4	8

TABLE II

SYMPTOMS

	No.	%
Protrusion	46	92
Stress incontinence	31	62
Pelvic discomfort (pelvis, lower abdomen, lumbosacral)	32	64
Difficulty in emptying bowel	3	6
Recurrent cystitis	16	32

TABLE III

PREVIOUS OPERATION FOR PROLAPSE

Case No.	No. of Operations	
13	2	Vaginal and abdominal
16	1	Abdominal
18	2	Vaginal and abdominal
27	1	Vaginal
36	2	Vaginal and abdominal
42	2	Abdominal and vaginal
47	1	Vaginal
48	1	Vaginal

16 %—one or more previous attempts

TABLE IV

PREVIOUS HYSTERECTOMY

Case No.		
1	Subtotal Fibroids	16 days prior to this operation.
10	Subtotal Fibroids	6 years prior to this operation.
18	Subtotal Fibroids	11 years after previous vaginal repair, 6 years before this operation.
27	Subtotal Fibroids	5 years after a vaginal repair, 2 years prior to this operation.

8 % Previous Hysterectomy.

TABLE V

CHRONIC CERVICITIS

	No.	%
Clinical	42	84
Histology	43	86

LEUKORRHEA

	No.	%
Symptoms	32	64
Cured	27	84.3

TABLE VI

IMMEDIATE POSTOPERATIVE COMPLICATIONS

Case No.	Complications
2	Acute parametritis, 29 days postoperative.
3	1. Left saphenous thrombophlebitis. 2. Acute cystitis.
17	Perineal stitch abscess.
21	Acute cystitis—rapid subsidence.
24	1. Acute cystitis—rapid subsidence. 2. Small secondary hemorrhage, 14 days postoperative.
27	Subacute cystitis—rapid subsidence.
31	Delayed healing at vault (diabetic).
32	Severe secondary hemorrhage from cervix (septic necrosis) 16 days postoperative.
42	Small secondary hemorrhage, 8 days postoperative.

9 Cases
18% Complications

TABLE VII

REMOTE POSTOPERATIVE COMPLICATIONS

1. ADHESIVE VAGINITIS

Case No. 7—Mid-point—no complaints of marital distress.
Case No. 12—Vault—no complaints of marital distress.
Case No. 43—Mid-point—easily separated without recurrence.

2. CICATRICIAL STENOSIS

Case No. 16—Junction lower and middle thirds—no marital ditsress.
Case No. 34—Mid-point, almost complete—widow.

TABLE VIII

RESULTS—RELIEF OF SYMPTOMS

	Cured		Improved	
	No.	%	No.	%
Protrusion	45	97.8	1	2.2
Stress incontinence	25	80	3	9.6
Pelvic discomfort	26	81.2	2	6.25
Difficulty in emptying bowel	3	100		
Recurrent cystitis	15	93.7	1	6.3
Average		90.5		4.9

Cases who considered operation a complete success — 45 or 90%
Cases who considered their condition improved — 5 or 10%
Cases who considered their condition unimproved — 0 or 0%

TABLE IX

OPERATIVE RESULT

	GOOD	FAIR	POOR
Anterior	No. Cases—46 92%	No. Cases—3 6% Case No. 1, 20, 36	No. Cases—1 2% Case No. 7
Vault	No. Cases—50 100%		
Posterior	No. Cases—49 98%	No. Cases—1 2% Case No. 13	
Calibre	No. Cases—46 92%	No. Cases—2 4% Case No. 12, 16	No. Cases—2 4% Case No. 7, 34
Total	95.5%	3%	1.5%

SUMMARY

1. A detailed technic for the performance of the Manchester type of operation for genital prolapse is briefly described, together with methods employed in preoperative and postoperative treatment.

2. A series of 50 cases with follow-up is reported with detailed analysis of type of prolapse, symptoms, previous attempts at cure, postoperative complications and results.

CONCLUSIONS

1. The Manchester type of operation is a highly efficient procedure for the relief of genital prolapse in most of its usual forms and under most conditions.

2. Without alteration of the principles underlying the operation, minor modifications can be adequately and successfully applied in various types of cases as occasion demands.

3. The importance of careful preoperative preparation and of post-operative care is stressed.

4. The more common hazards and complications are enumerated.

DISCUSSION

DR. HENRY N. SHAW (Los Angeles): I am sure I express the feeling of the Society in congratulating Dr. Nash on his excellent presentation of the technic of this procedure, and of his excellent results in this series of cases. This question of prolapse is extremely interesting to all of us who practice gynecology. I think one of the interesting things is that where everyone benefits by the pioneers in this work, we don't realize how comparatively new it is. In looking over the history of this particular procedure Donald, of Manchester, makes a note in his reports, that in the five cases he did in 1891, it was only necessary to use a pessary in one case. In fact, many of the early men did a perineal repair just to hold the pessary securely

in place. It's interesting how many people use the names of operations without much real knowledge of their origin. I was grumbling around about being called on to discuss the Manchester operation, when to my knowledge I had never done it under that name, and one of my colleagues in Los Angeles said, "Oh, yes, I've done quite a number of those. Dr. Manchester really originated it, but Fothergill elaborated it."

The technic is excellent; the procedure speaks for itself, as with Dr. Nash's results. But I do not agree with the men from that school who feel that it is adapted to all cases. One of the best reports on this was written by Fletcher Shaw, I think in 1933, and he said that it was used for all cases of prolapse. I feel that it is an excellent procedure in women in the childbearing period, but there are many cases in the postmenopausal group where the situation is better handled by a vaginal hysterectomy. This may be like the obstetrician who came to report at the State meeting on the experience of less than two cases, but in many of these prolapses in the postmenopausal type there is a large cul-de-sac of Douglas, and this can only be properly repaired by high ligation of the hernial sac, and bringing together of the cul-de-sac tissues firmly to prevent recurrence of the hernia or development of a hernia. Also, in many elderly women the uterus is diseased, and in such a case it is good insurance against later trouble to take out a useless uterus and insure an adequate support which will also prevent the troublesome enterocele. In my series of prolapses, four enteroceles have occurred following my efforts at cure and in searching for an alibi I feel that there may be something in this theory —that when you have repaired the front wall and also the back wall, where a bulge has developed, it throws the pressure on tissue which has previously been strained and the patient may develop a hernia through that weakened area. This I am sure, can be prevented by careful excision of the hernial sac and treating it as such.

I am very much impressed with the value of Dr. Nash's preoperative precautions. I have seen one patient die of a septicemia from an inadequately treated ulcer on the cervix. It is very important to clear up any cystitis, if possible. In many of these cases, however, the cystitis is due to the prolapse and the residual urine, and it may be difficult to clear it up entirely until the prolapse is adequately treated. Another thing that I think should be stressed is not only the curing of the infection of the bladder but a careful study of the kidney function. In many of these women, particularly in the older group, there occurs the same condition that is noted in prostatic obstruction in elderly men. The urologists have taught us that a careful evaluation of this point with adequate treatment, where an insufficiency is found, will often change what might have been a tragedy into a success.

DR. ALBERT V. PETTIT (San Francisco): Dr. Nash gives a very clear description of the Fothergill operation for the repair of cystocele and his results are excellent, as they should be. This operation is one of the very useful technics which may be used for the purpose of supporting the prolapsed urinary bladder. This operation resembles other described procedures in that it makes use of condensed areolar tissue containing a few smooth muscle fibers as the support.

Herniation of any of the pelvic viscera through the pelvic floor aperture pre-

supposes some defect in the closure mechanism, either congenital or acquired. Since this mechanism of normal closure consists of tonic striated muscle bundles of the levatores ani with an intact nerve supply, I believe that any discussion of the treatment of prolapse which omits mention of some method of restoring tone to these muscles is incomplete.

Confusion will be avoided, in my opinion, in descriptions of the Fothergill operation, or of any operation using subvesical areolar tissue, in the mobilization and reduction of bladder hernia, only if the importance of adequate perineorrhaphy is stressed, as a necessary adjunct.

DR. RAYMOND WATKINS (Portland): Dr. Nash has presented a very interesting paper which opens a number of points for consideration.

At the recent meeting of the American College of Surgeons in Chicago, Dr. Louis Phaneuf of Boston headed a panel discussion on Prolapse of the Uterus. Participating in this symposium were a number of outstanding gynecologists who presented various operative procedures commonly used for the correction of this pathology. Not only was the Manchester operation discussed, but the Watkins interposition operation, the Mayo vaginal hysterectomy, the LaForte procedure and anterior and posterior colporrhaphy followed by intra-abdominal uterine ligament shortening. Each operative procedure was shown to be of value if used where indicated and if the surgical procedure was properly carried out.

In Dr. Nash's discussion of the Manchester operation, he stated that this surgical type of correction might be used from 30 years of age onward. In women during the child bearing age, amputation of the cervix, which is part of the Manchester procedure, is quite prone to produce dystocia in future labors. For this reason, we think it contraindicated, if future pregnancy might be expected.

In women at or about the menopause, the interposition operation gives very satisfactory results where cystocele is the predominant finding and the cervix does not protrude beyond the vaginal orifice. One objection to this operation was that a few patients complained of frequency of urination following it. This has been almost entirely overcome by plicating the bladder fascia from the urethra downward over the bladder trigone before interposing the uterus.

Dr. Shaw discussed enterocele occurring after operations for the correction of prolapse. I presume he was alluding to surgical procedures such as the Mayo vaginal hysterectomy. We had a similar experience with this operation. In discussing the production of such enteroceles with Dr. Lillian Farrar, she suggested that we might be pulling the upper part of our united broad ligaments too far forward under the symphisis, leaving the cul-de-sac stretched out in such a way that it yielded to intra-abdominal pressure with a resultant hernia or enterocele. She suggested that the pubocervical fascia be united from the urethra backward over the trigone of the bladder and the upper part of the united broad ligaments sewed into the vagina at this point. This modification has been very gratifying for we have had no enteroceles occur since its adoption. It is important in The Mayo operation that the sac of a beginning enterocele be dissected out and the uterosacral ligaments united before plication of the broad ligaments.

DR. NASH (closing): I am delighted with the criticism. I hoped that there would be criticism and to me it has been most instructive.

I feel that Dr. Pettit has touched a most important point and I have to confess that I agree with him. I think that attention devoted only to the bladder is, of course, futile in the extreme, and I can see his point clearly that I did not sufficiently emphasize the mechanism of support of the uterus and the attention which should be paid to that mechanism in dealing with this condition. I am afraid that I allowed that to be taken for granted, since it would be difficult to deal adequately with that consideration in a paper of 15 minutes' duration, unless it were the only phase of the subject to be discussed.

I do not agree with Dr. Shaw in his contention that vaginal hysterectomy as such has any inherent superiority in this particular regard, in that I do not think that the actual repair of the prolapse is rendered more efficient by the removal of the uterus. Perhaps experience comparable with that of Dr. Shaw will teach me otherwise.

With regard to Dr. Watkins' point about childbearing, I think that it is a most important one and I understand that the Manchester school at the present time deliberately avoided its performance in women of the childbearing age who contemplate further pregnancy. Their experience has apparently shown them that there is diminished fertility and likelihood of abortion, but not any difficulty in full term delivery, with very few exceptions. Since I believe that to be true, it is accordingly my practice, as Dr. Watkins suggests should be done, to avoid the performance of this procedure in young women who contemplate further pregnancies, and those younger women who are included in my series had had their families, insofar as their desires were concerned. I think that in such cases the operation is as ideal as it is in older women.

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ARRESTED DEVELOPMENT OF THE RECTUM*

(Report of a Case and Review of the Embryology and Anatomy Concerned)

By E. A. Daniels, M.Sc., M.D.

Montreal

ARRESTED development of the rectum is an extremely rare congenital defect. Such an anomaly, according to Starr (quoted by Lockhart-Mummery[1]), is found only in a very small percentage of all births, and he states that it may be observed approximately only once in every ten thousand deliveries. It is reasonable to suppose however, that this figure is probably too high, since such cases are very rarely encountered by active hospitals and clinics. The author has recently observed and operated upon a newborn infant with such a congenital defect. The rarity of this lesion should make an anomaly of this type of sufficient interest to report. Although rare it is nevertheless necessary to understand the fundamentals of such congenital defects. Such a condition presents itself as a rule as a surgical emergency, and one must be prepared to carry out an appropriate plan of treatment. The danger to the child's life is imminent, and surgical intervention in a newborn infant is a grave undertaking. A knowledge of the evolution of the development of the terminal bowel and its anatomy in the newborn therefore becomes imperative.

Embryology and Anatomy Concerned

In the early stages of development in the human embryo the allantois communicates and is continuous with the hind-gut, and anteriorly enters into the formation of the body-stalk. A U-shaped angulation is thus formed at the point where the allantois joins with the hind-gut. The apex of this angulation becomes increasingly dilated downwards to form a pouch situated at the posterior end of the embryo, known as the post-allantoic gut. This is really a process of downward prolongation and sacculation of the hind-gut into which opens the allantois in front. The downward prolongation of the hind-gut (post-allantoic gut) continues to grow downwards and backwards past its anterior or cloacal opening, continuous with the growth of the hind portion of the body of the fetus. The communication between the hind-gut and the allantois in front soon becomes sealed off in the process of development. At a later stage the hind-gut and its posterior sacculation, already described, are completely separated from the allantois in front and are thus without any communication with the body or allantoic stalk. In the male the bladder, prostate and urethra are developed at the lower portion of the allantois, whereas the Müllerian ducts are developed from the upper portion of this structure. In the female the uterus and vestibule of the vagina are developed between the hind-gut and the allantois. Fig. 1 shows the above in diagrammatic fashion. It will be noted that the communication between the hind-gut and the allantois ultimately becomes a firm band of tissue, referred to as the obliterated communication between the hind-gut and allantois (Fig. 1). Should this communication fail to close, that is, when persistent, this occurs at a definite point as follows: in the male, at the prostatic urethra; and in the female at the posterior vaginal wall just below the cervix. Lockhart-Mummery states that the site of this communication is thus constant at the two above-mentioned points in male or female because of the constancy of the embryological picture. He points out that this does not follow any haphazard scheme, and for this reason a communication between the hind-gut and allantois is generally into the prostatic urethra in the male and at the top of the posterior vaginal wall in the female. In arrested development of the rectum the post-allantoic gut, that is, the posterior prolongation of the hind-gut, is entirely missing. The point of junction of the hind-gut and the post-allantoic gut occurs at the point of the reflection of the peritoneum from the anterior surface of the rectum. Consequently, in cases of imperforate rectum the blind termination of the hind-gut will be found opposite the recto-vesical pouch of peritoneum in the male and the recto-vaginal pouch of peritoneum in the female. Such were

* From the Woman's General Hospital, Montreal.

the findings in the anomaly to be presently reported.

The next step in the development of the ano-rectum is as follows. A slight depression or dimple appears on the body wall between the coccyx and the mid-point of the perineum, at a point parallel with the long axis of the rectum (post-allantoic gut). This is known as the proctodeum, which is simply an ectodermal depression in the area which will go to form the future anus. This is shown in the illustration. A membrane is thus formed, known as the proctodeal membrane, which soon breaks down or is absorbed, forming a communication with the post-allantoic gut. It can be thus seen that the rectum and anus are developed from three distinct structures: (*a*) the hind-gut; (*b*) the sacculation developed by the downward prolongation of the hind-gut, already referred to as the post-allantoic gut, and (*c*) the proctodeal depression. The point of junction between the proctodeum and the hind-gut, that is, the point where the proctodeal membrane has broken down, is seen in the adult rectum as the dentate or pectinate line. This dentate or pectinate line marks the point of junction between the squamous epithelium of the body wall and the columnar epithelium of the hind-gut.

One can visualize the type of congenital defect that might be observed if the above steps in the evolution of the development of the ano-rectum are understood, namely: (1) Failure of the downward development of the post-allantoic gut. The blind pouch marking the termination of the hind-gut is therefore found opposite the recto-vesical or recto-vaginal peritoneal reflection: (*a*) without a fistulous communication to the prostatic urethra in the male or posterior vagina in the female; (*b*) with a fistulous communication to the prostatic urethra or posterior vagina. The defect to be presently described belonged to 1(*a*). (2) Defects resulting from imperfect obliteration of the proctodeal membrane. Such a defect will therefore be found low-down at the dentate line.

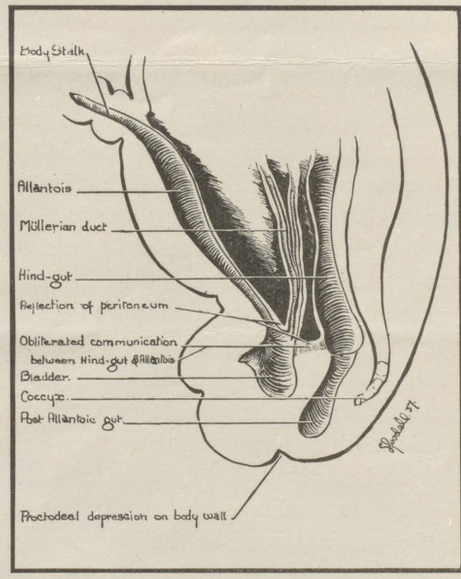

Fig. 1

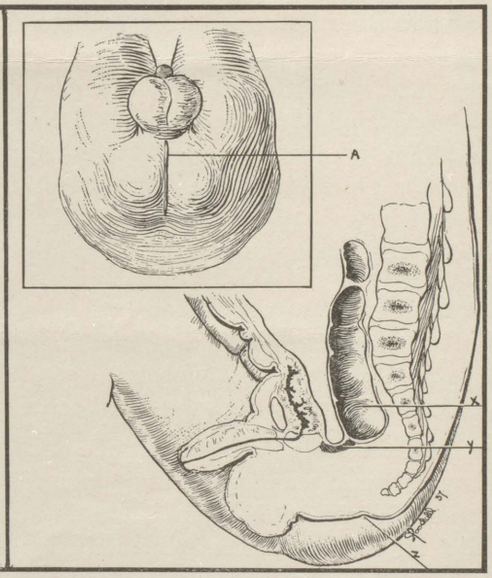

Fig. 2

Fig. 1.—The above illustrates the various steps in the evolution of the development of the rectum. It is seen that the future rectum is developed as a downward prolongation from the hind-gut, known as the post-allantoic gut.

Fig. 2.—The inset shows the line of incision from just behind the scrotum to the coccyx.

X.—In the large illustration, demonstrates where the hind-gut had become arrested in its development, opposite the recto-vesical pouch of peritoneum. The post-allantoic gut is absent.

Y.—Demonstrates the obliterated communication between the prostatic urethra and bladder (allantois), and hind-gut.

Z.—Represents the area where the proctodeal dimple was present, surrounded by sphincteric tissue. This was the site of the anal canal and was used for this purpose at operation.

TREATMENT

One is dealing with a newborn infant in the presence of a surgical emergency. It is wise to wait for several hours in order that the child may recover from the trauma incident to its passage through the birth canal. It is reasonable to suppose that this period should not exceed twelve hours, since there is no outlet for meconium.

The pre-operative care consists of warmth and 5 to 10 per cent saline glucose given interstitially in quantities of 25 to 50 c.c. every hour or two until the time of operation. It is quite difficult to administer any of the usual anæsthetics. The author in his case employed a pledget of gauze dipped in brandy and given to the child by mouth to suck. This was found quite adequate. In female children arrested development of the rectum is very often accompanied by a fistulous communication between the hind-gut and the posterior vaginal wall which provides an outlet for meconium and fæces. This defect is therefore not an emergency, and when such a communication is found it is wise not to disturb the infant until the age of ten years or more is reached. At that time a modified perineorrhaphy, closure of the fistula, and implantation of the blind end of the hind-gut into the proctodeal area can be done. In male children born with absence of the rectum in which there is a fistulous communication between the hind-gut and the prostatic urethra, then the condition becomes incompatible with life, firstly, because such an opening is not sufficient for the free passage of meconium and fæces, and, secondly, because of the imminent danger of urinary infection. Such a defect must be treated as a surgical emergency by both closure of the fistulous tract, and by providing an outlet for meconium and fæces.

Whenever possible the blind end of the hind-gut should be found and brought down to the proctodeal area before opening the gut. Lockhart-Mummery and other workers in this field believe that it should never or very seldom be necessary to perform a colostomy; and that in nearly all instances it should be possible to find the blind end of the hind-gut at the point of its arrested development, and by carefully practised surgery to bring this down comfortably and implant it into the proctodeal area. A long operation should not be indulged in, as this endangers the child's life. The rarity of the lesion does not afford the surgeon a very wide experience in dealing with these cases. It is therefore necessary to have a knowledge of certain landmarks which will be consistently found in the anomaly. One must also be guided by the experience of other workers in the field who have had enough cases of this kind to enable them to outline a general plan of treatment.

The perineum should be opened by a mid-line incision extending through the blind proctodeal dimple from just behind the scrotum or fourchette to the coccyx. The dissection must be kept as much as possible in the hollow of the sacrum, as otherwise there is considerable danger of wounding the genito-urinary organs. Blind probing through a perineal stab in the hope of finding the bowel pouch is regarded as a dangerous method of treatment. The peritoneum need not be entered if dissection is confined to the hollow of the sacrum. As already pointed out by Lockhart-Mummery, the blind end of the hind-gut will nearly always be found opposite the recto-vesical or recto-vaginal pouch of peritoneum. One can identify this by first encountering the obliterated communication between the hind-gut and allantois as a heavy band of tissue running forwards. This may be divided to facilitate the dissection and to determine whether it contains a fistulous tract. Forcing the child to strain or cry will enable the operator to actually palpate the bulging termination of the gut and its distended meconium content. The cellular tissue all around this blind pouch should be freed and effective mobilization of the hind-gut carried out. The proctodeal area, as already pointed out, develops quite distinctly from the hind-gut as a depression on the body wall, and will as a rule be found to contain good muscular tissue. This was found in the author's case. After freeing the hind-gut it is brought down without tension to the proctodeal area and opened. The divided bowel lumen is then sutured all around with fine linen thread to what appears as the anal area. It must be remembered that the anal orifice in the infant lies almost in a straight line parallel with the tip of the coccyx,[2] that is, more posterior than in the adult. This should guide one in determining the area of the future anus. During the operation the child

should be surrounded by warm blankets and shock thus minimized. Post-operative care consists of warmth, fluids and glucose interstitially, and a full quota of breast milk by mouth. The pædiatrician's advice should be available at all times.

CASE REPORT

A primipara, aged 23, in normal health. Pre-natal course uneventful. Pelvic measurements normal; labour normal. The baby was born at midnight, April 26, 1937. Birth weight 5 pounds, 6 ounces. The nurse first noticed that the rectal temperature could not be taken because of the absence of an anal orifice. This was brought to the attention of the attending obstetrician. The following morning at ten o'clock the author examined the child. There was a very small depression or dimple in the region where the orifice should have been. This was surrounded by a small amount of pigment. A perineal bulge could not be elicited by pressure on the abdomen or by forcing the child to cry. It was then decided that the blind end of the hind-gut had become arrested high up in the pelvis.

The infant was prepared for operation, and this was carried out at eleven o'clock—11 hours after birth. Brandy was administered as an anæsthetic and the infant was surrounded with hot water bottles. The lithotomy position was employed. The incision was made from a point just behind the scrotum to the coccyx, as shown in diagram A. The dissection was continued posteriorly and trauma to the prostatic urethra, seminal vesicles, bladder and rectovesical pouch of the peritoneum was carefully avoided. The obliterated communication between the allantois and hind-gut, as shown in diagram Y, was encountered just below the recto-vesical pouch of peritoneum. No fistulous communication existed and this band of tissue was divided. The blind end of the hind-gut was found, as shown in the diagram X, opposite the peritoneal reflection and in the hollow of the sacrum. The gut was freed anteriorly without disturbing the peritoneum, and was dissected free posteriorly and laterally. Mobilization of the segment of the bowel was effectively carried out so that it could be easily brought down without tension to the proctodeal area, shown as Z in the diagram. The blind end of the hind-gut was thus brought down to the proctodeal area through which ran the skin incision as shown in the inset A. This was anchored by a stay suture of fine linen and the blind pouch incised. Meconium at once freely exuded. The mucosal periphery of the opened hind-gut was carefully sutured all around the area which was regarded as the anal site, Z. The skin incision was then closed without drainage by deep through and through discontinuous sutures of fine linen.

The child was then returned to his crib surrounded by hot water bottles. Five per cent saline glucose was administered interstitially at intervals, and feedings of small quantities of breast milk commenced at once. (The mother could not nurse, but breast milk was secured). The post-operative course was uneventful. The sutures were removed on the tenth day, the wound healed without infection, and the bowel movements were satisfactory and bulky. The stools were both formed and mushy, and there appeared to be good sphincteric control. The infant left the hospital on May 21, 1937, weighing 6 pounds, 14 ounces, and appeared to be in robust condition. Weight November 2, 1937, was 14 pounds 6 ounces. Examination revealed good sphincteric control. The anal orifice easily admitted a No. 22 F. catheter. The child was on a normal diet and appeared robust and well.

There is no reason to suppose that the child's future will be seriously affected by this congenital anomaly, since the operative procedure appeared to have been successfully carried out. At operation sphincteric tissue was quite discernible in the area of the proctodeal dimple. This was carefully sutured all around to the mucous membrane.

SUMMARY

Arrested development of the rectum when encountered in a newborn infant is a surgical emergency, which should be dealt with in a definite manner. Although a formidable surgical procedure, it becomes necessary to carry it out in order to preserve the child's life. Such interference can give a happy result if performed at an early period and under proper conditions.

The author wishes to express his thanks to Dr. F. E. Thompson, for permitting him to take charge of this patient and for his kind collaboration; to Dr. M. Scherzer, the attending pædiatrician, for his conscientious care and interest. The writer is also very grateful to Dr. J. R. Fraser, of the Department of Obstetrics, Royal Victoria Hospital, whose department was good enough to supply us with a liberal quantity of breast milk. This in no small measure contributed to the successful outcome of the case. Thanks are due to Miss Shirley Goodall for the excellent illustrations.

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SIMPLE ANAESTHESIA

by

HAROLD T. DAVENPORT

M.B. Ch.B. M.R.C.S. L.R.C.P. D.A. F.F.A.R.C.S. (Eng.)

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INTRODUCTION

This pamphlet was written whilst the author was working in South India. It presents a plan of modern anaesthesia for use in less than ideal situations, to be taught in a rural hospital teaching programme. The request for advice came from Dr. P. Brand (the Principal), with the support of Dr. G. Lewis (Chief Anaesthetist), of the Christian Medical College, at Vellore.

It is essentially an initial primer which will no doubt be modified after more extensive practice. The considerations of expense, present practices, facilities and available staff are those of South India, but may be applied to other newly developing areas of the world.

The section containing drugs and apparatus needed are arranged so that other local supply sources can be inserted. Intentionally the description of the methods is very brief, and acknowledged references are omitted so that foreign language translation or interpretation are aided. The script takes less than forty-five minutes to read in English. Certain trade names have been used because they are universally known, but other products of the same active agents can be freely interchanged.

DRUGS NEEDED	A SOUTH INDIA SOURCE	ANOTHER SOURCE
Morphine Sulphate	First obtain a licence from the Collector. He then gives transport permit to buy it from a firm. Dahna & Co., 86 Naicken St., Madras	
Hyoscine Bromide	Bengal Immunity, c/o Mahendra & Co., 161 Nyniappa Naick St., Madras	
Ethyl Ether (Ether) *Explosive*	Alembic, 34 Angappa Naick St., Madras 1, P.O. 235, Georgetown	
Thiopentone (Pentothal)	May & Baker Co., P.O. 693, Madras, 2	

DRUGS NEEDED	A SOUTH INDIA SOURCE	ANOTHER SOURCE
Lidocaine 2% (Xylocaine)	Suhrid Geigy & Co., c/o Raka Corp., 138 Moore Street, Madras 1	
Gallamine (Flaxedil)	May & Baker Co.	
Spinal Nupercaine 1/200 & glucose 6% 3 ml. (1/400 & glucose 5% 2 ml.)	Ciba & Co., c/o Orientile Mercantile Dist., 99a Armenian St., Madras 1	
Atropine	Bengal Immunity, c/o Mahendra & Co.	
Neostigmin (Prostigmin)	Roche, c/o Voltas Put, Ltd., 115/116 Armenian St., Madras 1	
USEFUL ADDITIONS		
Adrenaline 1/1000	May & Baker Co.	
Calcium Gluconate 10%	Bengal Immunity, c/o Mahendra & Co.	
Trichlorethylene (Trilene)	Trilene, I.C.I. (Put), Ltd., P.O. 182, Calcutta	
Ethyl Chloride *Inflammable*	Alembic	
APPARATUS NEEDED		
Nylon syringes, 20 ml. size with eccentric nozzle, 10, 5 & 2 ml. size	South India Surgical Co. (Bldg) 128 & 142 Nayniappa Raick St., Madras 3 'Vandermic', England	
Needles, 20, 22, 18 steel wire gauge	South India Surgical Co.	
Mitchell needles, 3 sizes	Anaesthetics, H.7 Anandashram Proctor Rd., Grant Rd., Bombay 7	
Spinal needles, size 20, 22	Anaesthetics	
2 Open drop masks	Anaesthetics	
2 8oz. medicine bottles	Anaesthetics	
1 Macintosh laryngoscope, with adult blade	Indian Oxygen Corp., 7A Varthianatha Mudali St., Tordiarpet, Madras 21	

APPARATUS NEEDED	A SOUTH INDIA SOURCE	ANOTHER SOURCE
Endotracheal tubes, Magill 7–10, plain portex 0–10	Anaesthetics	
Cuffed 7–10	Indian Oxygen Corp.	
Set of Berman plastic oral airways	Anaesthetics	
Set of Bardex naso-pharyngeal tubes	Anaesthetics	
Set of Cobbs connectors	Indian Oxygen Corp.	
Macintosh director or gum elastic bougie	Anaesthetics	
A self-filling bag (Ambu type)	Indian Oxygen Corp. or S. & W. 43 Bredgade, Copenhagen Denmark	
or		
Inflating bellows (Oxford Pentland)	Pentland Equipment Co., Radley Road, Abingdon, Berks, England	
A non-rebreathe valve, intermittent positive pressure type (Ruben)	Indian Oxygen Corp. or S. & W. Copenhagen	
Endotracheal connector	Indian Oxygen Corp.	
Face masks, 3 sizes	Anaesthetics	
A draw-over vapourizer bottle (Marrett type) with graduated control and little resistance to gas flow	Anaesthetics	
2 lengths of corrugated tubing	Indian Oxygen Corp.	
A suction/blowing pump, electric or foot operated	South Indian Surgical Co.	
Suction catheters		
Oxygen cylinders with reducing valve and flow meter	Anaesthetics	

USEFUL ADDITIONS	A SOUTH INDIA SOURCE	ANOTHER SOURCE
Infant Flagg laryngoscope blade	Anaesthetics	
Clausen harness and ring	Anaesthetics	
Cyprane inhaler	Anaesthetics	
Magill forceps	Indian Oxygen Corp.	
Ferguson mouth gag	Anaesthetics	

RESUSCITATION

A knowledge of resuscitation procedure is required before any surgical operation, and should clearly be taught to all those involved.

Outline of Action:

1. Time is critical when major vessel pulses disappear, spontaneous breathing ceases, or pupils dilate. Do not hope. ACT IMMEDIATELY WHEN IN DOUBT. Oxygen must be supplied to the brain in less than three minutes. Note the time if possible and call help.

2. Give 3–4 rapid MOUTH TO MOUTH VENTILATIONS. Fully extend the head and blow into the patient's mouth or nose until the chest expands. Use airway, face mask or bellows if available (Figure 1). If the chest does not move, clear

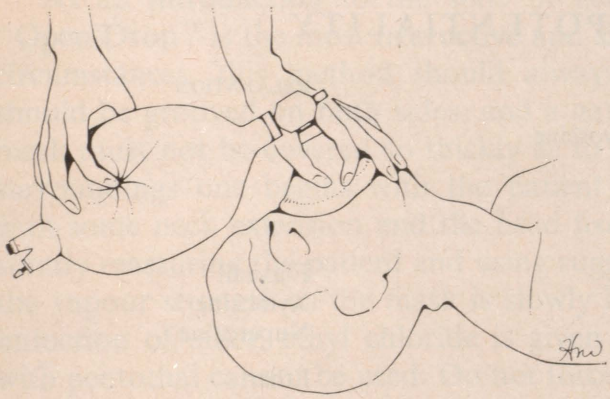


FIGURE 1

Air being blown into the lungs with a self-filling bag through a non-rebreathe valve. The head is extended.

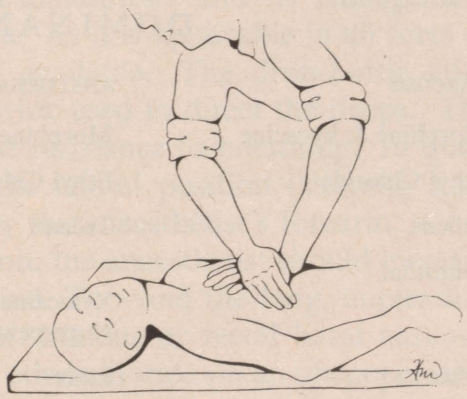


FIGURE 2

The heart being compressed by pressure on the sternum towards the spine on a hard surface.

airway obstructions with a sponge or suction. If a second person is present continue ventilation at 12/min.

3. With patient on a solid surface apply EXTERNAL CARDIAC COMPRESSION 60/min. Use one hand on the lower third of the sternum and place the second hand on the first (Figure 2). Just sufficient pressure should be exerted to produce effective arterial pulsations, constriction of pupils and spontaneous respirations; raising the patient's legs, and arms, may help by returning blood to the heart. (If not effective, a doctor may incise the 4th left intercostal space and compress the heart directly.) If one is alone, give intermittent mouth to mouth ventilation 3–4 times every 30 seconds.

4. Test every three minutes for spontaneous cardiac action and continue cardiac compression and artificial ventilation until the blood pressure and spontaneous ventilation are adequate.

[5. Later get an electrocardiogram. If fibrillation of the heart is present, use an external electrical defibrillator. If there is asytole, inject into the heart 5 ml. 1/10.000 adrenaline, or 10 ml. 10% Calcium Gluconate. Do intravenous cut down, and add continuous vasopressors, hypothermia, intravenous 30% Urea, or artificial ventilation as needed.]

THEORY

It is often best to use a combination of agents so that the dominant potentiality of each is exploited. (Balanced anaesthesia.)

DOMINANT POTENTIALITY

NARCOSIS	ANALGESIA	RELAXATION
Morphine & hyoscine	Morphine & hyoscine	
Ethyl Chloride	Ethyl Chloride	
Trilene	Trilene	
Pentothal		Flaxedil
	Xylocaine	Xylocaine
	Nupercaine	Nupercaine
Ether	Ether	Ether

If combined freely the techniques described can compliment each other and enable an administrator to choose anaesthetics which he can make safe and satisfactory in most instances.

PRELIMINARIES

Study of the patient's condition and preparation before operation greatly assists in subsequent care and anaesthetic choice. Any patient undergoing an operation, however minor, should be in the best condition possible. Decompensation of the heart, wet infected lungs, high temperature and/or dehydration, reduced amount of blood with or without hypotension, all must be dealt with preoperatively if morbidity is to be reduced. Such conditions as aortic valve and coronary vessel disease, emphysema, drug suppression of adrenal and autonomic systems can complicate any anaesthetic.

Monitoring with a stethoscope strapped to the precordium, or with a blood pressure apparatus on the arm, should be done at all times. Frequent checks of the temporal pulse, chest excursion and the colour of the blood in the wound should be routine.

METHODS

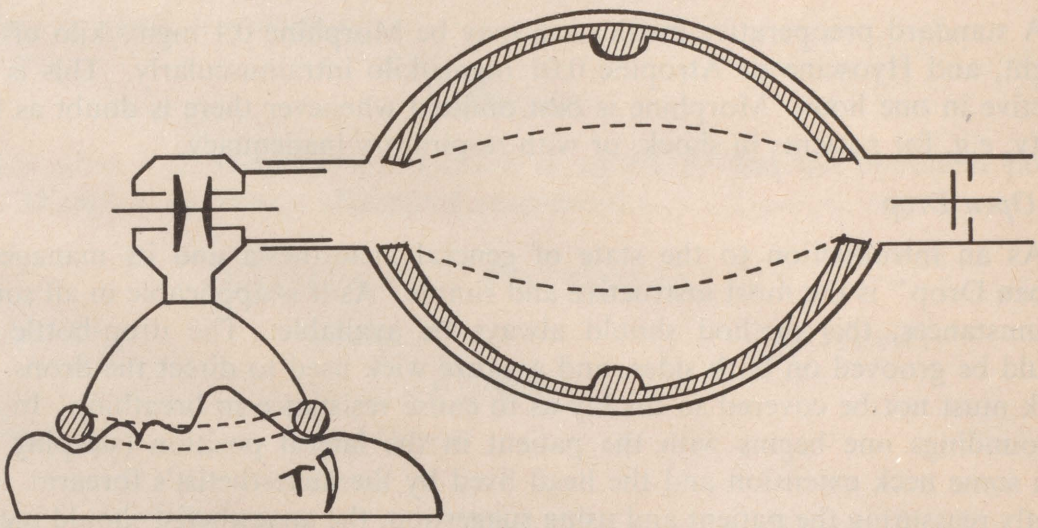
A standard preoperative medication may be Morphine 0.1 mgms/kilo of body weight, and Hyoscine or Atropine 0.01 mgms/kilo intramuscularly. This is fully effective in one hour. Morphine is best omitted whenever there is doubt as to its safety, e.g. for patients in shock, or with respiratory inadequacy.

1. *Open Drop*

As an introduction to the state of general anaesthesia and its management "Open Drop" is the most instructive and simple. As it is applicable in all sorts of circumstances, this method should always be available. The drop-bottle cork should be grooved on both sides, and a gauze wick used to direct the drops. The mask must not be covered so thickly as to cause resistance to breathing. In quiet surroundings one begins with the patient in the lateral position (sleeping side) with some neck extension and the head fixed by the anaesthetist's forearm. Constantly reassuring the patient and using suggestion, the anaesthetist should increase the vapour strength as the mask is slowly lowered. To avoid the long, unpleasant induction of ether, ethyl chloride is given if intravenous or rectal basal narcosis with pentothal cannot be used. Do not force the ethyl chloride on an uncooperative patient or whenever there is breath holding. Change rapidly to ether as soon as consciousness is lost—this is indicated when there is no wink reflex if the eyelash is touched. Next, place rubber dam (old gloves) over the eyes to avoid abrasions and chemical burns. The optimal level of anaesthesia—to obtain for the major part of most operations—is when the patient's pupils are centrally fixed and full ventilatory excursion of the chest occurs with each unobstructed breath (Stage 3, Plane 2). Ether is dropped steadily onto the mask to keep it damp (not saturated), and an artificial airway is inserted. Insufficient or irregular ventilation may indicate a light or a deep anaesthetic level, and the former is indicated if the eyes are moving. It is important that the anaesthetic level is dependent upon what stimulation is occurring and anticipation of the likely changes, as the operation progresses, makes for smoothness of administration. To achieve deeper levels one may need to muffle up the mask with gauze or towelling to increase the vapour concentration; this should only be done for short periods. This system really is a partial rebreathe type in which some carbon dioxide will accumulate and the oxygen concentration

become lowered so that a flow of 500 ml./min. of oxygen from a cylinder to the upper airway is beneficial.

With the open drop method, spontaneous ventilation is imperative, and a serious disadvantage is that ventilation cannot be augmented when necessary. If a self-filling bag, non-rebreathe valve and mask (Figure 3) is always available, artificial ventilation can then be readily given at any time it appears necessary.



A SELF-FILLING BAG AND NON-REBREATHING VALVE

FIGURE 3
Shows the internal construction of a self-filling rubber bag and non-rebreathe valve.

After an ether anaesthetic with "open" administration, some bland oil (castor) may be placed in the conjunctival sacs. The patient must recover consciousness in a lateral position, this being an inflexible rule in all anaesthesia. To continue open ether after endotracheal intubation, one can pour ether onto gauze stretched over a funnel which is attached to the endotracheal tube. The funnel must be below mouth level, and the connecting tube must be of large lumen and short length.

2. *Intravenous*

Pentothal, never stronger than $2\frac{1}{2}\%$ solution, may be kept until turbid and is cheaper made in bulk. Pentothal may be given into any suitable vein from a 10 or 20 ml. syringe. The vein should be distended by applying a soft rubber torniquet (1″ Penrose tubing) and stroking the limb. It is senseless to do multiple punctures in an effort to provide a pleasant induction. One must also develop skill with

inhalation inductions so that they are not unpleasant. With an initial injection of 3–6 ml., drowziness should follow rapidly. If not, faulty injection or a slow circulation time should be suspected. After unconsciousness supervenes, half of the amount already given may be further added (there is no routine dose), then other agents may be begun or minor procedures undertaken. As pentothal has some anti-analgesic properties if it is used alone, surgical stimulation may lead to movement of the patient. To avoid giving excessive amounts it may be preferable to supplement with other agents. The 1 gm. limit of barbiturate per anaesthetic in adults is a rough rule to avoid undue depression.

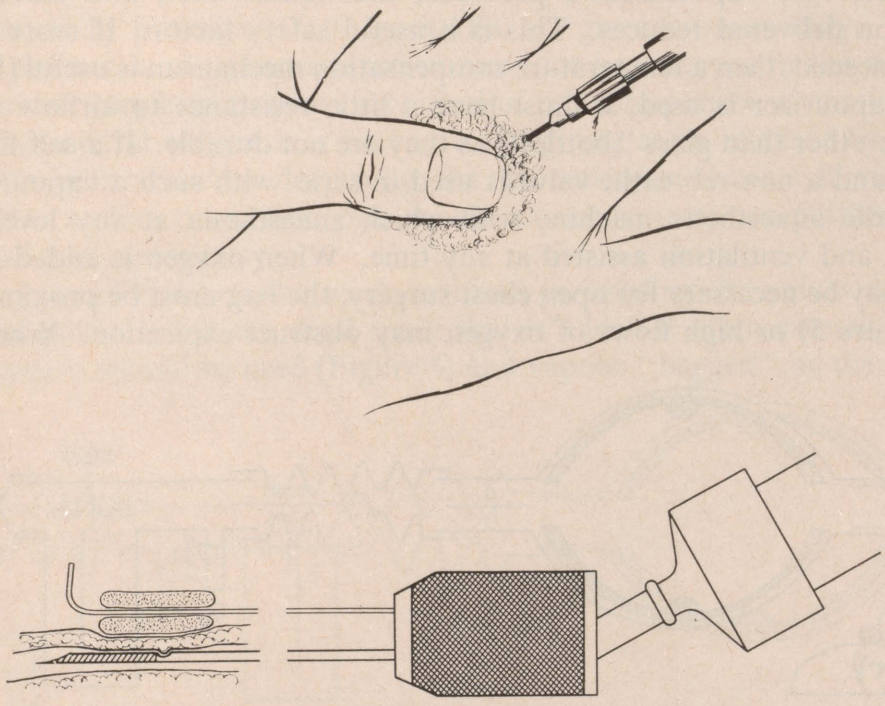


FIGURE 4
A Mitchell needle.

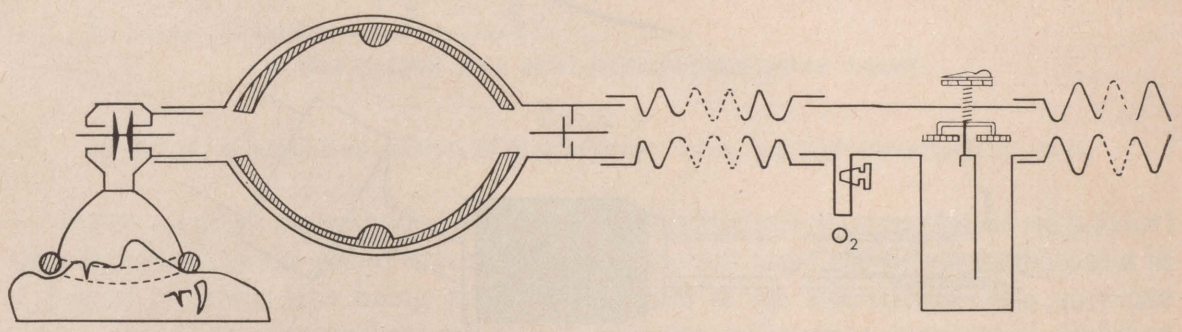
Until the airway can be guaranteed, pentothal should never be given. One must be prepared to apply artificial ventilation or cardiovascular support sometimes with this potent agent. If a needle which remains patent (Mitchell) (Figure 4) or drip infusion are used, then more injections may be easily given if desired.

Rectal pentothal, 10% solution (not essentially sterile) will make an uncooperative child sleep in five minutes if 40 mgms. per kilo of body wgt. is used after

routine premedication. The calculated amount of solution should be run in carefully from a syringe by gravity through a multi-holed catheter 8 cm. up the rectum. In this way evacuation is less likely, the excess solution can be later withdrawn to reduce post-operative sleep.

3. *Vapourizer and Relaxant*

An apparatus (Marrett draw-over type) which will permit a liquid anaesthetic agent to be vapourized in variable amounts, is more controllable and less wasteful than open drop. If two containers are used, then more than one agent may be given at once. As vapourization proceeds, the liquids cool, and therefore the concentration delivered reduces. This is a useful safety factor. If more physical accuracy is needed, then a temperature compensation mechanism is useful (E.M.O.). Whatever vapourizer is used, it must have a little resistance to airflow and preferably have other than glass "bottles" as they are not durable. If a self-filling bag or bellows, and a non-rebreathe valve is used in series with such a vapourizer, one has a versatile anaesthetic machine with which anaesthesia at any level can be maintained, and ventilation assisted at any time. When oxygen is added to a side nipple, as may be necessary for open chest surgery, the bag must be proximal to the patient (Figure 5) or high flows of oxygen may obstruct expiration. When venti-



AN ANAESTHETIC APPARATUS FOR SPONTANEOUS OR CONTROLLED VENTILATION —WITH OR WITHOUT ADDED OXYGEN.

FIGURE 5

An arrangement of anaesthetic apparatus suitable for most general anaesthesia. An endotracheal tube may be placed distal to the non-rebreathe valve instead of a mask.

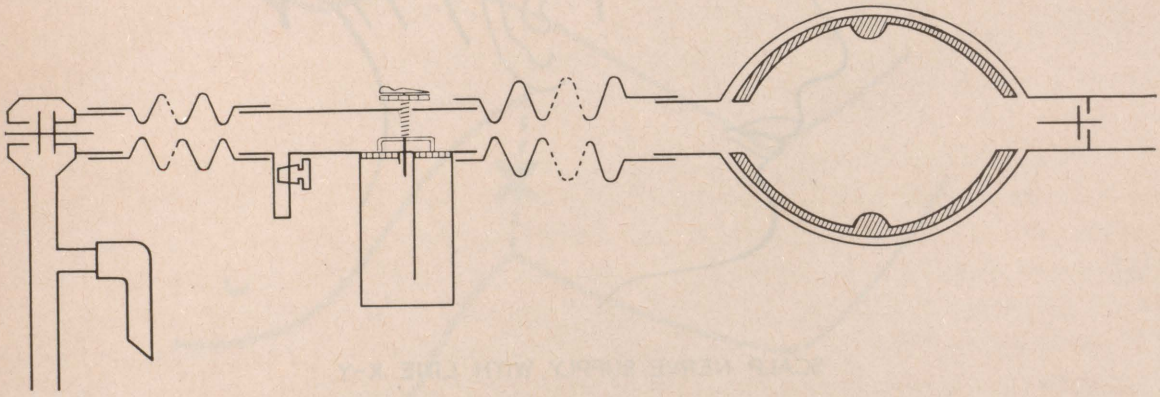
lation is assisted or taken over, one must be sure that sufficient air moves in and out of the lungs by constant observation of chest movements and listening to expiration. With intermittent positive pressure breathing, over-ventilation is preferable. However, predomination of a free expiratory phase over inspiration

is needed to minimize cardiovascular embarrassment (this may be achieved by releasing the pressure completely and having a slight pause on expiration); most especially is this important in critical states when resuscitation is being undertaken.

While utilizing light anaesthesia with ether, and/or trilene, better conditions may be obtained for some operations by using flaxedil, a relaxant. It is given intravenously in small doses of 20 mgms. intermittently—the effect of each addition being observed for five minutes. The average duration of its effect is 20–30 minutes, and it is cumulative especially with kidney dysfunction. If there appears to be a residual effect of the relaxant at the end of an operation, i.e. spontaneous breathing is inadequate, then whilst one continues to assist ventilation, atropine 0.02 mgms/kilo, followed by prostigmin 0.07 mgm/kilo, are given intravenously.

There are occasions when relaxation may not be essential in the surgical field but a relaxant may help to provide control of reflex responses, or control of ventilation. For wound closure it is best to avoid giving extra relaxant by using local anaesthetic infiltration or deepening the general anaesthesia. If relaxants are unavoidable at this time, then one should expect to continue patiently to assist ventilation post-operatively.

In very small children the work involved in opening the valves during spontaneous respiration and the dead space that is added is often excessive, so that a T-piece system should be used (Figure 6) and vapour "bagged" by the anaesthetist



A DIFFERENT ARRANGEMENT OF THE APPARATUS FOR INFANTS

FIGURE 6

A different arrangement of the apparatus to eliminate resistance to respiration.

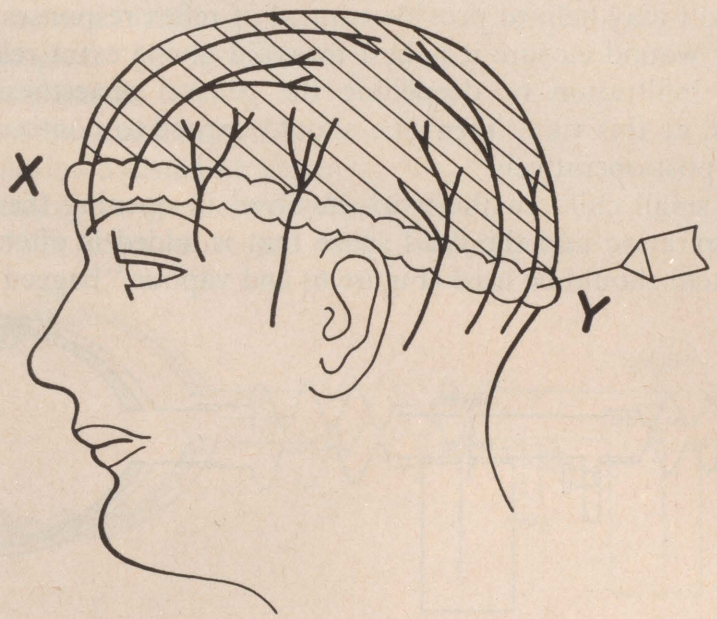
without relation to ventilation. Thus carbon dioxide is reduced and vapour provided without any extra patient work.

To cleanse this apparatus the mask and/or endotracheal connector with the non-rebreathe valve should be immersed in 60°C water (too hot for hand) for twenty minutes, and then dried in the open air.

While trilene is useful when there is an explosion hazard, it may cause rapid shallow breathing and cardiac arrythmia, and if unsupplemented it rarely gives enough relaxation so it is not as versatile as ether.

4. *Conduction Block*

(a) At all times consideration should be given to the possibility of using *local infiltration* as a sole method, or as a supplement to general anaesthesia. Even a partially failed block has an element which is helpful. For infiltration 0.5% Xylocaine is the choice agent. Adrenaline 1/200,000 ($\frac{1}{2}$ ml. 1/1000 per 100 ml. of Xylocaine) is added to various Xylocaine concentrations. As the Xylocaine



SCALP NERVE SUPPLY WITH LINE X-Y
OF LOCAL FIELD BLOCK.

FIGURE 7
Example of a regional field block.

solution is stable, it may be kept on hand with a continuous syringe (Dunn type) for use at any time. In all its injected concentrations the total amount of Xylocaine should never exceed 7 mgms. per kgm. of the patient's body weight—in the very young and old one should try to stay well below this maximum amount by use of

more dilute solutions. Asepsis must be strictly observed with all blocks, and a good skin antiseptic should be used liberally. Patients should be supine during all local anaesthetic injections, however minor the procedure.

Repeated aspiration during injections will avoid intravenous flooding with Xylocaine. However, until the block has vanished a drug reaction may occur, and then assisted ventilation (with oxygen) and a vasopressor may be required. A limited knowledge of regional nerve supply is helpful in some areas (e.g. Figure 7). Where there are endarteries, e.g. digits, penis, then vaso-constrictor drugs or large amounts of solution which may cause pressure on the vessels must be avoided.

Axillary Block

(b) For unlimited surgery on an arm a block of the brachial nerves in the axilla is simple. In the average weight adult 40 ml. 1% Xylocaine is injected around the brachial artery through a point midway between the lowest insertion of the chest muscles onto the arm (Figure 8). During injection the supinated arm should be extended laterally and the elbow flexed at 90°—a 22 S.W.G. size needle will not cause severe artery damage.

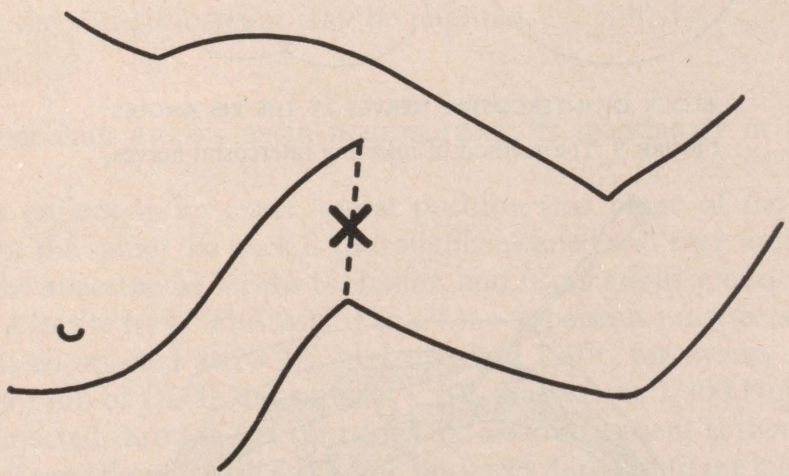
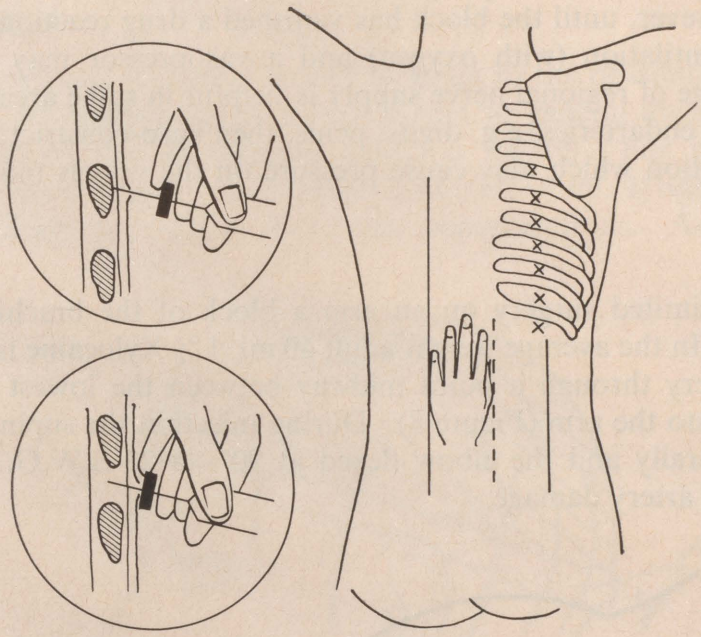


FIGURE 8
The position for injection of the brachial nerves running parallel with the axillary artery.

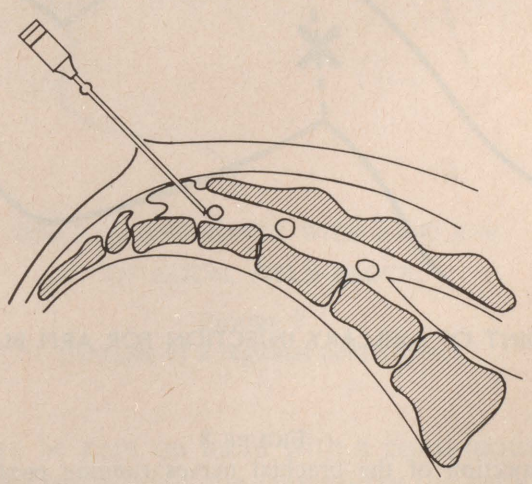
Intercostal Block

(c) For extremely risky abdominal operations an intercostal block may provide the best chance of recovery.



BLOCK OF INTERCOSTAL NERVES AT THE RIB ANGLES

FIGURE 9 The method of injecting intercostal nerves.



CAUDAL INJECTION FOR EPIDURAL ANAESTHESIA.

FIGURE 10 Simple puncture of the sacrococcygeal ligament for caudal anaesthesia.

Thoracic nerves 6–12 are injected with 1% Xylocaine 4 mls. each at the angle of the ribs whilst the patient is in the prone position with the scapulae raised by extending the arms (Figure 9), or the lateral positions may be used to inject one side after the other. A subcutaneous injection is not used and if a rubber needle marker is set at 1 cm. from the skin surface after the rib is encountered, the chance of pleural puncture will be reduced.

Caudal Epidural Block

(d) Using 1.5% Xylocaine and a 2 cm. long 20 S.W.G. needle at about 45° angle with the spine (prone or lateral position) about 5 cm. cephalad from the coccyx tip, the sacrococcygeal ligament can be punctured between the cornuae of the 5th sacral bone (Figure 10). If the land marks are indistinct, blind attempts in the midline can locate the ligament. Only the snap of ligament puncture is felt and the needle is not passed up the canal. Then in an adult 20 mls. injected will fill the sacral canal and 1 ml. extra be required for each dermatome higher, e.g. perinaeum S1 (20 ml.), legs L2 (23 ml.), lower abdominal T10 (27 ml.). Lack of resistance to injection and the absence of posterior swelling are confirmatory signs of the epidural position of the needle point. Always aspirate with the needle bevel in two directions, and if there is marked resistance to injection, slight withdrawal from the sub-periosteum may be required.

Spinal Block

(e) Immaculate asepsis as in neurosurgery, is mandatory in doing a spinal injection.

With the patient in an exact lateral position (the plane of the shoulders and pelvis must be the same) the back is thoroughly cleaned and then arched as much as it can be. The anaesthetist scrubs his hands, and using sterile gloves uses apparatus which is on a sterile tray. This tray with all the apparatus must be autoclaved with the agents to be injected at 20 lbs. pressure, and 120°C for twenty minutes. After subdural tap 1 ml. of 1/200 Nupercaine + 6% glucose (or 1/400 Nupercaine + 5% glucose) is injected through the spinal needle and the patient is then turned supine with a slight Trendelenburg tilt (10°), and his knees flexed until the legs are paralysed. T10 level will be reached in most instances so that operations below the umbilicus are possible. In spinal anaesthesia, as with caudal block, enough nervous paths may become affected that assisted ventilation and/or support of the blood pressure may be needed. Also during paralysis, one should be cautious not to cause injury which the patient will not appreciate till later, or use excessive movement which could give rise to postural hypotension. In carefully conducted series post-operative headaches have been the only common sequelae. It is due to cerebrospinal fluid

leakage, so should be treated with bed rest, hydration, and a caudal injection of 20–30 ml. of sterile normal saline if the headache is persistent.

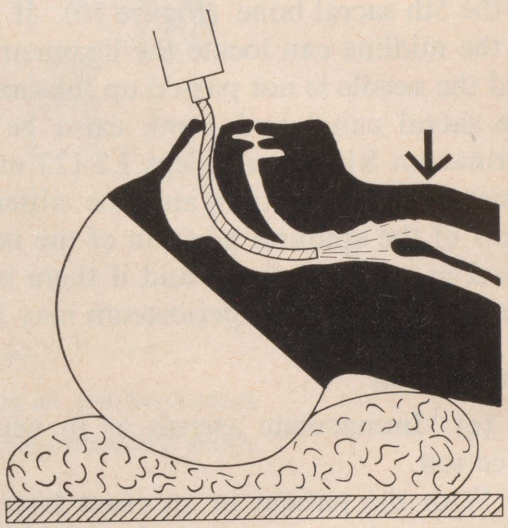
5. *Endotracheal Intubation*

This is best facilitated by using a thorough topical anaesthesia of the pharynx and glottic opening. This may be done in a conscious or a lightly unconscious patient as follows:

(a) 2% Xylocaine with 1/200,000 Adrenaline is used throughout. 2 ml. is put into the patient's nostril first (Figure 11).

LOCAL ANAESTHETIC INJECTED INTO THE PHARYNX THROUGH A NOSTRIL.

FIGURE 11
Injection of local anaesthetic through the nose onto the pharynx.

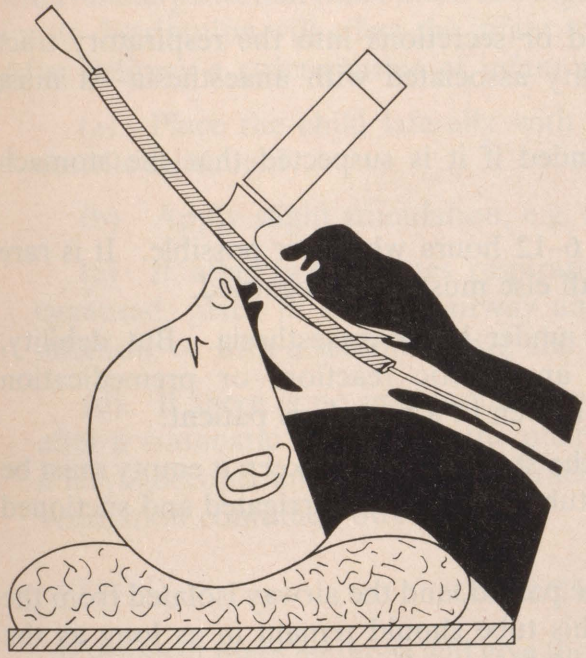


TWO MINUTES LATER LOCAL ANAESTHETIC INJECTED ON GLOTTIC OPENING, VIA A NASO-PHARYNGEAL TUBE, WITH PRESSURE ANTERIORLY ON THE LARYNX.

FIGURE 12
Injection of local anaesthetic via a nasal tube to the region of the glottis.

(b) Two minutes later a soft nasopharyngeal rubber airway is passed through this nostril until the end is just above the larynx, i.e. 5 cms. further on after meeting the nasopharyngeal wall in an adult. 2–4 mls. of Xylocaine is then injected down this tube while the larynx is restrained by anterior neck pressure so that the swallow reflex is delayed (Figure 12).

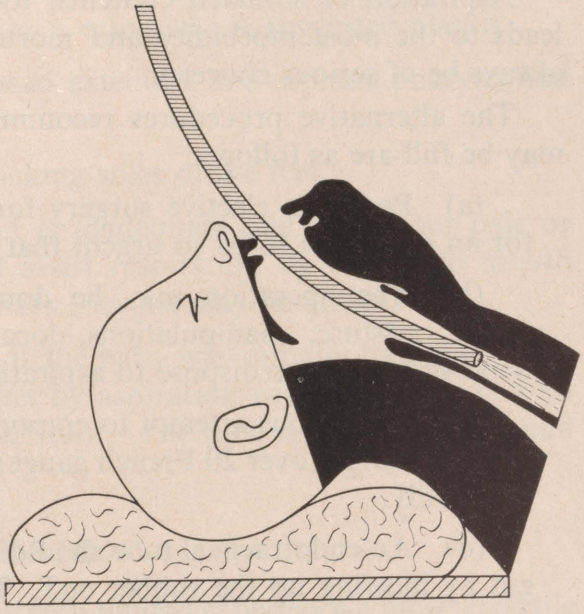
(c) Two minutes later a bite block (a rolled bandage or London Hospital dental prop) is placed in between the left molar teeth and the laryngoscope passed a little to the right of the mouth for a direct view of the larynx. If a pliable director through the endotracheal tube is then placed in front of the aretenoid cartilages (Figure 13) the correct passage can be assured. If the aretenoid cartilages cannot be seen, it is rarely justified to stab blindly in an effort to insert the tube.



TWO MINUTES LATER PASSAGE OF ORAL ENDO-TRACHEAL TUBE WITH DIRECT VISION LARYNGOSCOPY.

FIGURE 13

Endotracheal intubation. (Note: The laryngoscope does not pick up the epiglottis, and the soft director protrudes beyond the endotracheal tube.) The bite block between the teeth on the left of the mouth is not shown.



ANAESTHESIA OF TRACHEAL TREE AFTER INTUBATION.

FIGURE 14

Injection of local anaesthetic through the endotracheal tube into the trachea and bronchi.

(d) Once the tube is passed between the chords, it may be necessary to instill a further 2 ml. of Xylocaine into the trachea (Figure 14), or to deepen anaesthesia to reduce coughing. If the tube's position is not shifted, it is soon tolerated.

To avoid unintentional endobronchial intubation, new tubes should be cut before use to a length equal to twice the distance from the nose tip to the ear lobe. Also the chest should always be ausculated to verify air entry into both lungs.

Continued patency of the tube can be assured by passing a catheter through the lumen at intervals. After cleaning, the endotracheal tubes should be boiled to sterilize them, their shape being retained by placing a wire stillette through the tube.

SPECIAL CONSIDERATIONS

1. *Aspiration*

Aspiration of stomach contents, food or secretions into the respiratory tract leads to the most morbidity and mortality associated with anaesthesia—it must always be of serious concern.

The alternative procedures recommended if it is suspected that the stomach may be full are as follows:

(a) Postpone elective surgery for 6–12 hours whenever possible. It is rare for an operation to be so urgent that all else must be disregarded.

(b) The operation may be done under block anaesthesia. But debility, shock, posture, manipulations, local anaesthetic reaction, or premedication depression may predispose to aspiration even in a conscious patient.

(c) A vigorous attempt to empty the stomach and to keep it empty must be made. A large (over 20 French gauge) tube is passed and irrigated and suctioned repeatedly.

(d) An endotracheal tube should be passed, and the airway isolated from the gut by blowing up the rubber cuff. This tube should remain in as long as the protective reflexes are incompetent, i.e. left in until recovery is complete. The tube can be passed in a conscious patient as described above, the topical Xylocaine lasts thirty minutes, and reflexes will then return.

(e) Other safeguards are rapid induction with head raised and the cricoid structure pressed backwards to occlude the oesophagus; induction, intubation and recovery in the lateral head low position; unobstructed ventilation; avoidance of stomach compression; maintenance in deeper planes of anaesthesia; and the provision of an efficient suction apparatus in all surgical units.

2. *Obstetrics*

Under-oxygenation or fall of blood pressure of a mother can seriously affect the fetus—anaesthetic depression is another concern. Ideally one should assiduously avoid adding these factors to the asphyxial birth process. In critical conditions, local infiltration may be the only safe choice. Other blocks, spinal and caudal, must be meticulously conducted because under-ventilation of the mother, owing

to diaphragm splinting by the uterus, may be aggravated by involvement of the intercostal nerves. Also supine postural hypotension due to the abdominal mass compressing the vena-cava may be dramatically severe, this may be treated by tilting the patient sideways. One should be prepared to ventilate the patient and give vaso-pressors, and recreate a stable state *before* delivery. (Note: Oxytocic agents may complement vaso-pressors to produce dangerously high blood pressures). Intravenous pentothal (maximum 200 mgms.) if followed by a sufficient time interval for its distribution will affect the infant to little extent and greatly assist induction. After delivery a strict routine of infant care is essential. A suggested plan is:

(a) Place the child laterally with head extended and aspirate pharynx and nostrils thoroughly.

(b) Apply slight stimulation, e.g. flicking soles of the feet.

(c) If after two minutes respiration is inadequate and the colour pale or cyanosed, place in an oral airway and assist respiration by mouth to mouth inflation, or with a self-filling bag and mask (ocygen may be added).

(d) If there is no improvement, or if the baby was initially limp and white after a traumatic birth, clear the pharynx by aspiration with direct vision with a laryngoscope, and pass an endotracheal tube. The tube is then aspirated and ventilation continued through it.

CONCLUSION

It is hoped these methods will give those with limited experience a more versatile approach, or suggest some available improvement in their work in anaesthesia. Little explanation is given of these methods which emphasize safety, and it is therefore necessary to receive the advice of a specialist anaesthetist before modifying them in any way.

Further advances in apparatus and agents are now available in favoured parts of the world, but the basic ideas given here should be of value whatever the ultimate choice of anaesthetic methods in the future.

TOTAL VERSUS SUBTOTAL HYSTERECTOMY

J. R. GOODALL, M.D.
Montreal, Que.

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TOTAL VERSUS SUBTOTAL HYSTERECTOMY*

A Clinical and Technical Study

J. R. Goodall, M.D., Montreal, Que.

THIS is merely an expression of opinion based upon personal experience arising out of 550 consecutive cases of hysterectomy, about equally divided into the two categories. The two series are as alike as possible in every circumstance and antecedent. In the first half of the series, the subtotals markedly preponderate. As my experience and finesse improved, the totals greatly outnumbered the subtotals, so that for the past year, the total hysterectomies constitute about 90 per cent of all my hysterectomies. The uterine pathology included all the common diseases. Many were complicated by appendage troubles of almost every variety. The uterine preponderant diseases were fibroids, fibrosis, corporeal malignancy, advanced cervical disease, chiefly in the third and fourth decades, and states of pelvic allergy associated with uncontrollable uterine hemorrhage. There were only five cases of cervical cancer, after treatment by radium, in which hysterectomy was deemed advisable.

I wish to state most emphatically that it is fartherest from my intention to force the unskilled gynecologic surgeon into an operation, graver than that for which he is fitted by experience. But to the skillful surgeon, to whom a slightly longer operation presents no greater deterrent than the extra time that is expended, the total hysterectomy, *caeteris paribus,* will present great advantages to the patient, as well as great surgical satisfaction.

There are advantages and disadvantages to each of the two types of hysterectomy.

The disadvantages of total hysterectomy are:

1. Greater time expended at operation.
2. Greater skill required.
3. Greater blood loss.
4. Greater danger to vital organs.
5. Greater difficulty if the pelvic organs are fixed deeply in the pelvic cavity, or if patients are obese.

The advantages of total hysterectomy are:

1. Fewer immediate postoperative complications.
2. Fewer remote sequelae.
3. Smoother recoveries.

*Read at the Sixty-First Annual Meeting of the American Gynecological Society, Absecon, N. J., May 25 to 27, 1936.

The disadvantages require no further elucidation. I believe they will receive general acceptance. On the other hand, the advantages may require considerable discussion.

The average difference between a subtotal and total hysterectomy is between five and fifteen minutes, a negligible factor in the average case. The skill required is merely that derived from experience, preferably primarily derived from one who has perfected the technic, with an accurate knowledge of the relative and absolute propinquity of the vital pelvic structures, and care in avoiding undue tension upon tissues. Tension is one of the greatest deterrents to forming union, and one of the most frequent causes of postoperative pain. The great onus of care in these cases surrounds the invisible ureters. To some they become a mental bogey. A ureteral complex, you will excuse the term, is one of the worst of emotional acquisitions in the surgeon. Some men seem to be unable to avoid them, and their mental stress to go wide of them seems to act like a lodestone.

To the best of my knowledge, I have never injured a ureter or bladder in hysterectomy. That does not mean that it has not happened, but merely, if it did, it was not only unobtrusive, but not even recognizable when sought. Operative bladder injuries are rarer than ureteral complications, in the experience of the general run of gynecologic surgeons. It is my experience that it is extremely difficult to injure the bladder when it is empty and contracted. It is one of the most elusive organs.

The difference in the blood loss in the two types of operation may be negligible. In the ordinary run of cases it is so. But it can be considerable, especially in hemorrhagic cases, or when a clamp or suture fails in its requirements. A word of caution here may not be amiss. A careful preoperative inquiry into the patient's history, as to untimely or copious uterine hemorrhages, nosebleeds, migraine, and familial history of vascular instabilities, may make one cautious in carefully suturing raw surfaces completely, and insisting upon a most thorough hemostasis. Where the history is at all suggestive of a hemorrhagic state, the usefulness of a preoperative blood transfusion and a repetition of the same on the seventh day after surgical intervention will obviate the serious oozing at operation, and prevent late postoperative bleeding. It is a singular fact that vascular unstables are prone to develop an advanced state of hemorrhagic tendency about the eighth day. This, though frequently occurring as an expression of a low state of sepsis, is also a sequel of low platelet count at that period, and, in most cases, can be forestalled by a carefully matched transfusion. These cases should be matched for three-quarters of an hour, and any abnormality should be guarded against. These nonseptic hemorrhagic cases are prone to develop wheals at the point of any hypodermic injection, and other signs of skin irritability. A special paper is being devoted to this important subject. Should the secondary hemorrhage be due to sepsis, it will occur

usually between the eighth and twelfth day after operation. The sepsis is frequently ignored, because the temperature seldom rises more than a fraction above 99° F. and is usually continued over several days. It is in just these types of septic cases that either blood or vessel, or both, becomes so altered by the bacterial toxin, that diapedesis or rhexis occurs. Hemorrhagic states, as previously stated in my monograph on puerperal infections, rarely occur in acutely septic cases, but are most common in the continued cumulative low-grade infections, which do not rouse the body to the formation of antigens. When vaginal hemorrhage occurs, and it has occurred in my series of total hysterectomies in 2.25 per cent of cases, it has always been in these two types of cases: the vascular unstables, both septic and nonseptic; the vagina should at once be cleared of clots and packed gently with gauze with an uterine packer. To leave the vagina filled with clots is tantamount to inviting further bleeding. In every instance, this procedure, preceded by a transfusion, has promptly arrested the bleeding.

Generally speaking, total hysterectomy is easier in the parous than in the nulliparous, due to the greater mobility of the uterus, owing to the lengthening of its ligaments. Low set, fixed uteri may present unusually difficult surgical circumstances. Particularly is this so in cases of subacute or chronic pelvic inflammatory disease. But the difficulty, when overcome, brings a sense of great satisfaction, for it is in just these types of cases that it pays to remove the cervix with its susceptible mucosa.

About 70 per cent of patients who have total hysterectomies void spontaneously after operation, as against 45 per cent of subtotals. This circumstance, confirmed by all my house surgeons in the four different hospitals, seems contrary to all the laws of probability. An explanation has been sought, but nothing satisfying has been forthcoming. Is it that in the subtotal only, certain branches of the autonomic nervous system are severed, and that others are thereby thrown out of normal function; whereas, in the total hysterectomies, the majority, if not all, of the autonomic nervous system to the bladder is severed, causing the bladder, therefore, to become an automaton without sympathetic nervous control? In our present imperfect knowledge of autonomic vesical control and autonomic vesical distribution, it is quite impossible to vouchsafe anything but theories. Consult any work upon the results of section of the sympathetics upon vesical control, and one is led into a perfect orgy of contradictions and confirmations among the experimenters. The low percentage of spontaneous bladder evacuations in subtotals has been explained, by one of my colleagues, upon the basis of an inflammation spreading from the retained cervical stump to the bladder, an explanation that seems quite inadequate for many valid reasons.

Primary hemorrhage has never occurred in any one of the 550 cases, so that in this respect they are equal. Thrombophlebitis is a much more

common complication in subtotal hysterectomies. The comparison is most striking. The percentage was three to one.

In all clean cases, thrombophlebitis may be taken as an index of a man's technic, both in obstetrics and in gynecology. Let this be most emphatically stated. So many, whether from ignorance or self-deception, assume that thrombophlebitis is an unpreventable misfortune, quite beyond their control. There is a ready explanation for the preponderance of thrombophlebitis in subtotals. It is to be found in the cervical mucosa. Just a few lines about pelvic thrombophlebitis. It is rarely diagnosable, except in its secondary complications. It is rarely acute, and most dangerous when of low-grade infection, or in its defervescence after an acute or subacute attack. The general agent of thrombophlebitis is an infection of low virulence and in the vast majority of cases it emanates from a mucosal disease. In this respect, the mucosa of the cervix fills all the requirements. Hence the relatively higher percentage of thrombophlebitis in subtotals, as against totals. The aseptic technic in both operations in my series was essentially the same, so the higher percentage must have been due to an agent residing in the tissues. And now, when upon the subject, it may be opportune to stress something which has not received sufficient attention: inflammatory diseases run very different courses, depending upon the tissues in which they are placed. Infections in mesoblastic and ectodermal tissues usually run a purely local and rapidly conclusive course, or a virulent and rapidly critical stage with an equally rapid cure. Restoration to normal function is usually complete in proportion to the rapidity of the disease, provided the patient survives the storm. In mucosal diseases, however, the great majority are subacute or chronic from their inception. Those that are not tend to become so eventually, and the tendency for the disease to linger indefinitely is the rule. This tendency leads eventually to two distinct changes, hyperplasia or hyperfunction, or both. The virulence dies down, and the corporeal reaction is then seldom sufficiently active to effect a complete cure. Thrombophlebitides are preponderantly mucosal in origin. In the cervical mucosa we have a classical example of these changes, and the organisms of the cervix are ready, in their attenuated form, to become the active agents of thrombophlebitis. That these organisms are still markedly pathogenetic can be clearly shown by the frequent incidence of secondary septic hemorrhage, when cervical amputations and suchlike operations were the vogue. Many of us in those years saw women, ostensibly in the bloom of health, submit themselves to cervical operations, only to be found on the 8th to the 12th day almost at the point of death, through septic hemorrhage. Why? Because we amputated through diseased mucosa. Why the delay to the twelfth day? Because the organism was semivirulent. Many of these cases developed pelvic thrombophlebitis and succumbed to its accidents. Mucosal disease is one of the greatest

menaces to primary union. Can this mucosal menace be removed by cautery? That depends. In many cases, the disease is limited to the mucosa proper, an endocervitis. In others, the organism is invasive, causing a large, hard, cystic cervix, a cervicitis. In still others, there may be combination of both.

In cases of simple endocervicitis, coning out the cervix and cauterizing the residual portion of the canal may be effective. In the cervicitis types, cauterization, except it be very deep and destructive, is more harmful than no cauterization at all. The ideal remedy is total removal, when the skill and experience overbalance the risk.

Drainage was not used at all in any of the cases, and complete closure is a fixture.

Subtotal hysterectomy is often disappointing in its late sequelae. In a goodly percentage of my cases a leucorrhea developed, which did not exist prior to operation. In many cases, endocervicitis and ectropion developed after operation, necessitating treatment. This invariably leads to disappointment in the efficiency of operation. Both the above changes, I believe, can be attributed to the abnormal status of the cervix. Doubtless, in subtotal operations, the cervix must suffer changes in nutrition and nervous impulses, which eventually lead to a state of pathology.

That these abnormal changes are in part dependent upon the menstrual flux is shown by the infrequence of these abnormal developments when both ovaries are removed with the body of the uterus. In five of my patients, there were very troublesome cervical hemorrhages, that came on some months after subtotal hysterectomy. Four of these were operated upon for continuous metrorrhagia, that resisted every form of treatment; none of them showed any appreciable pelvic pathology, either by bimanual examination, or at the time of operation. Two of them had two sterilizing series of deep x-ray, with recurrence of hemorrhages after a period of amenorrhea. One had radium once, and three series of deep x-ray in doses estimated sufficient to sterilize, at least temporarily, if not permanently. It is my practice to cone out the cervix in all subtotals, and I feel very confident that no endometrium had remained, unless it had invaded the domain of the cervical mucosa. One is inclined to believe that, in some of these cases, vicarious menstruation is set up. This is a very logical conclusion, when one considers that the genital canal, from the fimbriated end to the hymen, is developed from a common "anlage," yet it exhibits four variations of epithelium, and four widely different functions. And when we consider that the endosalpinx, endometrium and vagina respond to the menstrual cycle, it would be extraordinary did not the cervix show a similarly periodic change.

In one such interesting case, a woman at 42 first came under my observation. She had had a subtotal hysterectomy for fibroids, five years previously. For the past

year she had regularly periodic discharges of blood, simulating menstruation, every three weeks. She was an intelligent English woman, who said that the blood was unlike menstruation in that it came on and stopped more slowly than normal menstruation. She came fearing cancer. Nothing abnormal could be found on examination. Operation for other causes had to be done, and the stump was removed at the same time. It showed no trace of pathology. It is necessary but to mention the incidence of carcinoma in the stump of a subtotal hysterectomy. The assurance to a patient that cancer of the uterus is an impossibility after total hysterectomy is a comfort to the patient in which the surgeon rightly shares. Cancer of uterus is a constantly recurring fear in the emotional life of every woman of postmaturity.

It is not a matter of indifference whether one allows the ovaries to remain. With their removal, the incidence of late cervical pathology is greatly reduced; the incidence of immediate and intermediate postoperative hemorrhage is reduced, especially in vascular unstables; and the late return of bleeding from the stump is practically unknown after total removal of the ovaries, except in cases of newgrowth. There is not the slightest doubt that ovulation, estrin and progestin formation keep the tissues in the pelvis in a constant state of flux, with varying nutritional changes, rendering them much more susceptible to changes bordering upon, or invasive of, the domain of pathology.

My final plea is, if one's skill and experience warrant, if the case is a suitable one, a retrospect of cases shows that the total hysterectomy has much to commend it, above a subtotal. Where, on the other hand, the surgical risk, arising either in the surgeon or in the patient, would make the immediate risk the greater, then pursue the course that your conscience will dictate. But do not stop there, as regards the welfare of your prospective cases. Make yourself as proficient in the total as in the subtotal. It can be done. The mortality in this series is nil. The rest is in the lap of the gods.

TECHNIC OF TOTAL HYSTERECTOMY

The vagina is cleansed with sterile liquid green soap, and thoroughly dried. Care is exercised to empty the bladder completely.

Catgut, of the plain variety, chiefly Davis and Geck product, was used throughout. No. 2 was used for all major ligature purposes (never any larger size), and No. 1 for all peritoneal work. A minimum of catgut in each case was used. In over 90 per cent of cases, six No. 2 ligatures were sufficient. These were, ligation of the infundibulopelvic ligaments or the uteroovarian broad ligament, depending upon whether the appendages were to remain, or not. The next two ligatures are upon the lateral upturn of the uterine arteries, and the last two are upon the lower uterine branches, as far down as the fornices. The vault of the vagina is sutured continuously in two layers, with single No. 1, or No. 2, catgut as described later, and the peritoneum of the pelvic floor is united always by a single continuous No. 1 suture. Curved noncutting needles are used throughout, except when sewing the cervical stump in subtotal hysterectomy. I cannot overemphasize the importance of this little precaution. Round needles transfix. Cutting needles make a buttonhole and cause much unnecessary bleeding.

OPERATION

The abdomen having been opened, the patient is placed in an exaggerated Trendelenburg position. The intestines are then packed off, and the pelvis explored. In the uncomplicated cases, one determines at once whether the appendages are to be removed or not. If these are to be removed, they are held up by the hand of an assistant or by the operator, and the broad ligament *''clear space''* is located. This is a space below the ovary, bounded internally and outwardly by the uterine vessels. It consists merely of two layers of peritoneum, without any appreci-

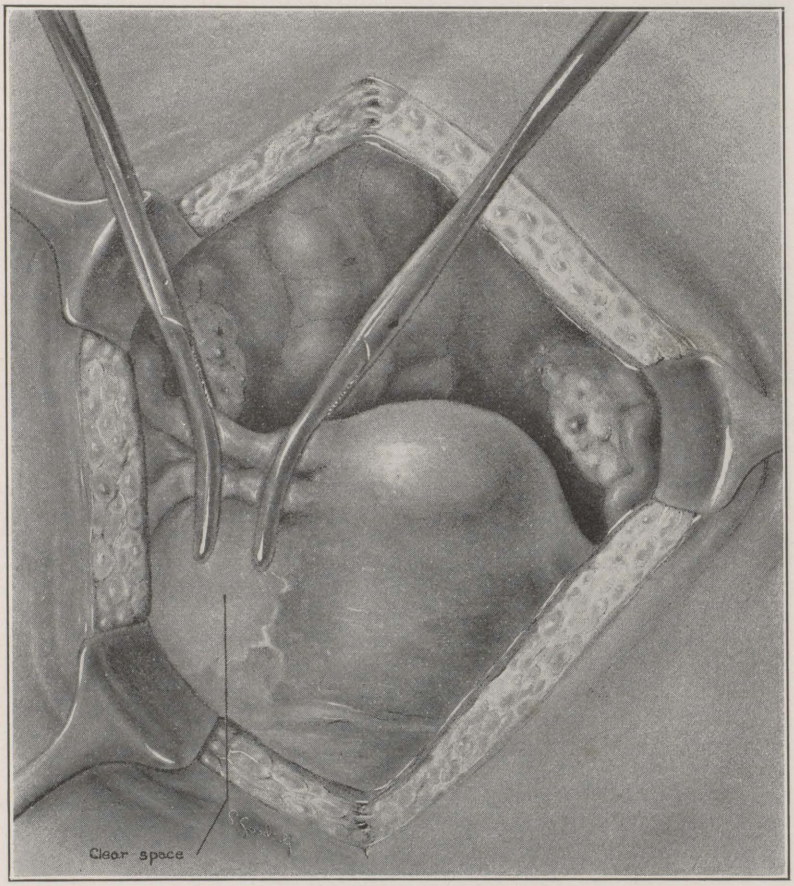

Fig. 1.

able tissues between them. If the finger impinges upon this area from behind, the digit will shine through anteriorly, or if the broad ligament is held up when tissues are lax, the space is translucent. A ''Kelly'' curved clamp (Fig. 1) is now placed upon the infundibulopelvic and round ligaments, having its tip in the clear space, and its convexity inward. A second and similar clamp is now placed upon the broad ligament between the ovary and uterus, with its tip juxtaposed to the tip of the first clamp. The appendages on that side are now completely isolated vascularly. In ordinary cases, the second clamp will include the proximal end of the tube, uteroovarian and round ligaments. This second clamp should be placed with its convexity outward. The appendages are now excised, being careful to leave at

least a quarter of an inch of tissue protruding through the forceps at the cut margin. This is to prevent slipping. The Kelly forceps should have longitudinal rugae along the whole blade, except at the tip, where they are corrugated, to prevent slipping.

The infundibulopelvic and round ligaments are now ligated by a figure of eight, which should transfix the round ligament, then through the "clear space," and again transfix the round ligament in the opposite direction. Ligation is then completed. The clamp should be released slowly as the ligature is tightened. I cannot

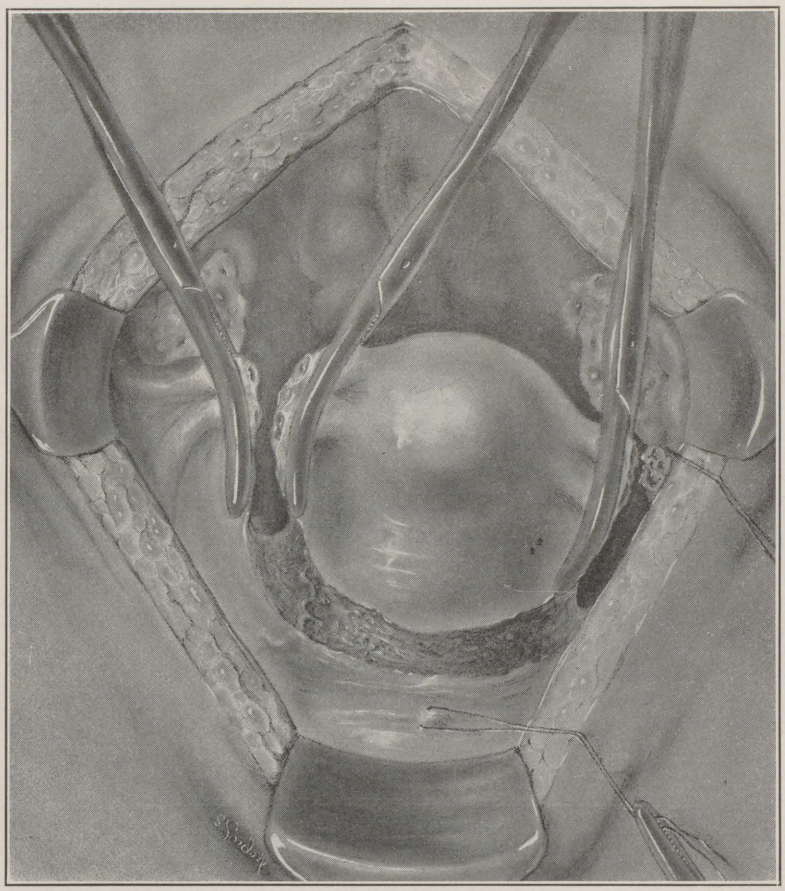

Fig. 2.

overemphasize the importance of this little procedure. Too quick release of the clamp, specially when dealing with the uterines, is inviting trouble, by allowing the tissues to escape from the grasp of the ligature.

The infundibulopelvic ligature is left long, and held by a forceps. On the opposite side the appendages are to remain. The "clear space" is again defined. The ovary and round ligament are pulled toward the pelvic wall and the first Kelly forceps is placed over the broad ligament, proximal to the ovary. Its tip should be in the center of the "clear space." Its convexity should be inward. A second is placed parallel to this, separated by one-third to one-half inch, with its convexity outward. These clamps include, in uncomplicated cases, tube, uteroovarian

and round ligament. Incision and ligation is now performed as described above, being careful to transfix the round ligament. There remain now but two Kelly clamps, which act as tractors upon the uterus. It is never necessary to grasp the uterus with volcella or any other form of tractor. These cause unnecessary loss of blood and tearing of tissue.

By its tractors the uterus is now pulled up and backward by the assistant, and the vesicouterine pouch exposed, by an anterior retractor. The anterior leaf of the peritoneum is now picked up on the left side at the vertex of the incision in the "clear space," and with a curved scissors, the peritoneum is incised in a curved

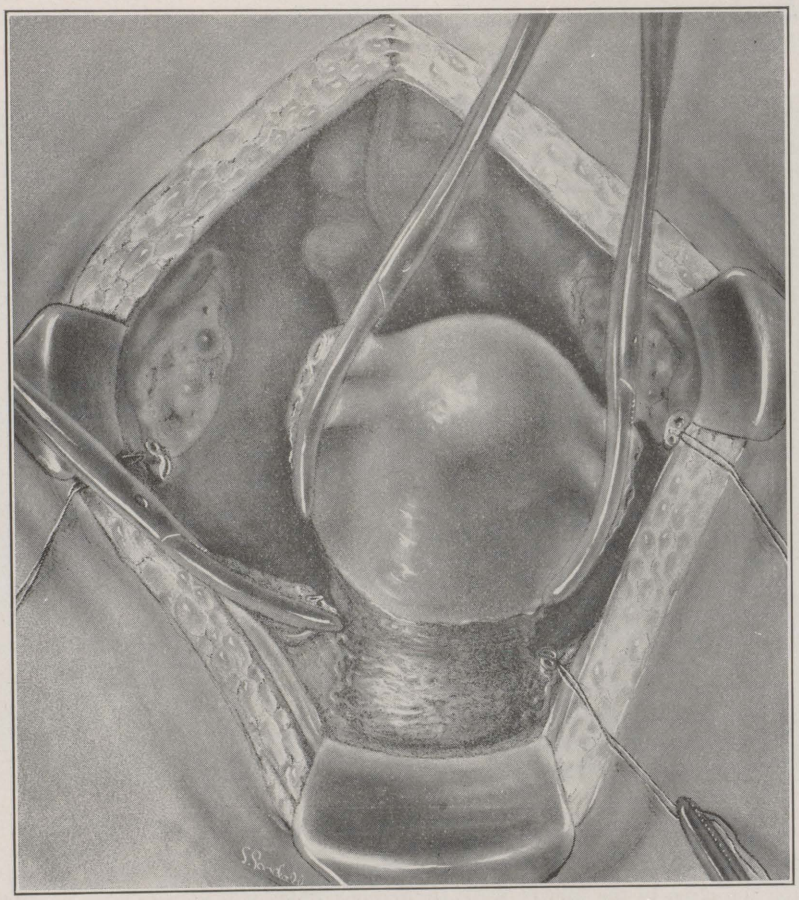

Fig. 3.

line in the vesicouterine peritoneal reflexion. The incision should end in the anterior leaf in the right "clear space." The operator now takes hold of the tractors with the left hand, and, with the index finger covered with one layer of sponge, the bladder is pushed down from the uterus and cervix. A No. 2 catgut now is made to transfix twice the bladder muscularis and vesical peritoneum (Fig. 2). This is not tied, merely held by an artery forceps. Traction is now put upon it by the second assistant, and a long special retractor measuring 2¾ by 4¾ is slipped below the reflected bladder, and the whole anterior wall of the uterus is now exposed. The uterus is again held by the operator and the separation of the bladder is now completed by the sponge-covered finger, down to the junction of the upper and middle

thirds of the vagina. An additional precaution lends a great deal of comfort. This consists in pushing the garnished finger well out on either side of the cervix and vagina, to separate the bladder from this region and to displace the ureters outwardly. A "Kocker" forceps with museux tip is now placed upon the upper uterine artery, at right angles to the uterine wall (Fig. 3). Its tip should impinge well into the uterine muscularis. Ordinarily it should be not less than ½ inch and not more than one inch above the portio. This can be easily determined by a finger of one hand in the anterior, and another finger posterior to the vagina. A similar clamp is placed on the opposite uterine at the same height. The artery is now cut ¼ inch from the clamp margin on both sides. In cases of large uterine tumors, it is neater to place on each side, a second similar clamp ½ inch above the first ones, to prevent a large escape of blood into the pelvic cavity. With a curved round needle the

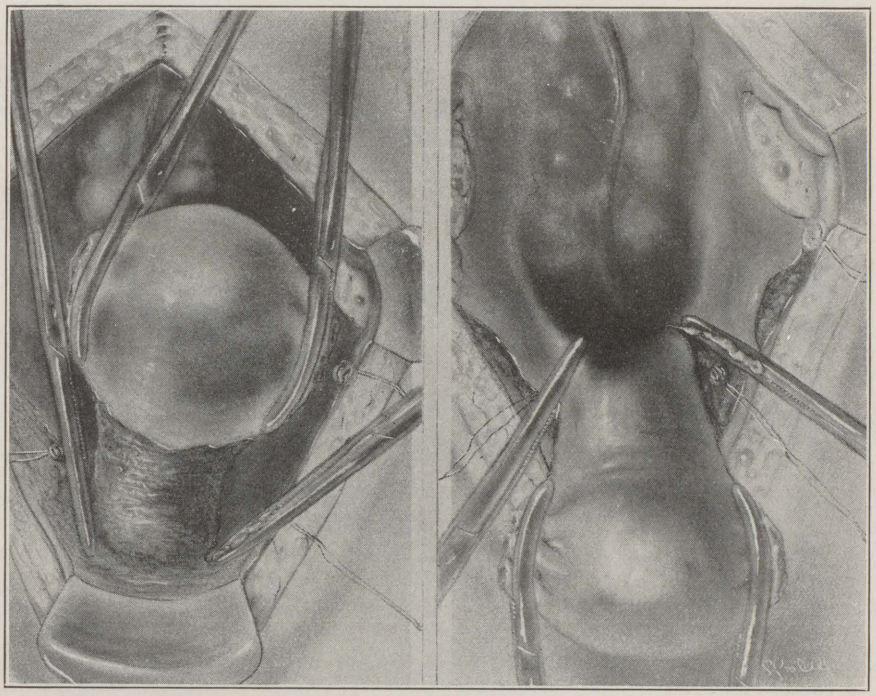

Fig. 4.

tissues are transfixed just below the forceps. The needle should not be driven through too close to the uterine wall, for reasons which will appear later. Ligation is now completed on both sides, and the clamps are removed.

The next step is securing the cervical branches of the uterines. Proceed as follows. First make sure that the bladder is clear, as above described, and held well out of the way of the cervix. Hemostasis should be complete. With the tractors pull the uterus up and out of the incision, and toward the symphysis, and expose the pouch of Douglas. Define the uterosacral ligaments (Fig. 4). Have your assistant pull the ligature on the upper uterine outward. Span the lower uterine with a "Kocker," placing the tip of the posterior blade mesial to the uterosacral of that side. Having made this impression posteriorly, turn your attention to the anterior blade. See that it is deep enough to go beyond the fornix. Then close it tightly on the cervix. Repeat the process on the opposite side. Care in placing these

clamps ensures safety to the ureters and bladder. The assistant now takes the handle of this forceps and draws outward and the operator cuts with a scissors boldly down the broad ligament, cutting always upon the cervix so as to leave one-fourth of an inch of tissue beyond the forceps on the cut side. The fact that you have reached the fornix is announced by a hollow sound quite different from that of the cervical tissues when severed by scissors. The process is repeated upon the other side of the cervix. When properly placed these last two straight clamps should converge at their points. The tissues uniting the lateral fornice to their broad ligaments are very loose and relatively bloodless, so that a portion of the lateral wall of the vagina can usually be freed, to equal that previously attained on the anterior wall. The assistant now holds the handle of the ''Kocker'' outward, the fornix of the vagina is now grasped with an ''Allis'' forceps on each side, the vagina is transfixed with a scalpel anteriorly, and the lower margin of the cut surface is caught with an Allis forceps. The circumcision of the vagina is now completed, above the two Allis forceps on the fornices (Fig. 5). The posterior vaginal wall is also grasped with a

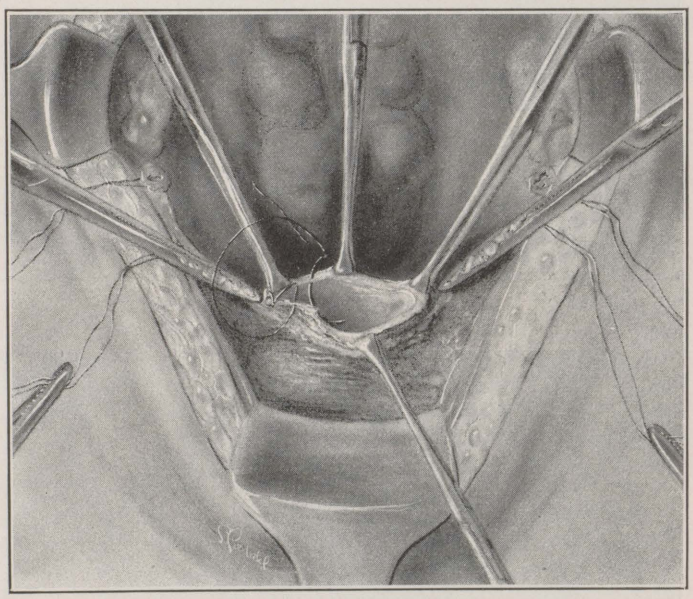

Fig. 5.

similar forceps. The vaginal vault is now defined by these four Allis forceps: one anterior, one posterior, two lateral. Any vessel in the paravaginal tissues (usually 'on the posterior wall) is caught within the grasp of an Allis. It is recommended that hemostat crushing forceps never be used where an Allis or other toothed non-crushing forceps will better serve the purpose.

It is never necessary to ligate the vaginal vessels. A continuous single No. 1 or No. 2 plain catgut (it should be long) is now used to close the vagina. One should begin at the patient's right, transfix from behind forward, and tie the first suture below and outside the Allis on the right lateral vault. The end should be held. Then continue across the vagina, being careful to close the left fornix completely. This suture should incorporate only the vaginal mucosa. Having reached the vicinity of the tip of the left Kocker, which still holds the lower uterines, the operator places a second layer over the vault, with the same continuous suture (Fig. 6). This should include posteriorly the intermediary tissues between the vaginal mucosa and peritoneum of the Douglas' pouch (including the latter, if so desired), and anteriorly

usually a well-defined cut layer of fascia between the base of the bladder and the vaginal wall. This is important in fixing the bladder from a sliding prolapse. The suturing should laterally stop short of the tips of the Kockers. The vault closure is now complete with the tying of the two ends of the continuous suture.

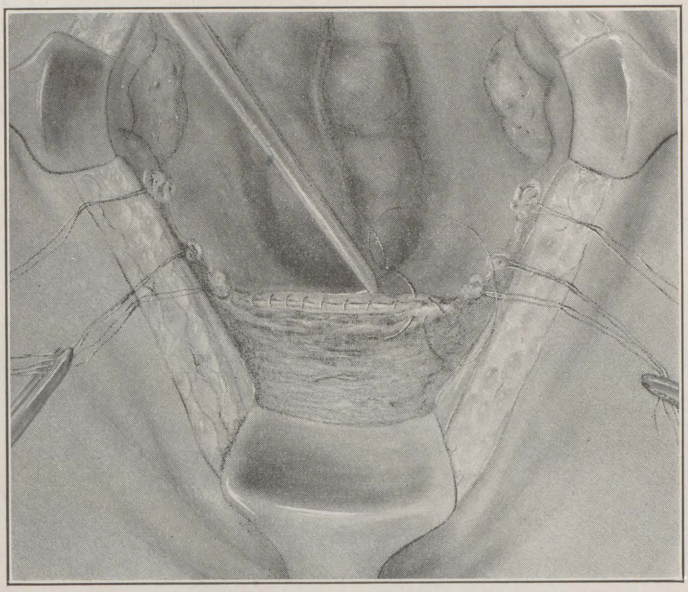

Fig. 6.

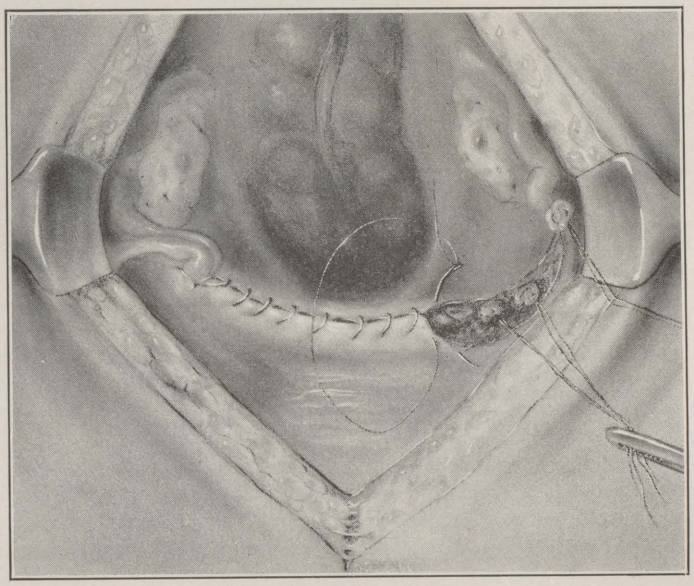

Fig. 7.

The lower uterines are now tied by transfixing the broad ligaments, just exterior to and slightly above the tip of the forceps, to insure that the ligature will remain distal to the forceps. The ligature is now looped and tightened down to the upper

margin of the tissue within the grasp of the forceps, as the handle of the Kocker is held toward the median line. The assistant now pulls the *handle* of the Kocker outward and the *tip* inward, and when so held, the operator tightens the suture, and, upon his dictum, the ligature is tightened synchronously as the assistant slowly releases the clamp. This ligature is inside the one previously placed upon the upper uterine. As a further precaution the ligature upon the lower uterines may now be tied over the ligature of the upper uterine ligature, in Staffordshire fashion. I have abandoned this method as unnecessary and productive of more knots for internal digestion.

Hemostasis should now be complete. The pelvic peritoneum is now closed as follows (Fig. 7). This is the most dangerous part of the operation. It is in this procedure that damage or ligation of the ureters most frequently occurs. I have catheterized the ureters in many cases, to demonstrate this. No. 1 single continuous is used. The posterior leaf of the clear space on the right is transfixed, then the suture is carried through the stump of the infundibulopelvic ligament (if appendages have been removed), or of the broad ligament (if appendages remain) internal to the primary ligature, and then it should pick up the layer of the anterior peritoneum beyond the round ligament. The assistant now pulls the ligature inwardly and one ties over the stump. The primary ligature and the single end of the continuous are now cut. One continues inwardly from right to left, closing posterior and anterior layers of peritoneum. As soon as one approaches the ligated uterines, one must proceed with greater caution. It is just here that the ureter emerges from among the uterine vessels and is covered only by a layer of peritoneum. Care should be taken that only peritoneum is included in the suture bite on the posterior wall. Once one has got past the right uterine ligature, one can proceed with impunity until the left uterine ligature is reached, where similar precaution is necessary. The left stump of the broad ligament or infundibulopelvic ligament is treated in a manner similar to the right. This line of suture draws the lateral supporting structures into a firm band of support in unison with the vaginal vault. The pelvic peritoneum has now been completely closed, and should be quite free from any exposed raw surface. Peritoneal toilet and closure of the abdominal wound complete the operation.

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CIRCUMCRESCENT AND CIRCUMVALLATE PLACENTAS*

JAMES ROBERT GOODALL, M.D., MONTREAL, QUE.
(From the Royal Victoria Montreal Maternity Hospital and Department of Gynecology and Obstetrics, McGill University)

PLACENTAL circumcrescence is an outgrowth of placental villi beyond the boundaries of the chorionic plate. Circumvallation is an added complication to the former condition whereby there is "ditching" at the chorionic circumference. This complication occurs only in about 10 per cent of cases of circumcrescence.

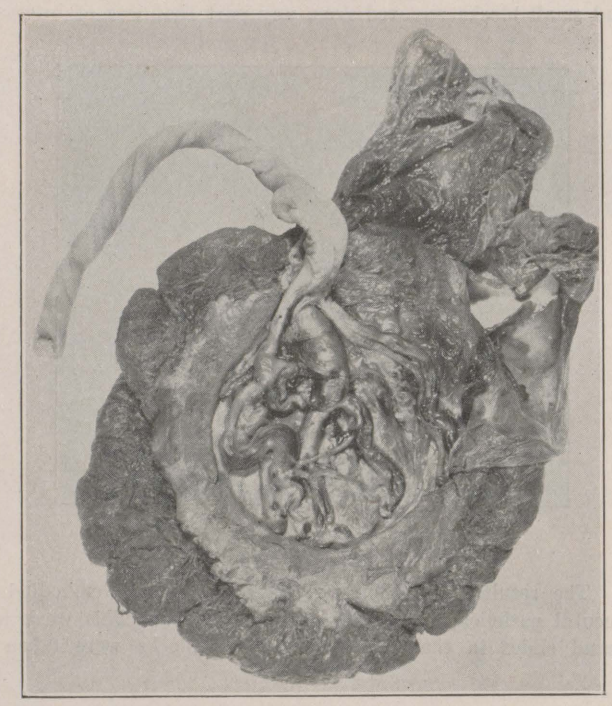

Fig. 1.—Concentric circumvallation.

Clinically, placental circumcrescence has a very fixed value, and anatomically the cases can be divided into two distinct, easily distinguishable groups: (1) concentric, and (2) eccentric or crescentic.

These two are similar in their mode of development but differ somewhat as to causation. The former, concentric circumcrescence, is a product chiefly of the first trimester of embryonic life; the latter, eccentric circumcrescence, of the second and third trimesters. Concentric circumcrescence takes at least two months to develop, after

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the cause is in operation. The developmental period of eccentric circumcrescence is not definitely known, though probably of the same duration. Both types are an expression of compensatory hypertrophy.

METHODS

This work is based upon a critical examination of 750 placentas. The work will eventually comprise over 1000. The specimens were carded and immersed in 10 per cent formaline solution immediately after birth. They were all examined critically under running water, within a few hours (from eight to twenty-four) after immersion, and any abnormal characteristics were noted. They were then labeled with name and date and reimmersed in a fresh solution of formaline and preserved in jars for from four to six weeks. After a thorough rinsing, they were then sectioned in parallel slices, 1 cm. thick, by means of a large amputation knife on a block. These hardened slices were then critically studied with a large magnifying lens, and small blocks removed from any part that was of interest for microscopic study. The results have been startling and will be embodied in a monograph on placental pathology to appear within the year. This work will comprise several thousand slides in that from two to ten blocks were taken from each placenta.

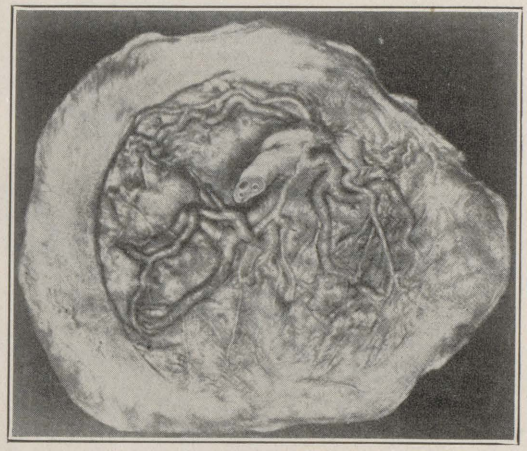

Fig. 2.—Eccentric circumcrescence.

MECHANISM OF PLACENTAL CIRCUMCRESCENCE

It would serve no useful purpose to review the history of research upon the circumvallate placenta. This was done so thoroughly by Dr. J. Whitridge Williams in 1929 that nothing could be clearer or more concise. In this work, Dr. Williams enumerated ten different theories, expressed his own criticism of each one, and finally elaborated his own, which was closely in concurrence with that of Meyer, 1909, who pointed out that the abnormality is the result of an originally too scanty development of the chorion frondosum. The fact that so many different views are held only goes to prove how imperfect is our knowledge of circumcrescence and circumvallation. In studying

the subject it will tend to clarity if we divide the work into three parts: I. The developmental relation of the chorion frondosum to the chorion laeve; II. The interrelation of the chorion frondosum and placental site; III. The relation of the chorion frondosum to uterine distension.

I. THE DEVELOPMENTAL RELATION OF THE CHORION FRONDOSUM TO THE CHORION LAEVE

At one stage of embryonic life, the whole embedded amniotic sac is covered with fluffy villi (Fig. 3). Later on, the growth of the villi becomes accentuated at a certain spot, usually situated proximal

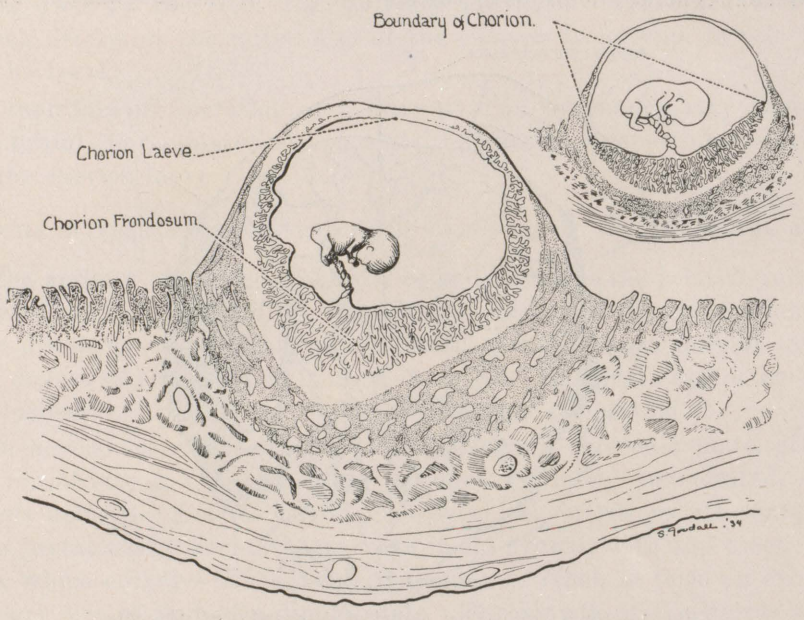

Fig. 3.—Diagram showing relation of chorion laeve and chorion frondosum.

to the uterine wall (Fig. 3, inset). This is the site of the future placenta and is spoken of as the "chorion frondosum" to distinguish it from the extra placental chorionic membrane, which is known as the "chorion laeve," upon which the villi atrophy. The fetal surface of the normal placenta is known as the placental plate and has very definite limits. The size of the placental plate is determined by many factors working either in collaboration or at cross purposes. First of all, it has been stated by one of the greatest authorities on embryology, that 50 per cent of the ova of the animal kingdom are defective. This may be inherent in the ovum before impregnation and may arise out of any one of many maternal causes, chiefly (1) endocrinologic, either hereditary or acquired, and (2) pelvic disease. To these may be added defects operating after impregnation and arising out of

diseases inherent in the uterine mucosa, which may also be primarily endocrinologic, or developmental (and therefore secondarily endocrinologic), or due to local or general constitutional diseases. These fetal and maternal factors working alone or in combination in early embryonic life determine the normal or abnormal activity of the trophoblastic layers, comprised of the syncitial and Langhans layers with their supporting mesoblastic framework. These, by their operation in early fetal life, determine the size of the chorionic plate, or chorion frondosum. It has been possible to demonstrate microscopically the widest divergent ranges of activity in these cells in different embryos of about the same clinical age—embryos removed therapeutically for maternal contraindications to the continuance of the pregnancy. These

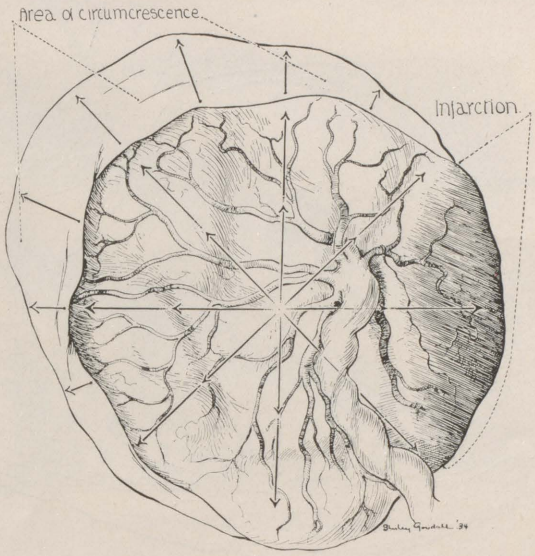

Fig. 4.—Lines indicating centrifugal growth of placenta.

specimens were therefore fresh and were hardened immediately upon removal. This same difference in trophoblastic activity was also noted independently by my colleague, Dr. J. P. Kearns. These factors, fetal and maternal, enumerated above, determine not only the size of the chorion frondosum, but also the possibility of the gestation reaching viability. When atrophy of the villi beyond the boundaries of the chorion has taken place, no matter what the needs of the fetus may be, regeneration of these can never occur. Once the relative dimensions of chorion frondosum and chorion laeve are fixed, they are fixed for all time. In other words, the limiting circumference of the chorionic plate becomes immutably set. In circumcrescence, though the subtended fronds may grow beyond the normal limits of the chorionic plate, their roots are always within the plate circumference.

Trophoblastic activity is an expression of the correlation of forces inherent in ovum, uterine bed, and nutrition. How like all other forms of life! They all find their determining characters in inheritance, soil, and food. Trophoblasts, acting under the foregoing influences, determine the size of the future placenta. Where all conditions are average and normal, the resultant placenta will be of average size. In oval dystrophy and maternal mucosal incompetence, a very large, poorly developed chorion frondosum may be the result, and *per contra,* when food supply and other conditions are in excess, the needs of the fetus may be adequated by a small, active, initial placenta. This excess of nutrition may be the expression of overactivity of the trophoblastic cells in producing excessive maternal periovular transudate, used by the fetus for its early nutrition. These are factors which determine the initial size of the chorion frondosum in relation to the laeve.

Other factors operating at a later date in pregnancy may change the initial characters of the placenta, but not its proportions relative to the chorion laeve.

II. THE INTERRELATION OF THE CHORION FRONDOSUM AND PLACENTAL SITE

The activity of the trophoblastic cells determines the relative initial sizes of the two chorions, the chorion frondosum and the chorion laeve. The distension and growth of the uterus determines in normal cases the degree of extension of both of these. Let us devote our attention exclusively to the chorionic plate, or chorion frondosum.

In normal cases the growth of the placental chorionic plate, after its initial size is determined by the agents already enumerated, depends upon the distension and growth of the uterus by its content. The juxtaposed placenta keeps pace in its growth with the placental site, which enlarges throughout pregnancy to reach at term an evolutionary size of that of a dinner plate. The growth of the uterus and placenta is not of equal pace throughout pregnancy. It varies at various stages of fetal evolution. The fetus does not reach its full growth until the full period of gestation has been spent. But it will be shown in the prospective monograph upon placental pathology that the placenta is a mushroom growth, of rapid and somewhat imperfect development, which obeys the laws of all life, in that it passes through its active childhood, its vigorous adolescence, reaches its quiescent balanced maturity, and passes into decadent senility. There is no such thing as a mature placenta at full term. They are all full of signs of old age. I have found only about 10 quasi-normal placentas at full term or at estimated full term. The others all presented various degrees of advanced senile and active disease pathology, and it will be one of the greatest problems in the forthcoming monograph to distinguish the changes due to senility, from those that are the result of

pregnancy intoxication and other diseases. It will not be opportune to enter deeply into the changes incident to placental pathology except to touch upon that which is germane to circumcrescence. Anything which interferes with the normal extensibility of the chorionic plate will prevent that placenta following with equal pace the growth of the uterine extension. The basic principle of normal interaction of placenta and placental site implies two equally healthy juxtaposed and adherent organs. Any disease of either upsets that normal balance of correlation. The greatest pathologic factor in upsetting that normal balance is chorionic sclerosis.

Chorionic sclerosis is one of the commonest, in fact the commonest, placental disease. It is, I think, an expression of placental senility, but like all senile decay it may be hastened by maternal and fetal disease. It is brought about always by a slow or rapid deposit of a clamping impediment—a deposit of edema and later fibrin tissue—chiefly about, but not confined exclusively to, the maternal decidual tissues and its accompanying blood vessels in and below the placental chorionic plate. Just as in human life senility may be very precocious, so may it be manifested in very early placental degeneration. It may occur in some placentas as early as the second and third month and is, I am inclined to think, evidence of an endocrine disturbance either in infant or mother or both. In eight whole placentas obtained through abortion at from the fourth to sixth month, exactly 50 per cent of them were markedly circumvallate with marked sclerosis, not only of the membranes and chorionic plate, but also in two cases a very decided scleroderma of the fetuses. Chorionic sclerosis of this type can be studied to best advantage in hardened specimens.

There is an additional sclerotic factor in the placental plate found in about 10 per cent of cases at term, less frequently in the earlier months of pregnancy. It is a perivascular exudate involving the fetal vessels, both arteries and veins, of the chorionic plate. This is at first an edema, reactionary to some unknown cause; later, the exuded plasma concentrates and appears like a thin layer of milk exuding from the vascular system between the mesoblastic layers of the chorionic plate. It may be well to point out that the mesoblastic tissues of the chorion are divided into two laminae, which can be easily separated, and between which the fetal vessels course before perforating the lowermost to invade the villous arborizations. The perivascular exudate described above, spreads ordinarily between the component layers of the chorion like a thin layer of milk oozing out of the blood vessels. But in many instances it may spread over the whole circumference of a part of any affected vessels, leaving the other portions of the fetal surface of the blood vessels unaffected. When this occurs there is usually a definite, easily perceptible narrowing of the vessel by the constricting exudate. Doubtless also the

involvement of vasa vasorum plays an important part in this vascular sclerosis, and spreading exudate. The important features arising out of this perivascular exudate are twofold. First, the spreading sclerosing substance between the chorionic layers and, second, the interference with normal nutrition of the chorionic plate by the diminished vascularization. The density of the chorion can be seen in its every phase as a sequence to this perivascular exudate. This type of sclerosis is best studied in the fresh placenta.

But one may ask, "How does sclerosis of the chorionic plate and its subjacent tissues produce circumcrescence?" By placing a restraint upon the centrifugal growth of the placenta in its endeavor to keep pace with the expanding placental site, which, in turn, is proportionate

Fig. 5.—Normal placenta, showing direction of growth tension.

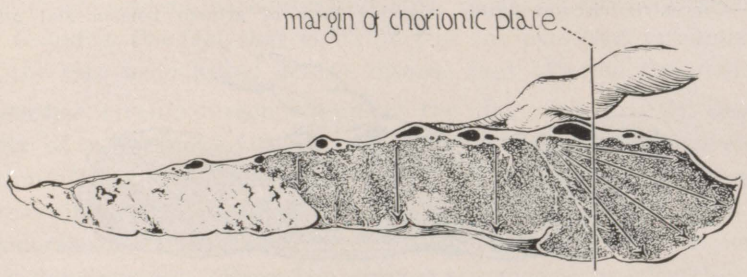

Fig. 6.—Infarcted placenta, showing compensatory hypertrophy lines of growth tension—eccentric circumcrescence.

to the whole uterine distension and growth. Normally the growth of the placental site and superimposed placenta is radial from a central point. These normally grow *pari passu*. But the impaired expansibility of a sclerosed chorion places an embargo upon its speed and vital activity, so that it can no longer keep pace with the evolutionary growth of the placental site. The centrifugal pull of the expanding placental site is exercised upon the chorionic plate, not only through the medium of the intervening anchoring villi, but also through the centrifugal circumferential pull of the chorion laeve in its endeavor to follow the growing uterus to which it is attached. But, if through sclerosis, the expansible properties of the chorion are below the normal, the villi will gradually respond to the centrifugal pull to the best of their ability, so that in a fully developed circumcrescence the pull

and hypertrophy cause the villi to extend their uterine ends beyond the area of the boundaries of the chorionic plate though their roots are always attached within the chorionic circumferential margin (Figs. 4, 6, 7, 8). Contrast the arrows of growth extension with the normal in Fig. 5.

The angle of villous growth therefore will not be directly downward at right angles to the chorionic plate, but will be at a more and more acute angle the farther one recedes radially from the central point of the placenta. In concentric circumcrescence this pull is equal in all radii. In eccentric circumcrescence the pull is radially unequal. This differene will require an explanation. In concentric circumcrescence, the process is usually slow, mild, and often manifests itself in the first half of gestation. The causative sclerosis is slow in

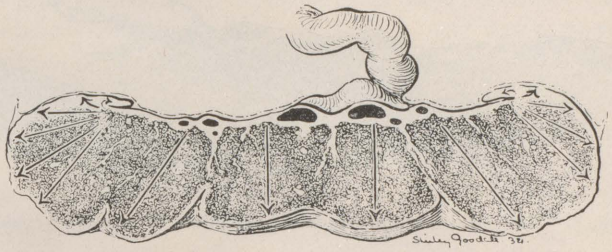

Fig. 7.—Concentric circumvallation, showing lines of growth tension and plication of chorion.

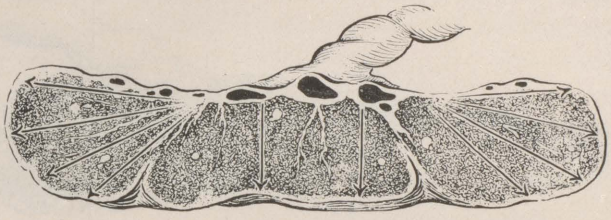

Fig. 8.—Concentric circumcrescence, showing lines of growth tension.

its development, relatively slight in degree, affecting chiefly the chorionic plate, but leaving the villi inappreciably altered. As the placental site expands *pari passu* with the general uterine growth, the chorion lags behind by virtue of its impediment. The villi follow the expanding site. The expansion is naturally greatest at the periphery. The resultant is an outstretching of the villi, attached fetally within the circumference of a relatively inexpansile chorion, and maternally to the expansile placental site. The disparity between the expansibility of the two villous attachments, and the duration of the imbalance will determine the degree of circumcrescence. Let us consider hypothetical cases. First, a mild degree of maternal or fetal disease produces a mild sclerosis of the chorion. The villi have not suffered appreciably. The imbalance between chorion and placental site will grow more marked as time goes on; compensatory hypertrophy of

the placental villi will take place manifested by increased thickness; and growth of the villi will be at gradually increasing obliquity from the placental margin to the circumference of the placental site. In these mild cases of long duration, circumcrescence may reach its greatest development, and it will always be concentric because the chorionic plate and its subtended villi will not have suffered at any one, more than at any other, part of its circumference. One may modify the intensity of the chorionic sclerosis to any degree imaginable, even to that of complete inexpansibility, and the response will be the same, though more rapid in its development. But one encounters in such cases a new factor that may modify the picture. In severe scleroses, the perforating fetal blood vessels may suffer much damage also and indirectly thereby affect the subtended villi, nutritionally and functionally, reducing compensatory villous hypertrophy almost to the inappreciable degree. Under these circumstances, there will be slight circumcrescence in a thin placenta incapable of hypertrophy. You may modify the component causative factors of concentric circumcrescence in intensity, duration, and period of gestation, and the effect will be a circumcrescence varying first, in degree; second, in concomitant villous hypertrophy, expressed in placental thickness; and third, in influence upon fetal growth and viability. The essential factor of concentric, as contrasted with eccentric circumcrescence, is to be found in the fact that at no part of the placental circumference have the villi been killed by the disease that affected the chorion.

Eccentric circumcrescence implies the death or greatly impaired vitality of a segment of the placenta, resulting in infarction and absorption of that area. The eccentric circumcrescence is nearly always greatest diametrically opposite the infarction in the placental area. Circumcrescence under these circumstances becomes again a compensatory development. Eccentric circumcrescence is usually the sequel to placental infarction and chorionic sclerosis. Concentric circumcrescence is a sequel to chorionic sclerosis alone. As infarction is rare in the first half of pregnancy, so also do we seldom find eccentric circumcrescence in the first five months of fetal life, except in cases of definite nephritis manifest in the early pregnancy months; which is relatively rare. On the contrary, we find mild grade of generalized chorionic sclerosis without villous infarction more commonly in the first semester of fetal life.

If chorionic sclerosis is the commonest placental disease at full term and is the chief causative agent of circumcrescence, why should circumcrescence be a relatively uncommon sequel? The answer is simple and satisfactory. Just as in cardiac or other organic disease, hypertrophy takes time to become appreciable, so we find that time is required in placental hypertrophy. It has been possible to estimate, in certain private cases, that it requires at least two months after the

cause has been in operation, for a circumcrescence to be appreciable. For this reason, sclerosis of the chorionic plate must necessarily begin before the end of the seventh month of gestation for circumcrescence to be appreciable at full term. Therefore, chorionic sclerosis is the commonest of placental degenerations. The very great majority of these cases are a late development of intrauterine life—too late to evoke hypertrophy and circumcrescence. Naturally sclerosis is relative in its degree of intensity, and inhibition to the extension of the chorionic plate is also relative. The history is often as follows: a more or less severe toxemic attack lasting two weeks or more in the early months of pregnancy, followed by natural immunity. Such a condition must occur before the end of the seventh month. If it occurs at a later date, it will produce merely a compensatory hypertrophy of the villi and a great thickening of the placenta, provided cell potentiality is such as to permit hypertrophy. It is probably not appreciated fully that quasi-normal placentas vary in thickness from 1 to 6 cm., the rough average being about 3 cm. If the sclerosis occurs slowly and uniformly and does not destroy or greatly affect the villous vitality, the villi will elongate universally, and will be pulled out at the margin of the placenta, causing the placental margins to be rounded and bulky, surpassing the boundaries of the chorionic plate; under these circumstances the circumcrescence will be concentric because at no part of the margin have the villi been destroyed. But the story is a different one in the case of an eccentric circumcrescence. In these cases, when the flood of disease that caused the chorionic sclerosis has passed over the placenta, it also left an universal disease of all the placental parenchyma in which certain parts of the placenta were more severely affected than others. Consequently some villous portions involving a segment of the placenta and its subtended circumference succumb to the acute or subacute process, whereas parts more remote suffer less and are regenerated or suffer apparently not at all. Usually the farther one recedes diametrically across the placenta from the affected area, the less is the effect of the blight apparent. On cross-section of such a placenta one sees the diseased margin is thin and fibrous, and under the microscope one finds that the villi will have undergone cloudy swelling and fatty degeneration and absorption, leaving the more resistant maternal fibrin-laden tissues in an advanced state of hyaline degeneration, often with cavitation. As one looks back from this highly degenerated area, the placental tissues are found in lesser degrees of destruction; some function is still retained, though the inherent cell potentiality is usually so low as to permit only precarious survival, but not hypertrophy. As one looks back still further, potential cell power increases so that not only is repair of damage possible, but hypertrophy to compensate both for the lost function in the diseased portion and for meeting the increas-

ing demands of fetal growth may also be possible. This compensatory hypertrophy is beautifully illustrated in Fig. 6, which represents the usual condition in cases of eccentric circumcrescence. The hypertrophy which first precedes, and then goes on *pari passu* with the circumcrescence, is also detectable in the great length of the fronds in the circumcrescent area. In the normal placenta, the fronds gradually grow shorter as one approaches the chorionic margin, therefore in marked contrast to the long hypertrophic marginal fronds of the circumcrescent placenta. Contrast Fig. 5 with Figs. 6, 7, and 8, in which it is shown that in the area of compensatory hypertrophy, the fronds become of extreme length and divergence. It is a noteworthy fact brought to my attention by my colleague, Dr. John Fraser, that an attack of acute placentosis, which may, of course, be of any degree of acuteness, produces less destruction in the growing and mature placenta than an attack of similar intensity would produce in the decadent stage of placental senility. It therefore follows that concentric circumcrescence is more common in the earlier fetal months because the resultant temporary blight usually leaves no trace of its passage except a stiffening of the supporting structures, and the regenerative forces of fetal tissues in the full vigor of their adolescence permit a restoration which could not be expected in the stage of decadent placental senility. It therefore follows also that eccentric circumcrescent hypertrophies preponderate over the concentric type because of the foregoing two factors, (1) the greater incidence of toxemia in the later months of pregnancy, and (2) their greater destructive power in placental senility, at which stage not only is there sclerosis of the chorionic plate, but also partial or complete atrophy of a segment of placental fronds. But what is "placentosis"? It is a new name for one of the commonest of placental diseases; it is the precursory stage of white infarctions; quick in its manifestation, varying in its intensity, selective in its incidence, and transitory in action; susceptible of repair if not too intense, and deadly to both placenta and child if intensive and diffuse; often subacute and creeping in placentas showing all stages of simultaneous repair and destruction; affecting not only one-half of a placenta to the degree of total atrophy, leaving the other half less affected and capable of carrying on, but also affecting to a marked degree one placenta of a twin pregnancy so as to place the attached child's life in jeopardy, yet leaving the other but slightly disturbed. This and other diseased conditions must be left to be dealt with fully in a subsequent work. It is this placentosis which operates with so much more destructive effect upon the senile placenta, in marked contrast to the lesser changes in younger placental elements.

The question arises at once, "Why should a placenta which has not fulfilled its complete function become senile from the seventh month

on, when the fetus must still grow and its demands must be met?'' There are many reasons for this. First, senility is a relative term. A man, fifty years old, has a greater capacity for certain types of work and lessened capacity for other kinds of labor than when younger, yet his biology will undoubtedly register signs of senile decadence. But it is work of a different kind that is demanded of such. So it is with the placenta. The average concept of the placenta is that its only function is to bring oxygen and food and take away catabolic elements and that therefore the demands upon the placenta should increase with the growth of the fetus. This is but a superficial view of the situation. In the early stages of fetal life, cell activity is estimated by physiologists to be twenty-five times that of the human adult; it is also estimated that the rate of cell division and metabolism slow down gradually as fetal maturity is approached. Fetal life is like terrestrial life, it progresses from its embryologic stage to its maturity at term. It would be unorthodox to expect the young boy's metabolism in the old man. But of far greater significance are the other properties of the placenta which center chiefly in its capacity as a storehouse for the elements necessary for the growth of the fetus, such as endocrines, glycogen, etc.—elements which the fetus is incapable of generating in its anaplastic state, but which generation it gradually assumes for itself as it reaches maturity. May I make this very clear by just one example. An animal whose pancreas is excised will die very quickly from glycogenic disturbance due to the withdrawal of the pancreatic endocrine. If the same operation is performed upon another animal in the early stages of pregnancy, the result will be the same. But a similar operation upon an animal in the late stages of pregnancy will have no appreciable effect upon the mother during the duration of the pregnancy, but she will die promptly after delivery. There is but one interpretation, and that is that the fetuses take over their own functions at the proper time and, in case of need, may even supply a deficiency in the mother. It is doubtless the assumption of functions by the fetus at different stages of its development that makes an early large placental reserve unnecessary in the later months of gestation. Moreover, need I mention that the placenta, like every other form of living thing, has its own life history, and is not exempted from senility? And among placentas there are the averages, and the prematurely senile, and the retarded decadences, just as in humans.

III. THE RELATION OF THE CHORION FRONDOSUM TO UTERINE GROWTH AND DISTENTION

Just as the trophoblasts determine the initial size of the chorionic plate in relation to the chorion laeve, so there are factors later in pregnancy which determine the relation in size and thickness of the

placenta to the size of the uterine content. The normal placenta grows *pari passu* with the growth of the placental site, and this latter is proportionate in its extension to the uterine distention. Factors such as hydramnios, acute or chronic, may cause a marked extension of the placental site, in keeping with the increase in the uterine content, and if the chorionic plate is sufficiently elastic to keep pace with the rapid extension of the placental site, the placental thickness will become correspondingly reduced. In the same manner a gradual reduction in the uterine content will have a corresponding effect in reducing the placental site and with it the circumference of the placenta, causing an increase in the thickness and compactness. This reduction reaches its acme of rapidity and degree in the retraction after birth of the child. The placental site so reduces itself that the placenta follows it for a time by increasing its thickness and compactness, but its elasticity relative to that of the tissues underlying the placental site causes a "buckling," which is the first stage of placental separation.

So it becomes evident that through the trophoblastic agencies the initial relation between the chorion frondosum and chorion laeve is fixed; that uterine growth and distention determine the relation of placental site to uterine content; and expansibility or sclerosis of the chorionic plate determines the relation of placenta to placental site.

Circumvallation.—Circumvallation is an added complication to an hypertrophic circumcrescence. Circumcrescence must always precede circumvallation. As the name implies, it means a "ditching" at the margin of the placenta and an apparent or real cupping of the placental plate so that it presents a concave instead of a convex surface toward the fetus. This concavity is most marked in the early fetal concentric hypertrophies and is best illustrated in nature by the growth of a mushroom or toadstool. In the case of the toadstool or fungus, the smooth upper surface corresponds to the chorionic plate, and the undergrowth of plates corresponds to the placental fronds. If weather conditions are normal, the upper and under surfaces keep pace in their growth. If there is too much moisture the "fronds" grow too rapidly and split the "chorion." This, of course, cannot occur in the placenta. But, if the atmosphere is too desiccating, it dries the upper surface of the fungus, and to accommodate the progressive undergrowth with which it cannot keep pace, owing to its desiccated sclerosis, it cups and presents a concavity instead of a convexity to the surface. The similarity to the placental stages is very striking. But there is an additional factor to explain the "ditching" at the margin of the chorionic plate. This "ditching" or circumvallation may be concentric or eccentric, depending upon whether the case was previously of the one or the other type. But "ditching" need not involve the whole circumference of the placenta even in a case of concentric hypertrophy, nor for that matter need it involve the whole of

the segment of the placenta showing eccentric hypertrophy. The vagaries of circumvallation are many. In a few cases there may be numerous circumvallations concentric with the major one at the margin of the chorion plate. These minor ones may be as numerous as six, merely duplications of the surface layer of the chorion. Circumvallation is the late result of a reactionary edema expressive of a fetal outpouring of lymph as a diluent to the maternal fibrin-laden tissues, behind which is probably a maternal toxemia. This edema lies under the amnion and invades the layers of the chorion. The amnion is usually raised by the fetal fluid from its attachment to the chorion. The chorion is made up of two or more distinct but firmly adherent layers,

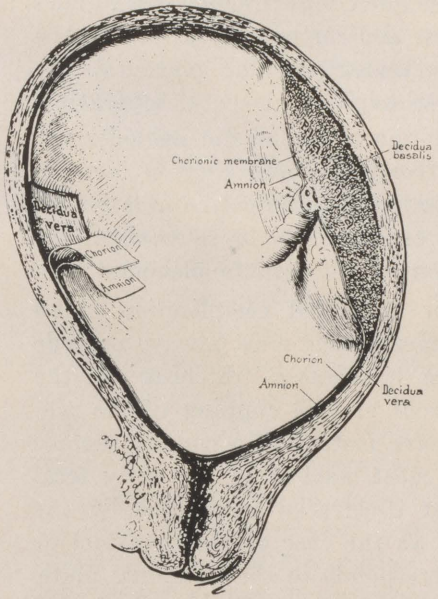

Fig. 9.

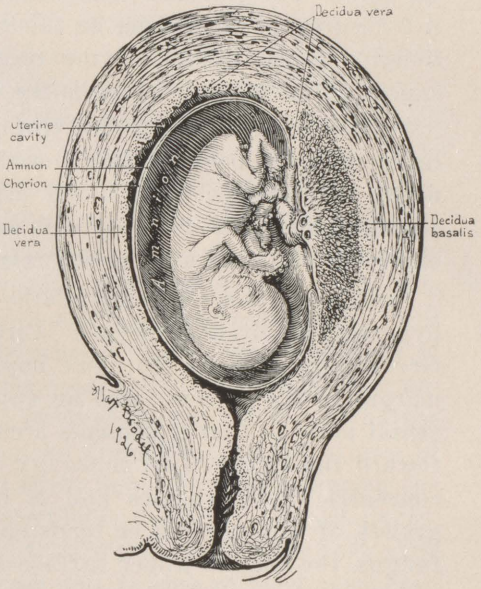

Fig. 10.

between which the larger fetal vessels run before they perforate the subjacent cribriform plate to become the main stem and support of the fetal fronds. The edema is most marked where the maternal decidual tissue is in greatest quantity and is undergoing degenerative changes. Decidual tissue is most abundant in the placental area on the surface of the circumcrescence and is most liable to degeneration because it has been raised from its natural maternal bed and, as a consequence, is further removed from its blood supply. No matter what the degree of circumcrescence, fibrinosis of greater or less degree marks the boundaries of the chorionic plate. Consequently it is at this spot that the edematous reaction is most marked, and plication takes place, single or multiple, of the loose chorion, superimposed upon the circumcrescent surface. (Contrast Figs. 9 and 10.) It is a property of pla-

cental edema that it soon changes its physical characters and becomes a residuum of fibrin tissue which gradually solidifies and contracts, thereby adding greatly to the sclerosis of the chorion plate. In the early stages it is flocculent and easily torn. In the older stages, where circumvallation has taken place, the fibrinosis may take on great density and a ropelike strength. I have excised this circumvallate ring in many cases, and find that the ring alone will frequently sustain a pull of from 5 to 15 pounds, depending not so much upon the thickness of the circumvallate cord as upon its duration and consequent change from fibrin to fibrosis. The larger placental vessels always dip through the chorion within the ring of circumvallation, to pursue a centrifugal radial direction to feed the fronds in the hypertrophic circumcrescence.

Clinical Significance of Circumcrescence and Circumvallation.—J. Whitridge Williams stated in his concluding remarks in this work in 1929 that circumvallation had no clinical significance. Schumann, writing in the *Curtis System of Gynecology and Obstetrics* also wrote as follows: "This anatomical anomaly has no clinical significance except in its occasional association with widespread placental infarction."

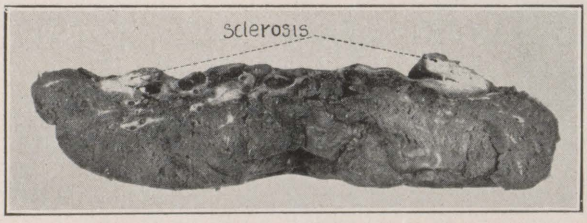

Fig. 11.—Sclerosis of circumvallation.

From what has already been written it can readily be seen that this statement is not correct—either in its conception of the association of two diseases or in its appreciation of the clinical significance. First of all, the two conditions, circumvallation (and therefore circumcrescence) and "widespread placental infarction," are not mere "occasional associations." They lie in the relationship of cause and effect. The "widespread infarction" means that a storm has passed over the placenta and that a large portion of the placenta is placed *hors de combat* by the resulting infarction; the compensatory hypertrophy and circumcrescence is an expression on the part of the placenta to keep up its reserve and preserve the life of the fetus. These then operate not as accidentally associated diseases, but as cause and effect, and the sclerosis of the chorion, as a resultant of the storm, restricts the expansibility of the chorion, preventing its keeping pace with the compensatory villous hypertrophy. Chorionic sclerosis may vary in its intensity and inhibitory effect from, let us say, 5 to 100 per cent. In the minor types villous compensatory hypertrophy will not be appreciably in-

hibited, and the chorionic defect can be made up entirely by the hypertrophy. Placentas vary from 1 cm. to 6 cm. in thickness. The thinner types are usually the result of atrophy; the thicker ones, of hypertrophy. Circumcrescence is always associated with hypertrophied villi, longer and more arborescent; unless, subsequent to the compensatory circumcrescence, the placentas are overtaken by another attack of destructive agencies. Its clinical significance can be best judged by a careful examination of all cases of hypertrophy. Naturally in the majority of circumcrescent placentas cases with birth at term, there will not be any appreciable effect upon the size or growth of the child because the hypertrophy will have compensated for the diseased area of the placenta and placental reserve may not be appreciably lowered; and therefore, the fetal nutrition may have suffered but slightly, or not at all. That is the history in most instances of birth at term. But the clinical significance is quite another story when we are dealing with concentric circumcrescence of the early months of fetal life. Here the restriction of the extensibility of the chorionic plate places a limit to the villous hypertrophy and circumcrescence, and though these two factors may keep pace with the fetal growth for a time, there will come a period when the placental growth cannot meet the demands. This disparity will increase with the weeks and eventually a macerated, emaciated fetus is cast off, or a sclerosed, emaciated octogenarian is born, which survives if it is old enough. It all resolves itself into a question of:

1. The time of the placental disease in the period of gestation
2. The intensity and duration of the causes which affect the placenta
3. The extent of the damage
4. The recuperative cell power (potentiality) of the surviving placental villi
5. The degree of restraint placed upon the chorionic elasticity by the resultant chorionic sclerosis.

It has been clearly shown by Kearns that whereas it requires a certain length of time and a certain pressure to force a glutinous substance through the vessels of a normal placenta, in placentas in which there are large infarctions, much less time and a much reduced pressure are required to force the same fluid through the compensatory hypertrophied portion. This shows that the fetal circulation is accelerated through the compensating area, either by vasomotor dilatation or by development of new and large vessels; that vascular exchange between mother and fetus is correspondingly hastened, and that this placental nutrition is heightened permitting increased cell activity and power of villous restoration.

1472 Sherbrooke Street.

PRIMARY CARCINOMA OF THE TRACHEA

ORMOND S. CULP, M.D.
Montreal, Canada
From the Pathological Institute, McGill University

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PRIMARY CARCINOMA OF THE TRACHEA*

ORMOND S. CULP, M.D.
MONTREAL, CANADA

PRIMARY tumors of the trachea constitute a comparatively rare group of lesions that are of particular clinical and pathologic interest. The clinical aspect is concerned with methods for making a correct early diagnosis and instituting more effective treatment, while the pathologic problem centers about the morphology of the tumor and its possible malignant tendency. This brief report is confined to the pathology of carcinoma of the trachea. An attempt will be made (1) to review the literature, (2) to make an analysis of the types of primary carcinoma that already have been described, (3) to record the anatomic findings in two additional cases.

As early as 1767 Lieutaud[9] described a fibroma in the trachea as an incidental autopsy finding. No other tumors were described until after the introduction of the laryngoscopic mirror in 1854. Rokitansky[104] reported an autopsied case in 1857 which was one of the first malignant tumors to be observed. Türck[16] in 1861 first utilized the newly discovered indirect laryngoscopy to diagnose a tracheal growth in the living subject, but Langhans[79] in 1871 was the first to report a primary carcinoma confirmed by detailed histologic study.

The development of the bronchoscope did much to aid the early and accurate diagnosis of these tumors. Killian[19] first diagnosed a primary tumor of the trachea by endoscopy. Reports of tracheal growths became progressively more frequent with improved laryngoscopic and bronchoscopic methods and with more complete postmortem studies.

INCIDENCE

In 1898 von Bruns[124] assembled the available statistics on primary tumors, in general, after having observed 7 such cases. These were classified as shown in Table I. They totaled 147 cases, of which 21 per cent were carcinomata. Krieg[7] made the next comprehensive study in 1908 and found a total of 201 cases with no appreciable change in the proportion of carcinoma. Fifty-one additional newgrowths were collected by Lombard and Baldenweck[10] in 1914, while in 1929 D'Aunoy and Zoeller[41] presented the last exhaustive survey of the subject with a total of 351 cases. These included 91 instances of carcinoma (see Table I).

*From the Pathological Institute, McGill University. Professor Horst Oertel, Director.
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An attempt has been made to complete these general statistics by including all the reported primary growths to the end of June, 1936. The classification used in the previous studies has been followed as closely as possible. Most of the literature during the past seven years has dealt with malignant tumors. Of the 82 new cases of primary growths recorded during this period, 56 were described as carcinomata. Of the total 433 primary tumors on record at the end of June, 1936, 147 (or 34 per cent) were carcinomata (see Table I).

TABLE I

PRIMARY TRACHEAL TUMORS

TYPE	VON BRUNS 1898	KRIEG 1908	LOMBARD & BALDENWECK 1914	D'AUNOY & ZOELLER 1929	PRESENT STUDY 1936
Fibroma	23	25	29	33	36
Papilloma	33	41	51	59	63
Lipoma	3	4	5	5	5
Chondroma, osteoma, tracheopathia osteoplastica	29	42	47	65	71
Adenoma	5	6	6	8	9
Lymphoma	2	2	2	3	4
Intratracheal goiter	7	14	19	25	28
Amyloid tumors	–	–	1	2	2
Mixed tumors	–	–	–	2	3
Angioma	–	–	1	2	2
Cylindroma	–	–	–	1	1
Sarcoma	14	21	23	26	31
Carcinoma	31	40	54	91	147
Carcinosarcoma	–	–	–	1	1
Endothelioma	–	–	–	1	1
Histology undetermined or doubtful	–	6	14	27	29
Total	147	201	252	351	433

Although primary carcinoma appears to be the most frequent type of malignant tumor of the trachea, the following observations indicate that it is of relatively rare occurrence. In 5,063 autopsies Fraenkel[50] found 7 cases. This is the highest percentage (1.4) of any series on record. From 1918 to 1930, Figi[48] reported 5 patients with primary carcinoma at the Mayo Clinic. D'Aunoy and Zoeller[41] were able to find only one case at the Charity Hospital of New Orleans over a period of twenty-three years. They point out that statistics from the Pathological Institute of Basle for thirty-five years showed only one growth of tracheal origin out of 1,078 cases of malignancy. Broman[34] added one case from a series of 545 malignant growths found at autopsy at the Cook County Hospital, Chicago. Bilz[2] found none of tracheal origin among 700 cases of carcinoma at the University of Jena. Bejachs[1] was unable to find any among 1,300 cancer autopsies at the Berlin Charité, and Mielecki[12] was also unsuccessful in his review of 560 cancer patients. Stenn[114] contributed the only case at the Billings Hospital, Chicago, and cited Lund as having found 2 cases in 25,000 autopsies

at the Philadelphia General Hospital from 1867 to 1934. He also noted that Symmers found none at the Bellevue Hospital, New York, that Evans found none in 12,000 autopsies at the Los Angeles County Hospital, and that Wright found none in 5,000 autopsies at Guy's Hospital, London.

A personal survey yielded no primary carcinoma of the trachea in 15,000 autopsies at the Johns Hopkins Hospital, Baltimore, none in 7,000 autopsies at the Baltimore City Hospitals and none in 3,900 autopsies at the New Haven Hospital. The cases herein reported are (1) the only carcinoma of tracheal origin in 9,000 autopsies at the Pathological Institute of McGill University, Montreal, and (2) the only one in 12,700 autopsies at the Montreal General Hospital.

CLINICAL ANALYSIS

There is very little about the clinical picture of the disease which is characteristic. A multitude of initial symptoms has been described for the various cases in this series, but none are indicative of carcinoma per se. The commonest early symptom was a tickling sensation in the trachea, followed by an irritating cough and persistent hoarseness (due to pressure on the recurrent laryngeal nerve). In some instances, however, this entire phase was lacking, and the earliest symptom was dyspnea or hemoptysis.

According to Jackson[4] the chief symptoms of any tracheal tumor are: (a) wheezing respiration, (b) nocturnal attacks of dyspnea, (c) dyspnea on exertion, (d) asphyxia, unless averted. Gilfoy[52] claimed that the symptom complex of intermittent dyspnea, cough, perhaps occasional hemoptysis, apparent good health and absence of obvious intrathoracic signs suggests a tumor in the tracheobronchial tree and warrents endoscopy. Because of the mildness of early symptoms, most patients failed to seek medical attention until the process was advanced. The chief diagnostic distinctions were asthma, the more common mediastinal tumors, carcinoma of the esophagus, empyema, bronchiectasis, pulmonary abscess, pneumonia, and enlarged thyroid. All writers agree that diagnosis depends upon the combined methods of palpation, x-ray, laryngoscopy, bronchoscopy, esophagoscopy, and occasionally open exploration.

Almost every conceivable form of therapy has been tried, varying from the more simple endoscopic measures to radical surgery. Figi[48] offered the following as a summary of the treatment of tracheal carcinoma: (a) Lesions in the upper half are best treated surgically with exposure of the growth by tracheofissure and destruction of it with electrocoagulation. A large tracheal tube is then worn from six months to one year and simplifies later treatment if necessary. (b) Excision of a segment of trachea has been carried out in some cases but seems

to offer no better results than diathermy and involves greater risks. (c) Implanted radium and surgical diathermy are recommended for the lower half of the trachea. (d) In all cases the regional glands should be given radium or deep x-ray therapy.

Regardless of the treatment, the prognosis for carcinoma of the trachea is generally very poor. Apparently the only real hopes for cures are the early lesions in the upper half, and even then the disease is not recognized usually until well advanced. Most patients in this series died within one year, but a few isolated patients were living as long as seven years after the initial treatment.

The terminal event was usually (1) suffocation, (2) pneumonia, frequently in instances of tracheoesophageal fistulas, (3) massive hemorrhages.

PATHOLOGIC ANALYSIS

Data concerning carcinoma are frequently misleading because accurate clinical diagnoses are seldom made, histologic reports are meager and postmortem confirmation is often lacking. It is possible that some cases in this review should not have been included, while other cases of mediastinal tumors of uncertain origin should not have been omitted. Such errors of commission and of omission probably would about balance each other. This analysis is based, therefore, on a review of the 147 recorded cases.

A. *Age Distribution.*—The youngest patient on record was a girl 18 years of age, while the oldest patient was an 82-year-old man. Of the 111 reports in which the patient's age was given, 100 cases (90 per cent) were distributed fairly equally among persons between 30 and 70 years of age with only slightly increased frequency in the fifth and sixth decades (see Table II).

B. *Sex Distribution.*—Practically every reviewer has emphasized the marked predominance of males as victims of tracheal cancer. Sex was recorded in 116 cases of the present series and, of these, 63 per cent occurred in men (see Table II).

TABLE II

AGE	SEX			METASTASES			TOTAL CASES
	MALE	FEMALE	NOT GIVEN	PRESENT	NONE	NOT GIVEN	
10-19	0	1	0	0	1	0	1
20-29	4	1	0	1	3	1	5
30-39	11	9	0	9	8	3	20
40-49	14	14	0	14	8	6	28
50-59	21	10	0	18	6	7	31
60-69	15	6	0	14	3	4	21
70-79	3	1	0	3	0	1	4
80-89	1	0	0	1	0	0	1
Not given	4	1	31	2	0	34	36
Total	73	43	31	62	29	56	147

C. *Location of Tumor.*—The trachea has been divided arbitrarily into the upper, middle, and lower thirds. Carcinoma occurred most frequently near the bifurcation, i.e., in the lower third. Next in frequency was the upper third, and least often it was situated in the middle portion (see Table III).

TABLE III

	ANNULAR	ANTERIOR WALL	ANTERIOR AND LATERAL	POSTERIOR WALL	POSTERIOR AND LATERAL	LATERAL ONLY	NOT GIVEN	TOTAL CASES
Upper third	3	3	5	11	9	4	10	45
Middle third	0	2	0	5	2	1	10	20
Lower third	2	10	4	16	3	4	22	61
Not given	0	1	1	3	0	0	16	21
Total	5	16	10	35	14	9	58	147

The tumor formed an annular mass about the trachea in only five instances. It was usually a well-localized process in the wall similar to the first case herewith reported (see Fig. 1). By far the commonest area for involvement was the posterior wall alone, and when combined with those involving both the posterior and lateral walls, these tumors constituted 55 per cent of all the cases in which an accurate location was given (see Table III).

According to this study, therefore, the commonest site of carcinoma of the trachea is in the posterior wall of its lower third.

Strauss[14] expressed the belief that the frequent involvement of the posterior wall is due to the drainage of lymph posteriorly. Von Bruns[124] and Schrötter[109] attributed it to the predominance of glandular epithelium in the posterior wall, in contrast to the cartilaginous nature of the anterior wall. On the same basis they explained the infrequency of circumferential growths. Simmel[111] claimed that the lower third of the trachea is most exposed to trauma and upon this basis explained the frequency of tumor growths in this region.

D. *Metastases.*—Of the 147 cases reviewed, metastases were mentioned as being present or absent in only 91 instances. In 62 cases (68 per cent) metastases were described, while they were definitely absent in only 29 cases (see Table II). There seems to be no correlation between the incidence of metastases and age periods. Thus the decades in which carcinoma is most frequent show a corresponding increased incidence of metastases.

It is a peculiar feature that the commonest type of carcinoma to metastasize was the squamous cell variety. Of the 38 cases recorded, 23 had definite metastases (see Table IV).

The site of the secondary growths varied greatly, but they were usually regional, involving the esophagus (other than by direct extension), the mediastinal, peribronchial and cervical glands, and occasionally the lungs. Isolated instances of metastases to the skeleton, gastrointestinal tract, liver, spleen, pancreas and kidneys were recorded also. As D'Aunoy and Zoeller[41] pointed out, these tumors are in contradistinction to the laryngeal carcinomata which rarely metastasize and show a predilection for a certain limited number of organs. It appears that primary carcinoma of the trachea may be associated with secondary growths in any tissue or organ.

E. Histogenesis.—According to Maximow,[11] the epithelium of the trachea is of the ciliated, pseudostratified, columnar type and rests on a distinct basement membrane. Numerous goblet cells are scattered throughout this epithelium, and the lamina propria contains many small glands like those of the larynx. These glands are mostly external to the elastic fibers and open by short ducts on the free epithelial surface. With this histologic structure in mind, one would expect the adenoma and adenocarcinoma cell to be a frequent constituent of tumors of the trachea. Thus Langhans[79] in 1871 made the first important histologic study of tracheal carcinomata and concluded that most of them were derived from the epithelial cells of the mucous glands. This view was supported by the later investigations of Virchow[122] and Hamacher.[56]

TABLE IV

DIAGNOSIS	METASTASES	NONE	NOT GIVEN	TOTAL CASES
Definite Morphology:				
Squamous-cell carcinoma	23	5	10	38
Adenocarcinoma	10	6	10	26
Basal-cell carcinoma	1	5	2	8
Cylindric-cell carcinoma	3	2	2	7
Columnar-cell carcinoma	0	1	2	3
Uncertain Morphology:				
Alveolar carcinoma	2	2	2	6
Papillary carcinoma	1	1	2	4
Medullary carcinoma	10	0	3	13
Scirrhus carcinoma	2	0	1	3
Unclassified:				
Carcinoma	8	6	19	33
Epithelioma	2	1	3	6
Total	62	29	56	147

Considerable confusion then resulted from the contradictory statements of later authors concerning the predominant cell types in these tumors. Oppikofer[95] considered the squamous-cell carcinoma to be the most common, while Fraenkel[50] openly disagreed. Kaufmann,[5] quoting Nager, Oestereich, Schmiegelow and Kahler, thought the squamous cell variety was the one most frequently encountered, while Figi[48] contended

that adenocarcinoma was the most common. Hence the literature offers a host of conflicting statements which have not been clarified in the more recent pathologic text and reference books.

A histologic survey of the 147 cases was attempted, but, because of differences in terminology and the lack of adequate microscopic data, many cases were very difficult to classify accurately. Furthermore, earlier observers followed a very indefinite classification of cancers. Some were diagnosed on a morphologic basis (which is the only acceptable method), while other authors described only the general tumor cell arrangement and were content to identify the growths merely as carcinoma or epithelioma (see Table IV).

In general, the carcinomata seem to fall into two main groups, those arising from (1) glandular epithelium and (2) surface epithelium. The predominant cell in the latter group may be columnar, cylindric, basal, or squamous. Of these, the squamous-cell carcinoma is the most common. Thirty-eight unquestionable cases were found in this series. Only 26 definite adenocarcinomata were found, but it is probable that many of the alveolar and medullary growths belong in this group (see Table IV). It is noteworthy that the adenocarcinoma does not predominate as the early writers believed, but it would appear that the squamous-cell carcinoma is equally as common.

It is not difficult to explain the occurrence of the adenocarcinoma. It arises from the mucous glands of the trachea. The presence of mucoid secretion is noteworthy evidence. Inasmuch as no squamous epithelium is found in the normal trachea, the occurrence of squamous-cell tumors presents a morphologic problem. Two schools of thought have evolved, each supported by histologic evidence.

(1) Drasch in the Anatomie Humaine (Poireo) pointed out that islets of squamous epithelium frequently occur in the trachea as embryonic arrests. Such epithelial islands have been observed repeatedly by many investigators in the absence of neoplastic diseases. Inasmuch as both the trachea and the esophagus develop from the embryonic fore-gut, it is thought that nests of esophageal epithelium are included in the tissues of the trachea at the time it separates from the ventral portion of the esophagus. Reiche,[102] Nager,[89] Deland and McFarland,[44] Heymann,[62] and a few others were convinced that the tumors of their experience were best explained on the basis of Cohnheim's theory of embryonal inclusions. In the absence of any evidence of cellular metaplasia, this explanation seems quite justified.

(2) Many tumors, however, showed unmistakable evidence of metaplasia. Breslich[32] was able to follow the steplike transition from columnar ciliated epithelium to squamous cells. The tumors reported by von Meyenburg,[125] Gilfoy,[52] Baratoux,[19] Nielson,[91] Barth,[22] Teubert,[117] and Tiling[119] also illustrated varying degrees of metaplasia.

Krompecher[8] studied basal- and squamous-cell carcinomata which occurred in structures lined by columnar epithelium and decided that these tumors originated from proliferation of the basal cells. He claimed that these undifferentiated cells are morphologically similar to the basal cells of stratified epithelium and are distributed irregularly on the basement membrane of the mucosa between the cylindric cells. Normally they divide and differentiate into columnar epithelium to replace the lining cells which are injured or desquamated, but under certain conditions they may differentiate into transitional or stratified squamous epithelium and give rise to basal- or squamous-cell carcinomata.

In the trachea of rabbits with acute inflammations, Kawamura[6] observed a metaplasia of columnar ciliated to transitional epithelium. Teutschlaender[15] noticed that the bronchi of certain rats after bronchopneumonia were lined by transitional or stratified squamous or even hornified squamous epithelium, and traced the development of these changes step by step from the basal cells.

Goldzieher[3] described parts of the bronchi of a patient who had died of diphtheria as being lined by a wide layer of basal epithelium. In different places this was covered by either cylindric or squamous cells. In his opinion these unusual changes resulted from a further differentiation of the proliferating basal cells to cylindric or squamous types.

Three squamous-cell carcinomata of the lung described by Siegmund[13] developed in bronchiectatic cavities. The single-layered, cylindric, epithelial lining of the dilated bronchi was transformed into a many-layered, basal or transitional epithelium from which Siegmund thought the tumors originated. There can be no doubt, therefore, that metaplasia *does* occur in the epithelium of the air passages, and it seems probable that this frequently precedes neoplasia. Case 1 adds further support to this view.

CASE REPORTS

CASE 1.—*Clinical Course:* This 68-year-old man had increasing dyspnea and dysphagia for six months before coming to the hospital. Physical examination revealed marked respiratory stridor with indrawing of the intercostal spaces, emaciation, complete paralysis of the left vocal cord, slight left facial paresis and a firm nodule the size of a marble between the origins of the left sternocleidomastoid muscle; x-ray pictures showed displacement of the trachea and mediastinum to the right by a solitary dense shadow at the level of the third rib. Both lung bases were congested and there was a moderate polymorphonuclear leucocytosis. The clinical impression was a mediastinal tumor with lobular pneumonia.

Because of the pneumonia, no instrumentation was attempted. The patient had a fever of 99° to 102° for the next three weeks with periods of temporary improvement following repeated administration of morphine and atropine. For several days he was able to take a semisolid diet fairly easily. He became progressively weaker, however, and had episodes of severe chest pain, persistent coughing and a low grade fever. He died forty-three days after admission to the hospital with the clinical picture of diffuse bronchopneumonia complicating a mediastinal tumor of unknown origin.

Postmortem Findings: (A-386-36) Exposure of the thorax showed the upper mediastinum to be the site of an extensive tumor mass. The trachea was displaced 1 cm. to the right of the midline by the tumor, and its lumen seemed to be narrowed to about half its normal size.

On opening the larynx and trachea there was a firm, gray mass on the left lateral wall 5 cm. below the vocal cords. This was 4.8 cm. long and 2.5 cm. wide. The tumor involved all coats of the trachea producing a firm, granular, ulcerated surface 3 cm. long and 1.6 cm. wide. The mass reached a level 2.4 cm. above the left main bronchus (see Fig. 1). The transverse sections showed that the tumor extensively involved the left mediastinal tissues, forming an irregular, firm, gray mass 4.4 cm. wide. This extended posteriorly 5.6 cm. where it became firmly attached to the thoracic vertebrae (see inset Fig. 1).

The left subclavian artery formed its left lateral border and was partly surrounded by the mass. The innominate artery extended up the right lateral border. The left common carotid artery was surrounded completely by tumor and crossed to lie beside the left subclavian. The left innominate vein was filled with a firm, gray thrombus resembling tumor tissue. The left internal jugular vein was dilated to 1.2 cm. in diameter and was filled with laminated, pale and dark red clot. The other great vessels in the neck and mediastinum were free.

Inferiorly the tumor extended to the aortic arch and superiorly reached the level of the isthmus of the thyroid, the left lower pole of which was adherent to the capsule. The supraclavicular lymph glands were dissected and found to be free of tumor.

The esophagus was displaced to the left and formed the lateral border of the tumor in its lower half. The coats were invaded, producing a firm, gray, oval, nodular mass 3 cm. long and 2.8 cm. wide, over which the mucosa was intact except for an area 6 mm. in diameter near its center (see Fig. 1). This showed a shallow defect, the floor of which was flat and pale gray. The esophageal lumen was narrowed to about half its normal diameter by the bulging tumor mass.

On the posterior surface of the esophagus, 3.5 cm. below the lower limit of the tumor, there was a small, gray, granular elevation 1 cm. long and 6 mm. wide. On section this appeared as an irregular thickening of the mucosa, while other coats were not definitely indurated. There was a similar, slightly smaller, granular area 3.8 cm. below this which also showed no evidence of induration (see Fig. 1). These two areas resembled metastatic growths.

The right pleural cavity contained 175 c.c. of cloudy yellow fluid, and there were numerous loose pleural adhesions. There were also firm adhesions at the base of the left pleura. Both lungs showed the gross and microscopic picture of a diffuse aspiration bronchitis and purulent bronchopneumonia.

Incidental findings included multiple gastric ulcers, glandular hyperplasia of the prostate, multiple false diverticula of the sigmoid, productive cholecystitis with cholelithiasis, focal arteriosclerotic gliosis in the right cerebrum, patchy pachymeningitis hemorrhagica, and cloudy swelling of the parenchymatous organs.

Tumor Histology: The upper two-thirds of the tumor consists of cells in loose alveolar arrangement. These resemble transitional epithelium. They have large, pale, round, or oval nuclei with scattered nucleoli. There are many degenerating forms and the larger cell masses show central necrosis. Between these cells are a few, scattered, hyperchromatic, irregular, spindle-shaped nuclei without fibrils. In several places the apparently transitional cell replaces the marginal columnar epithelium of the trachea where the basement membrane is completely lost (see Fig. 4).

Sections across the lower third of the tumor show masses of immature pavement epithelium with typical cancer whorls and abundant keratinization, replacing

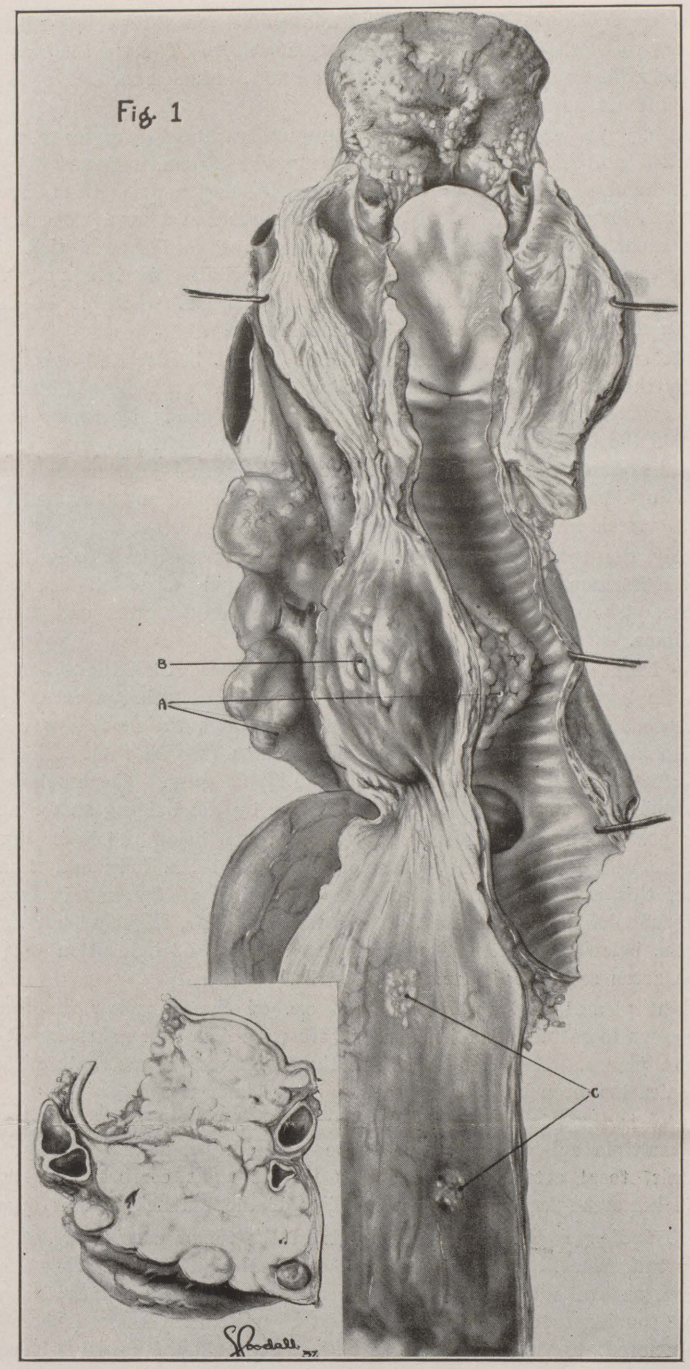

Fig. 1.—Posterior view of the opened trachea and esophagus. *A*, The tumor mass displaces the trachea to the left, all coats are infiltrated and there is extensive ulceration. *B*, Small erosion of the esophageal mucosa over the bulging tumor. *C*, Metastases in the lower esophagus.

Inset represents a cross section at the level *A*. The tumor replaces the left tracheal wall and fills the mediastinum. The intact overlying esophageal mucosa is seen in the upper right, and the thrombosed left innominate vein is at the lower right border.

the tracheal coats and surrounding cartilaginous rings (see Fig. 2). These cells show no evidence of intracellular bridges. There is here an abundant, dense, fibrous tissue stroma. The immature squamous epithelium in places fuses with the immature transitional cells described above (see Fig. 3). The tumor cells

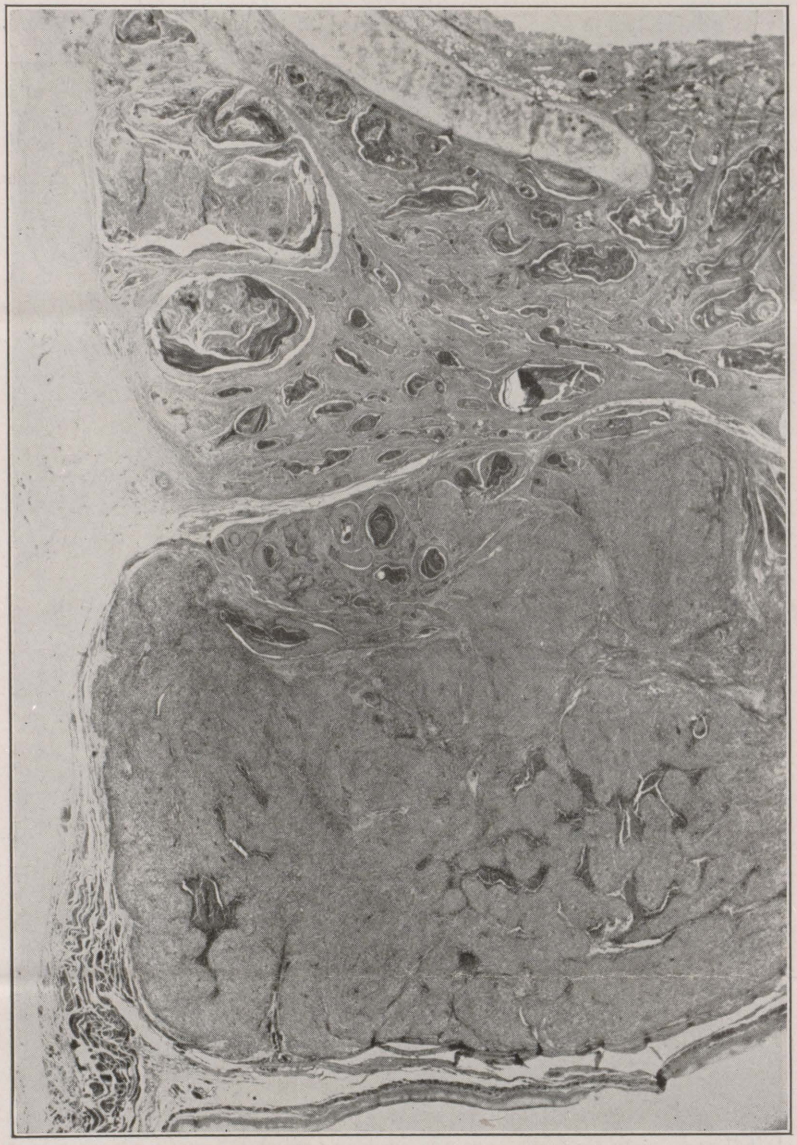

Fig. 2.—Low power photomicrograph of a transverse section from the lower third of the tumor. The trachea above may be identified by the cartilaginous ring. The wall of the trachea is infiltrated with immature keratinizing squamous epithelium. The esophagus below is partly invaded by a solid mass of transitional epithelium. In the center of the picture is a zone of metaplasia. (×50).

invade many mucous glands, but there is no evidence of mucoid secreting neoplastic forms. Many lymphatics are dilated and filled with both immature squamous and transitional cells.

The esophageal epithelium is hyperplastic with intact basement membrane over the tumor, except for the small defect noted grossly. At this area there is a narrow zone of necrosis and underlying fibrous tissue. There is no evidence of neoplastic cell invasion at the margins of this defect. The granular elevations in the lower part of the esophagus (Fig. 1) show replacement of the hyperplastic epithelium by masses of immature squamous cells (see Fig. 5). These cells lie mostly in dilated lymph spaces and are confined to the mucosa and submucosa.

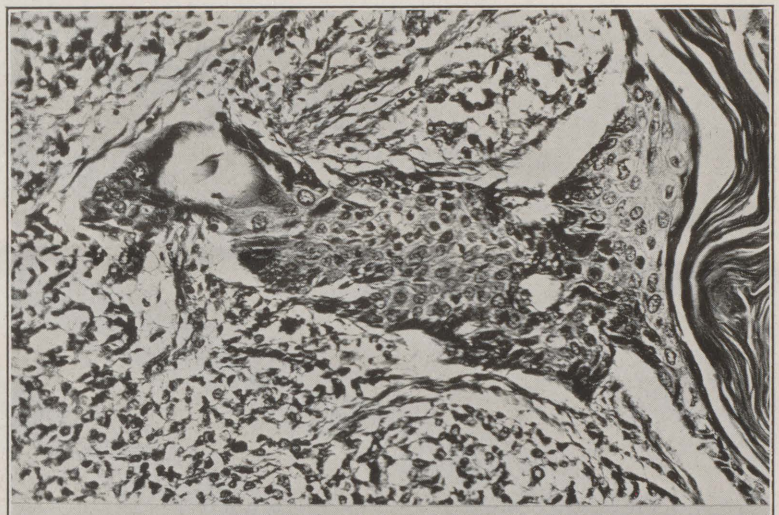

Fig. 3

Fig. 4

Fig. 3.—High power photomicrograph of Fig. 2 at the junction of the squamous and transitional cells. Note the keratinization at the extreme right. The adjoining column of immature squamous cells gradually fuses with the more loosely arranged surrounding transitional cells. (×280.)

Fig. 4.—Photomicrograph of the trachea from the upper third of the tumor. A wedgeshaped mass of dark staining carcinoma cells replaces the marginal columnar epithelium and extends downward radially from the mucosa. (×280.)

Anatomical Summary: Metaplastic squamous-cell carcinoma of the trachea. Direct invasion of the mediastinum, regional lymph glands, and left innominate vein. Lymphogenous metastases to the esophagus. Diffuse aspiration bronchopneumonia.

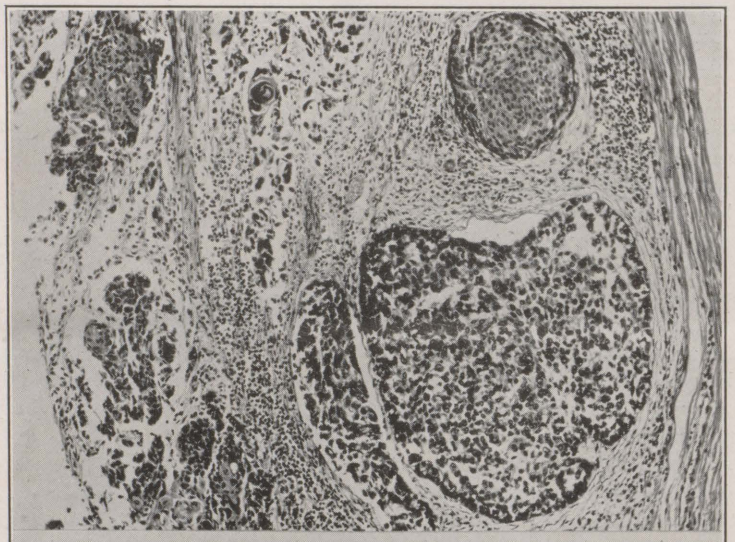

Fig. 5

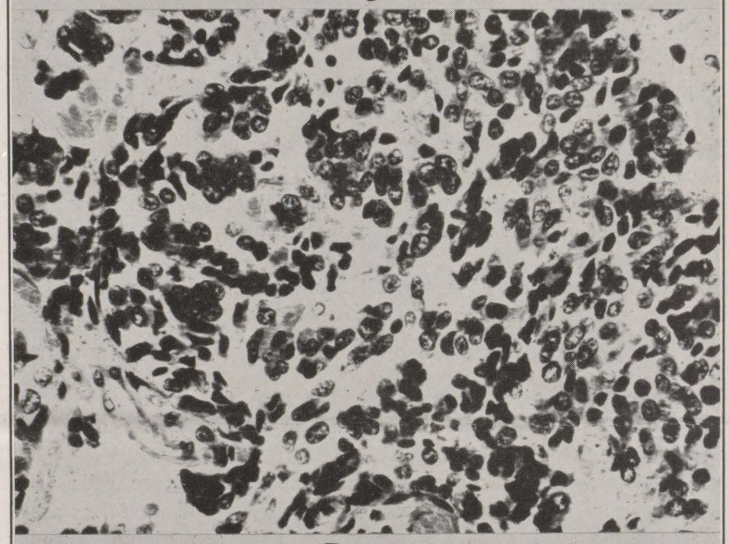

Fig. 6

Fig. 5.—Photomicrograph of one of the esophageal metastases showing the irregular mucosa containing masses of immature squamous cells. Three lymphatic channels are dilated and plugged with tumor cells. The stretched and intact muscular coat is at the extreme right. (×240.)

Fig. 6.—High power photomicrograph of the main tumor mass in Case 2 showing immature squamous epithelial cells and smaller hyperchromatic irregular columnar cells. (×580.)

CASE 2.—*Clinical Course:* This patient was a 54-year-old woman whose illness began May, 1908, when she swallowed a fish bone which stuck in her throat and

was extracted later at home. During the same month she first noticed the sensation of a marble moving up and down in her throat. This became gradually worse with increasing dysphagia. During the next nine months dyspnea was slowly progressive until inspiration became forced and almost gasping. A small round hard slightly tender lump appeared on the right side of the neck near the thyroid cartilage in August of the same year. This increased in size until November and then remained essentially the same size until her admission three months later.

Physical examination showed emaciation, intense dyspnea and orthopnea, some respiratory stridor, marked dysphagia, enlargement of both lobes of the thyroid with fixation to the surrounding tissues, and signs of emphysema. The clinical impression was malignant goiter.

She had repeated episodes of severe coughing and cyanosis. On the day following admission she was said to have been unable to breathe for about a minute, became black in the face and responded finally to strychnine. Two days after admission the thyroid was exposed under general anesthesia and part of the right lobe resected. As this procedure was completed, the patient suddenly stopped breathing and a tracheotomy was done at the level of the third, fourth and fifth tracheal rings. It was difficult, however, to insert the cannula because of infiltration of the wall, and by the time the emergency measure was completed the patient failed to respond to any stimuli and died apparently of asphyxia.

Postmortem Findings: (M. G. H., A-24-09) Autopsy revealed absence of the right lobe of the thyroid and the tracheotomy wound at the level described above. The latter opened into the middle of a large tumor mass which accounted for the difficulty in passing the tracheal cannula. The lumen barely admitted a No. 10 English rubber catheter. When the larynx and trachea were opened, the vocal cords appeared normal, but a tumor was found to extend from the lower end of the thyroid cartilage downward 4 cm. on the left side of the trachea and then slant off toward the right for an additional 3 cm. At one point it completely encircled the trachea, was 4 cm. wide and 2 cm. in its greatest anteroposterior dimension. The mass also extended into the region of the right lobe of the thyroid where the recent operative procedure had severed part of the tumor. There was some ulceration of the tracheal mucosa below the cricoid cartilage on the left posterior wall, and the tumor presented a very firm, grayish-white surface which cut with much resistance.

The esophageal lumen was so narrowed that it admitted a small penholder with difficulty. This narrowing was due to the pressure of the tumor, described above, encroaching on the lumen of the esophagus from the front. The mucosa appeared everywhere intact.

The lungs showed emphysema. Other incidental findings included productive pleurisy and peritonitis, brown atrophy of the myocardium, passive congestion of the liver and slight arteriosclerosis.

Tumor Histology: Sections show irregular masses of large cells with hyperchromatic nuclei in which nucleoli are numerous. Between these forms are diffusely scattered columnar-shaped cells. These tumor cells are divided into irregular masses by bands of dense fibrous tissue. Mitotic figures are numerous. There is no evidence of keratinization or whorl arrangement (see Fig. 6).

Anatomical Summary: Metaplastic transitional-cell carcinoma of the trachea with invasion of the mediastinum and thyroid.

SUMMARY

(1) Of the 433 primary tumors of the trachea on record at the end of June, 1936, 147 or 34 per cent were carcinomata.

(2) The clinical symptoms were essentially the same as those for any tracheal neoplasm and most of the patients died within one year of the initial examination.

(3) The highest incidence of carcinoma was in males between 40 and 60 years of age.

(4) The tumors were located most frequently in the lower third of the trachea on its posterolateral walls.

(5) Metastases were found in 68 per cent of the cases and were mostly regional.

(6) Adenocarcinoma and squamous-cell carcinoma were the commonest types and were found in about equal frequency.

(7) The adenocarcinomata probably originate in the mucous glands of the trachea.

(8) Some of the squamous-cell tumors were assumed to be newgrowths from embryonic arrests of esophageal epithelium in the trachea, while others were assumed to be the result of metaplasia.

(9) Two case studies of primary carcinoma of the trachea have been added to this series.

NOTE: The first case report was obtained from the records of the Montreal Homeopathic Hospital (No. 1743-36) and the second one from the files of the Montreal General Hospital (No. 172-S-09). I am deeply grateful to the staffs of both institutions for their cooperation. I especially wish to acknowledge my indebtedness to Dr. W. H. Chase of the McGill Pathological Institute for his valuable assistance in preparing the pathologic reports of these cases, and to Miss Shirley Goodall for her excellent illustration.

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THE PATHOLOGY AND TREATMENT OF INFLAMMATORY DISEASES OF THE CERVIX

JAMES ROBERT GOODALL, M.D.
and
R. M. H. POWER, M.D.
Montreal, Que.

From the Wards and Research Laboratory of St. Mary's Hospital

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"The Pelvic Tonsil"

James Robert Goodall, M.D., and R. M. H. Power, M.D.,
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THE cervix uteri is a halfway "stop" between two important highways. It is frequently the repository of the defects and weakness of both of these. It is influenced by the physiology and pathology of both of these avenues of approach. It is a barrier to infection, often suffering much in its defense of the sanctum uterinum. Unfortunately, it is situated in the very middle of the "silent area" of the pelvis—that territory bounded above by the peritoneum and below by the perineum, in which gross and grave pathology can, and does, exist without causing symptoms. This area, being part of the visceral field of the body, is almost devoid of tactile nerve endings (just as other intracorporeal viscera) and therefore pain—that announcer of physical ills—is quiescent in the pelvis, and disease may take hold and advance to incurable proportions before the victim is aware of its presence. The painlessness of cervical disease is proverbial. The delicate cervical columnar lining, set upon a functionally unchanging substratum of fibromuscular tissue, is in contrast to the ever changing uterine mucosa which sheds most of its sins of defect every month and renews its vitality with pregnancies. The cervical glands retain the surface scars of battle, the uterine mucosa casts them off—even the major scars of pregnancy. The cervical glands, racemose and deeply burrowing, become harbingers of infection by becoming retention cysts, and inflammatory disease of the fibromuscular tissue influences the superimposed protective columnar or squamous cells, producing in these either hyperfunction or hyperplasia, or both, and, if the process is more destructive, we find a loss of these protective surface epithelia and the initiation of erosions. Long-continued inflammatory irritation may at any time change controlled hyperplasia into uncontrolled invasion which spells cancer.

To understand the pathology of inflammatory diseases of the cervix and the consequences which follow from these, one must know the minute histology and physiology of this organ.

HISTOLOGY AND PHYSIOLOGY OF THE CERVIX

Unlike the uterus, the cervix has no highly specialized intermediate cellular structure such as that in which the endometrial glands are

always imbedded. The racemose, deeply burrowing, cervical glands are imbedded in common clay, an ordinary fibromuscular tissue, differing in no particular manner from similar structures in other parts of the body. This fibromuscular tissue has several functions to perform. It acts as a nucleus into which the supporting tissues of the pelvic floor find an anchorage, thereby allowing freedom of movement to the superimposed uterine body. Movement to this portion is essential to the proper performance of its highest function. Second, the cervical fibromuscular tissues act as a sphincter to the uterine contents. To permit wide dilatation of this sphincter without undue destruction, the muscular element must greatly outweigh the fibrous element, thereby permitting of an elasticity which would be otherwise impossible. This normal proportion of muscular (sphincteric) and fibrous (supportive) tissues is frequently altered, always to the detriment of the muscular element, by errors in development, abnormal puerperiums, infection, newgrowths and age.

Musculofibrous tissue of the cervix differs in its pathology in no particular sense from similar tissues elsewhere in the body, except owing to the presence of two factors: the influences brought to bear by being invaded by glands, and the influences of the functional phases associated with procreation.

The glands of the cervix are a protective mechanism; they are constituted by digitations of a common duct lined by a single layer of tall columnar cells. These are mucous goblet cells, in which normally the tall cell has a nucleus near the base and an open end toward the lumen, like the cup of a tulip. Below these is a layer of flattened cells constituting the so-called basement membrane. Normally, the quantity of secretion is just sufficient to fill the cervix with a tenacious stringy plug which protects the uterine cavity from invasion. It flows slowly like a glacier. This plug liquefies, when in contact with the acid secretion of the vagina, and acts further as a lubricant to this canal. The reason for the digitations of the cervical glands is found in the desire to increase the extent of functioning surface and thereby lessen the function of each individual cell. It is a maximum of function with a minimum of exposed surface. Unlike the endometrium, the cervical tissue has a permanency which may act to its own detriment. The uterine mucosa, by shedding its surface, also sheds many of its diseases, as will be pointed out in another paper by me. The cervix retains the scars which it cannot heal by the ordinary corporeal reparative processes. The processes associated with procreation come but slightly, if at all, to its aid. The cervix undergoes slightly transitional changes with menstruation, and marked hypertrophy, increased vascularity and glandular function during pregnancy, with regression during the normal puerperium, but it has not the happy faculty of casting off the scars of this maelstrom

of cellular disturbance. It must depend upon the general recuperative properties of the body as a whole for its reversio ad integrum. The tissues of the genital tract suffer from a state of unrest, a flow and ebb, that may affect other parts of the body, but to a much less degree. This state of flux may operate to the advantage, or disadvantage, of the affected tissues. But it is often productive of pathologic states, or it may greatly modify these when they are present.

Inflammatory changes of the cervix may be simple or special, acute or chronic, or they may spend themselves chiefly upon the cervical glandular structures affecting the fibromuscular layers minimally, or they may affect these component tissues in the reverse order.

General.—Most inflammatory cervical diseases begin as an acute process and degenerate into chronic catarrhal states. Others are acute and short-lived. It is characteristic of acute mucous membrane diseases that, when they end abruptly, they leave a minimum of change in tissue or function. Chronic inflammatory disease, on the other hand, is productive or destructive, according to its intensity relative to the body reactions. Productive inflammatory diseases, that is, the more chronic processes, express their production in either hyperfunction or hyperplasia. The destructive types produce loss of tissue, expressed as erosions or ulcerations.

There are other functions which initiate productive changes in the cervix, essentially causing enlargement of the fibromuscular tissues, chiefly with minor incidental, or major accidental, changes in the endocervical epithelium. These are the cervical hypertrophies of pregnancy without normal retrogressive changes in the puerperium, leaving a permanently enlarged cervix usually associated with cystic disease. Other productive cervical diseases may arise out of the stimulus of corporeal fibroids. These, of course, simulate a pregnancy, causing hyperplasia of all the tissues as in pregnancy. After all, fibroids are the barren woman's children. When the uterus has prepared the functional endometrial and allied changes repeatedly without issue, fibroids develop as a pseudopregnancy. The influence upon the cervix is frequently quite similar. Such hypertrophic changes when not followed by retrogression, as in the normal puerperium, result eventually in a large, hard cervix, which, when associated with cystic disease, may assume large proportions. Since this is most commonly the aftermath of an abnormal puerperium, the cause, or causes which inhibited the reduction usually also affect the uterus, causing it to remain in a state of chronic subinvolution, thereby remaining globular, tender, large, superimposed upon a cervix similarly affected. In fact, the condition of the cervix is being recognized more and more as the index of the state of the uterus, and this applies as much to the mucosal as to the fibromuscular changes. Such conditions of the cervix should be distinguished by the name of "chronic interstitial

cervicitis,'' as contrasted with purely catarrhal endocervicitis, in which the disease is restricted wholly, or chiefly, to the glandular structures.

Acute Catarrhal Endocervicitis.—Acute catarrhal endocervicitis may arise from any acute infection of the cervical glandular surface. It may occur independently of pregnancy or as a result of the traumatism of labor. The common agents of acute and chronic disease are the gonococcus, the streptococcus and the long-continued irritation of trichomonas with its associated symbiotic organisms. The former is, in the vast majority of instances, merely a surface and subsurface infection, producing hyperfunction and cystic degeneration, owing to duct constriction and round-celled infiltration. The secretions are usually purulent, but may later be merely superabundant or even show no appreciable departure from the normal. The postpartum cervical changes incident to an acute infection were described by me* where a streptococcal film is frequently seen over the cervix in the early days following delivery. As chronic endocervicitis is the common sequela of an abnormal puerperium, a few lines devoted to the subject may be enlightening.

It is variously estimated that from 50 to 70 per cent of women show a chronic endocervicitis following pregnancy. I was one of the first to draw the attention of the medical profession to this abnormality. It is not surprising that it is so frequent. The condition of the cervix and its mucosa immediately after labor, especially after first labors, cannot but cause one to respect the great recuperative power of damaged tissues in the puerperal state. When one sees the ecchymotic endocervix, torn from its moorings, frequently hanging like a veil, bloody, bruised, and lacerated, and the fibromuscular tissues torn, divulsed, gray and necrotic by counterpressure from pelvic bony structures against the presenting part, we repeat, one is struck with admiration at the recuperative power of the puerperal woman. A repeated examination five or six weeks later, in a large number of cases, shows the miracle. But the miracle is seldom complete. The fibromuscular structures, like all mesoblastic tissues of the body, have a larger recuperative power than a more highly specialized tissue. Consequently, the glandular tissues suffer more and recover less of their high specialization. Columnar surfaces are the great weakness of the body. It is from these that most diseases find their portal of entry, or local permanency, so that local infections of their surfaces have a faculty of becoming chronic, irritative and vitiative of normal functions.

The diseases of the nasal mucous membrane bear a close resemblance to those of the cervix uteri.

*American Journal of Obstetrics & Gynecology **16**: 339, 1928.

Chronic Endocervicitis.—The picture of chronic endocervical disease from a microscopic standpoint varies greatly with its intensity and duration. In the mildest form there is merely increased activity of the surface columnar epithelium; chiefly that part nearest the cervical canal is most profoundly affected. The deeper one recedes from the canal into the digitations of the glands, the more normal the columnar cells become. This is quite the commonest picture.

The Hypersecretive Types of Endocervicitis.—If we examine the columnar cells more closely near the canal, we find that the goblet part of the cell, that is, its free margin, is more ragged, and is expelling huge quantities of mucus (Fig. 1). The nucleus has moved away somewhat from the base of the cell, and may occasionally ascend as far as the middle, leaving a clear space underneath it. Tinctorially the nucleus does not stain so deeply. A drop of this cervical secretion, pressed between a slide and cover slip, shows a most impressive

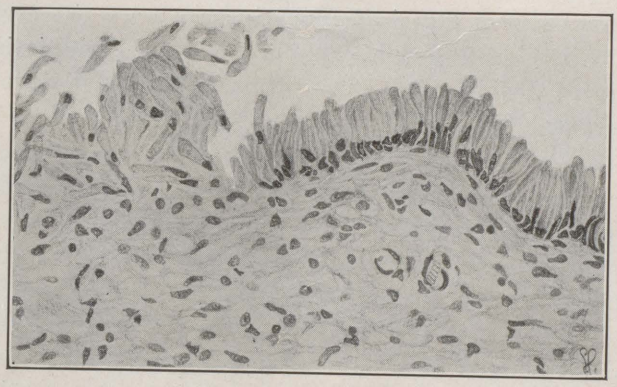


Fig. 1.—Showing to the right marked hyperfunction of the columnar cells, with desquamation of the cells by a sort of explosive excess of function.

picture of thousands of mucous masses shaped either as clubs, sausages, or ovoids, lying in a stream of thinner matrix, with their long axis in the stream line. Each globule has a surface of higher tension which causes it to retain its shape until this is dissolved. These globules are highly granular, and are very beautiful and highly refractile. They may be easily mistaken for the highly refractile encysted types of trichomonas. But their arrangement in streamline fashion at once affords a differentiation. In the more advanced and more intense chronic infections, this state of hyperfunction expressed as hypersecretion may cause a complete explosion of the secreting (Fig. 1) cells, with or without attempts at repair in the deepest basal (Fig. 2) membrane. In still other more acute stages, a wholesale blight may affect all the columnar cells, causing them to be cast off in a state of partial disintegration, not only of the surface, but of all the deeper glands. Secretion is not a prominent feature of this stage, rather destruction captures the attention.

Hyperplastic Type of Catarrhal Endocervicitis.—In other cases, hyperplasia of the glandular linings becomes the predominant feature. This may spend itself chiefly upon the glandular elements affecting the

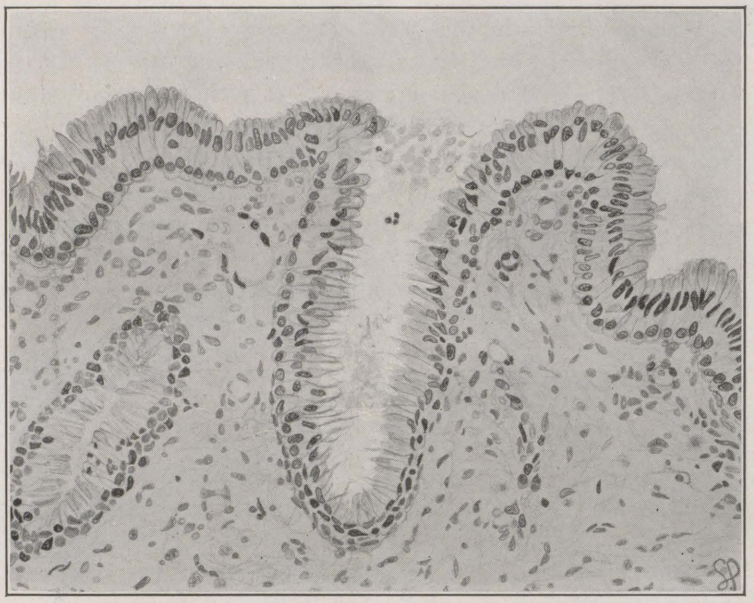


Fig. 2.—Catarrhal endocervicitis. Note the duplication of columnar lining. The more superficial layer is being desquamated by the deeper layer under the stimulation of disease. The underlying tissues are edematous.

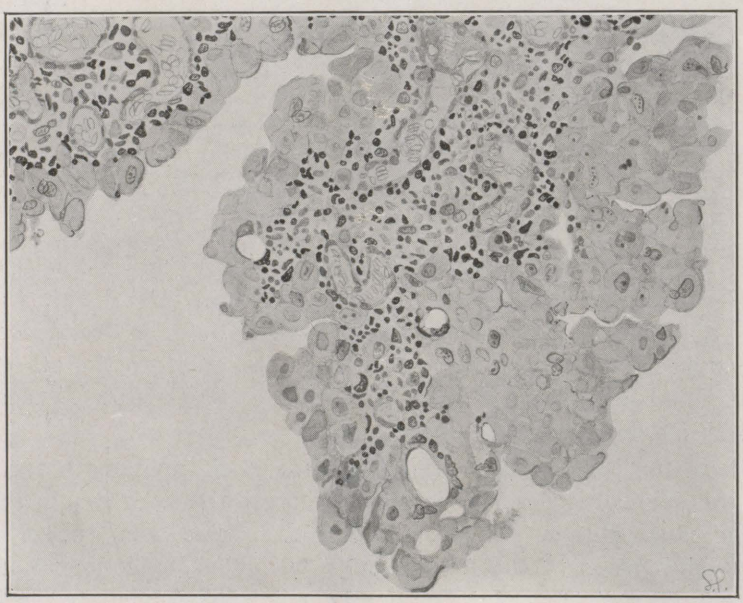


Fig. 3.—Subacute inflammation of the endocervical tissue. There is round-celled infiltration, great vascularity and inordinate multiplication of the columnar cells that are often multinucleated and syncitial in character.

fibrous tissue minimally. Hyperplasia of the glandular elements results in a many layered covering, instead of a single layer (Fig. 3). In many instances, this gives rise to a heaping up of tissue in which secretory function is partially, or totally, lost and cell energy seems to spend itself chiefly in division. These can so closely approach a cancerous stage as to qualify as precancerous, a vague term without justification. The cell division may affect either the columnar cells or the squamous covering. These two types of cells may so revert to their embryonic characters as to be indistinguishable. The columnar cells lose their columnar mucus-secreting characteristics, and the squamous cells cease to be keratinized, become active, and the surface cells are shed before they can become adult as in the normal state. The underlying fibromuscular stroma is usually but slightly affected by the mild infection.

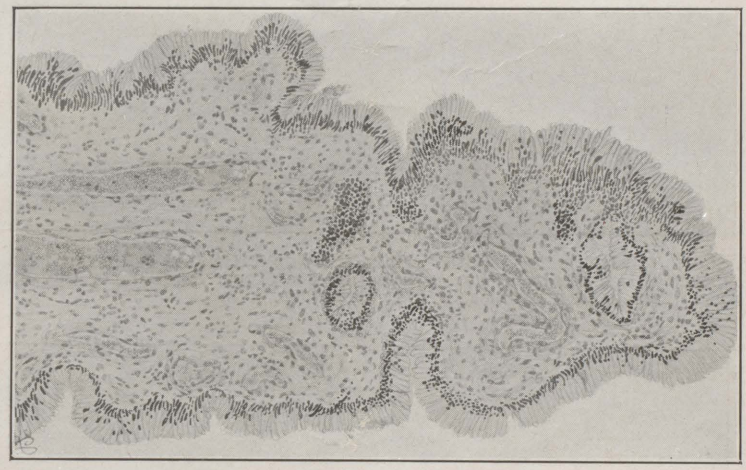


Fig. 4.—A polyp of cervical canal, showing catarrhal changes in the columnar covering cells, edema of the fibrous core and multiplication of blood vessels, a vascular edematous polyp in which both supporting and special cells are involved in a vascular hyperplasia.

Acute Interstitial Cervicitis.—It will be readily understood that the division of cervical diseases into catarrhal and interstitial, and into acute and chronic types, is a purely arbitrary one for descriptive purposes. These types pass insensibly from the one into the other. But the division is one that is generally accepted for similar disease processes in other parts of the body.

Where the inflammatory attack spends itself upon both the lining and the deeper tissues (predominantly postpartum cases) in addition to the mucosal changes, described above, one finds diffuse round-celled infiltration of the deep tissues, with the development of large cervical polyps (Fig. 4), frequently much pendunculated, with an infected, desquamated surface at certain points, and a loose edematous fibrous core. Thrombophlebitic changes are frequent and conse-

quent diapedesis or large venous hemorrhages may occur, not infrequently followed by sloughing. Cystic disease of the glands usually occurs only in the chronic state.

Chronic Interstitial Cervicitis.—This is usually the result of an infection following full-term labor or abortion. The characteristic clini-

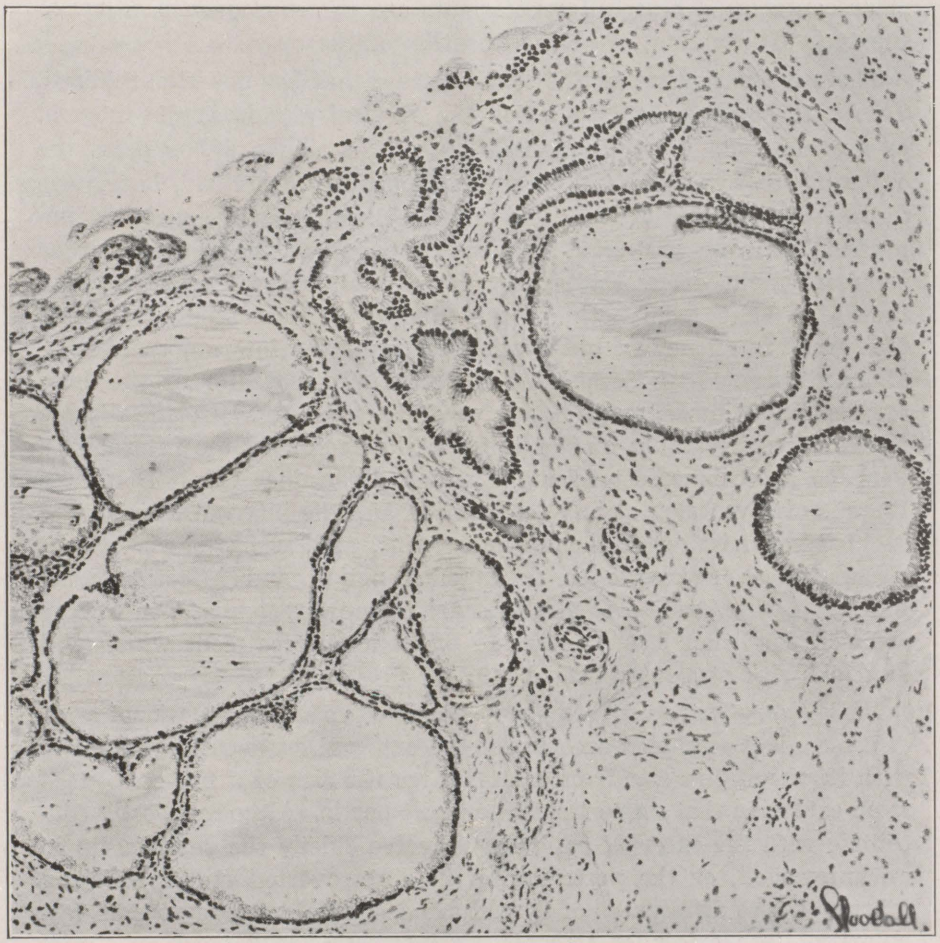


Fig. 5.—Showing eroded surface and multiple cystic disease of the cervical glands, with pressure degeneration of the lining cells. This is at the external os.

cal signs are an inordinately large cervix, the result of chronic subinvolution, with numerous nabothian cysts, which greatly add to its bulk (Fig. 5).

Owing to the hyperplasia of connective tissue and the frequent collections of lymphocytes resembling lymph nodes developed, we think, from the reticular tissue lining the lymph spaces, fibrous constriction of the ducts of the racemose glands occurs, and retention cysts occur

as a consequence of secretory pressure. When the intracystic pressure equals the cellular secretory pressure, then secretion ceases and regressive atrophic changes occur in the lining epithelium, causing all degrees of flattening and frequently complete atrophy of the lining cells. The contents of the cysts may vary from thick tenacious mucus at first, later, purulent, or inspissated white mucus, or liquid clear contents. These different results are the consequence either of the type of infection, or of the duration of the cysts.

There is one feature of this cystic disease that has not been realized. This is the frequency with which the cervical glands at the internal os are affected. Cystic degeneration of the glands at, or near, the external os are visible and therefore frequently described, but cystic disease of the internal os with partial occlusion of the canal and imperfect drainage are very frequent occurrences, in fact, almost as common as the discreet type, but of infinitely more interest because these can arise from both an ascending or a descending infection. The great frequency of old cystic disease of the internal os has another interest. In a descending infection of tubercular origin there are certain points of predilection or sedimentation where the disease metastasizes. Notably one of these is the region of the internal os. Perhaps it is imperfect drainage which determines this. Doubtless there is always delay at any constriction and the internal os is a very decided constriction of the uterine cavity, and, where stasis occurs, circumstances favorable to deranged function and infection must follow. Cystic disease of the internal os, now recognized as a very common condition, has a profound effect upon treatment which will be dealt with at a later period.

Ectropion.—Ectropion, commonly described as erosion, cervical ulcers, et cetera, is a reddened area about the external os, usually concentric with it, varying in size from a small area the size of a five-cent piece to an extension that may involve the whole portio. Although mostly concentric with the external os, it may involve chiefly the anterior lip in an anteverted, or the posterior lip in a retroverted and retroflexed uterus. These differences of spread are accountable to degrees of vascularity. The origin of ectropion is interesting. It can never exist without antecedent endocervicitis from which it springs, and it cannot be cured without first or simultaneously curing the endocervicitis. Endocervicitis is primarily an infection, usually a chronic one ab initio, or degenerating into a chronic state from an acute one. Infection determines blood to the part, which, if prolonged, leads to the development of new capillaries. This congestion leads to certain defects of function, which expresses itself in hyperfunction (leucorrhea) or hyperplasia (division) or both. Hyperplasia does not necessarily have to be accompanied by hyperfunction, so that grave states of endocervicitis may be devoid of signs of leucorrhea. When congestion

has gone on for a considerable period, it leads to hypernutrition or deranged nutrition. The changes described above under chronic endocervicitis now develop. It is only a matter of time before the squamous epithelium, covering the portio, begins to feel the effects of this congestion, and the part that will feel it most forcefully will be the tissue in the immediate neighborhood of the canal, namely, the external os. The basal layer of the squamous epithelium here begins to multiply under the stimulus of hypernutrition. As the division is speeded up, the cells have not time to become adult, imbricated and keratinized before they are pushed off by the press of new cells. The result is a reddened area made up of granulation tissue composed of embryonic squamous cells. This process gradually, but very slowly, extends to give rise to an increasing area of ectropion. If one examines such a section microscopically, beginning from a normal portion of the cervix and thence gradually approaching the region of the internal os, one finds the squamous layer gradually losing its thick keratin layers, and the whole covering growing thinner until at the margin of the ectropion there remain only the basal layers, arranged as deeply staining vertical cells with a layer or two of embryonic cells covering these. Further on, only a single layer remains, and, still further, even this disappears, leaving only occasional islands of cells of squamous origin on a granulating, small-celled infiltrated surface. Islands of highly modified squamous cells may still be found here and there, especially in the neck of the glands which open onto the surface. It is from these that recovery takes place when effective treatment has been applied.

It may be stated with assurance that the common teaching that under inflammatory stress columnar covering may replace squamous epithelium over an area of ectropion is not only wrong theoretically, but also wrong in our experience. Our specimens were taken entirely from the total hysterectomies at operation. The vagina was washed out with a gloved hand only, and liquid green soap. Immediately after the operation the uterus was opened lengthwise, examined, recorded, and a strip about ⅓ cm. thick was then taken from the fundus to the portio, and immersed immediately in bichromate of mercury in formalin. In the majority of cases this strip was cut in one section. If too long for section, it was cut across above the internal os and its continuity was maintained on two slides instead of one. Under these most favorable conditions, where tissue was not more than one hour out of the body before being hardened in its easily penetrated strips, it can be definitely stated that normally squamous cells end abruptly where columnar begin. The transition is from one definite type to the other in a clear-cut line (Fig. 6). In the normal there is no room for any doubt on this point. Second, squamous lining normally dips down into the gullets of those cervical glands

which open onto the squamous portion. This is for protection of the more delicate columnar lining. Third, there is no definite line where columnar lining ceases in the region of the external os. It varies in the height it may invade the cervical canal in different individuals. Fourth, columnar epithelium never replaces squamous epithelium where the underlying soil is hostile to the more hardy

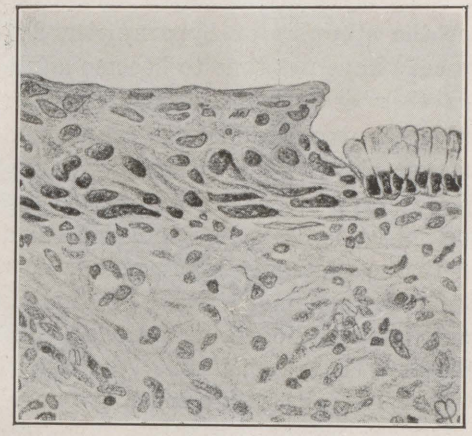


Fig. 6.—Normally the transition from squamous to columnar cells is abrupt.

Fig. 7.—Where cervical glands open on the portio, the more hardy squamous lining dips into the mouths of the glands to protect the more delicate, highly specialized columnar lining.

squamous covering. Where squamous cells cannot survive, it is not possible that a more delicate columnar cell could accommodate itself. Under certain circumstances of change, linear arrangement of the basal layer of a squamous covering may simulate that of columnar cells which are undergoing hyperplasia. But this cannot be interpreted, by any stretch of the imagination, into an interpretation of a replacement of squamous by columnar cells. We repeat, such a con-

tention is not only illogical, but contrary to experience. Wherever there is much surface contact with externals, nature interposes squamous epithelium as the safest guard, and the transition from squamous to columnar surfaces is proportionate in degree to the diminution of these contacts. This is a general rule of physiology. Columnar surfaces, and transitional epithelia, on the other hand, under the influence of irritation and frequent contacts, can become hardened and stratified to resemble a squamous covering, but serration and other features are generally absent. The cervical canal and the distribution of its cellular elements will repay a consideration. The canal varies considerably in shape and size under varying circumstances. Normally, it is fusiform, small at top and bottom with a slightly wider center transversely to the body. The anterior and posterior walls lie in contact, each with its frondlike arborvitae arrangements of its folds which by their inclination favor downward drainage. The glandular elements penetrate more deeply into the substratum and are more numerous near the external os. A few open onto the portio. Glandular elements grow less penetrating and with fewer digitations, the nearer one approaches the internal os. The glands are set in adult fibromuscular tissue. One does not encounter the specific interstitial cells of the endometrium until well beyond the internal os, and as soon as one meets these cells, the imbedded glands change immediately into specific uterine tubules.

The depth of tissue involved in glandular invasion at the internal os is singularly thin, and, in abnormal states, is frequently cystic. One can readily see how easily the region of the internal os can be injured and cicatrized by inflammation or traumatism, where glandular recuperative power must be slight where it is so scant.

Under inflammatory disease the canal gradually widens. In nulliparae and virgins this widening involves chiefly the middle portion. The internal and external orifices are more resistant, owing to their greater protection by the uterus above, and by the squamous covering below. The result is a craterlike enlargement of the canal with stasis of secretion or blood. At postmenstrual periods stasis increases the effect of infection. Eventually the periphery of the external os becomes secondarily involved in ectropion and the os widens spontaneously. Presumably similar changes take place at the internal os. It is common experience to find both the external and the internal ora open widely when one cauterizes nulliparas or virgins where prolonged cervical disease has been present. That the cervical disease eventually invades the endometrium by extension upward, we have abundant proof, which will be reserved for another work. That it produces ectropion by extension downward admits of easy demonstration. The surface layers of the endometrium, however, remain immune to this upward extension, owing to their desquamation and re-

newal at each menstrual phase. The upward extension of chronic endocervicitis is limited to the deep layers of the uterine mucosa.

CAUSATIVE AGENTS

There are immediate and contributing agents. The immediate agents are of course microbic. These are chiefly streptococcic, but also gonococcic, trichomonas symbiosis, and rarely of other types. Contributing factors are of the greatest importance. Of highest frequency, of course, are the results of full-term labors and septic abortions. In the former, the traumatic and disruptive influences are the agents which supply the favorable soil. But defective drainage during the recovery stage lends an additional disadvantage. When a woman lies supine, the vagina drains uphill at an angle of about 35 degrees. As a consequence, the traumatized cervical mucosa lies in a cesspool of lochia, from which, after forty-eight hours postpartum, many strains of microorganisms can be cultivated. Each succeeding pregnancy adds its quota to the initial invasion. Carcinoma of the cervix is a rarissima avis in the nullipara. It is a disease restricted to the parous woman. One might almost say, uniformly so. The cause lies not in accidental tears of the cervix, but in the irritation of a chronic endocervicitis or its consequent ectropion. Tears of the cervix play no part in this process, except as a contributory factor in exposing the delicate cervical mucosa to traumatism, or in making it more easily accessible to infection. A great deal too much importance has been attributed by clinicians to cervical tears. Their repair is a matter for profoundest consideration, not only as regards the dangers, but also owing to the almost complete futility of the operations that are commonly used for this purpose. Another contributory cause is found in that type of individual whose mucous membranes are all susceptible to overgrowth, tonsils, adenoids, intestinal and endometrial. By heredity, or acquisition, they are prone to develop disease of the mucous membranes. Other chronic diseases which lower the general resistance may be strong contributing factors in the development of cervical disease.

SYMPTOMS AND SIGNS

The symptoms of endocervicitis are local, extensive and general.

In the nonpuerperal state, acute endocervicitis produces at first leucorrhea, copious and purulent. Later, there frequently develops a feeling of weight in the pelvis. Menstruation may be retarded and then prolonged and profuse. How much of these last symptoms may be due to involvement of the endometrium and appendages, which may develop by extension, it is impossible to tell. Pain, in uncomplicated inflammatory cervical disease, is conspicuously absent. Extension frequently takes place to the endometrium, which is not diagnosable until the appendages are reached. Pain now becomes a conspicuous

symptom. Local or general peritonitis of a mild or severe degree follows. Every acute case tends to become chronic in time. Occasionally spontaneous cure follows immediately upon the acute stage. General symptoms of malaise, backache, loss of appetite, indicative of a toxemic state may or may not follow. Occasionally, though fortunately rarely, general blood infection, without appreciable involvement of the pelvic organs other than the cervix, may develop, causing a general septicemia without localization, or multiple involvement of synovials, endocardial, pleural, meningeal and lymphatic. Pyemia is rare.

In the acute puerperal state there are all degrees of activity. In the majority, the disease remains a local involvement of the cervical mucosa and its lacerations. There are no distinguishable symptoms in the early days of the puerperium. The appearance of the cervix after the third day, described in a previous article and lecture before the Philadelphia Obstetrical Society, shows an edematous state, frequently with a film, a streptococcal membrane over the visible part of the cervical canal. This may be easily wiped off. Symptomatically, however, one finds that the puerperal temperature does not remain normal. There is a daily rise to 99° or 100° F., over many days, with a total absence of pain. This is indistinguishable from a low grade pyelitis, or an inconspicuous pelvic thrombophlebitis. Forty per cent of cases of pyelitis are pain-free. Pelvic thrombophlebitis may be suspected but cannot be diagnosed, except by its mechanical complications. So the mildly febrile pain-free cases in the puerperium may be either acute endocervicitis, pyelitis, or thrombophlebitis. In about 90 per cent of cases the correct diagnosis will be endocervicitis. It is not advisable to expose the cervix and confirm the diagnosis. The cervix is retarded in its involution, as is also the superimposed uterus. This applies equally to the ligamentous structures. Extension of cervical disease may be by continuity of mucous membrane, or by the lymphatic channels. In the former type, pain due to peritoneal involvement is frequently late in development, often involving weeks, with the interval of only comparative well-being. May we outline a recent case the better to illustrate this important, and all too frequent sequela of labor?

I was called in consultation to see a case which had been delivered in hospital one month previously. Her labor and puerperium had been clinically absolutely normal, and symptom-free. She went home on the twelfth day. Ten days later, she had a severe pain in the pelvis, midline, which subsided after one-half hour. Two days later a similar attack developed. This was repeated two days later, but it did not subside this time. Her doctor sent her back to hospital. When I saw her, she had an acute abdomen limited to the lower half, with exquisite tenderness especially over the right side. Naturally, owing to the recent labor, one thought of pelvic complications. Vaginally and rectally nothing but a fullness in the pelvis with great tenderness on imparting movement was elicited. Leucocytes 22,000. There had been nausea and vomiting in the last six hours. Appendicitis could not be ex-

cluded, but pelvic peritonitis was suspected. The abdomen was opened along the right rectus. The appendix was free in its proximal two-thirds, but was involved secondarily between two swollen, indurated tubes, curved each about its ovary. The appendix was liberated and removed and the abdomen closed. The patient made an uninterrupted recovery. There was absolutely no history of gonococcal infection.

It is now becoming more and more recognized that such ascending infections of a mild streptococcal nature are exceedingly common. They are insidious and slow in development. They have been of frequent occurrence in my experience. Smears showed only streptococci. Most of these are mistaken for gonococcal disease. They run a similar, but milder course usually. Sterility may follow, but not so consistently as in gonococcal disease. In the cases where the endocervicitis spreads by lymphatic extension, the first sign of involvement of the peritoneum usually comes on during the early puerperium. A sudden severe state of pain in one or the other lower quadrant, more frequently the left, usually leaves no diagnostic doubt. There is tenderness and a sudden rise of temperature which subsides under appropriate treatment.

Examination, both bimanually and by exposure of the cervix, of all puerperal cases five to eight weeks postpartum, should be a routine. Under these circumstances endocervicitis will reveal itself by a prolonged lochia, followed by a copious leucorrheal discharge, probably a degree of subinvolution of both cervix and uterus, very likely tender uterosacrals and backache. On exposure the cervical mucosa secretes a tenacious clear or semipurulent plug. The canal is more patulous than normal and the mucosa has an unhealthy edematous, pale red appearance. In the chronic state, which is by far the most common type that one sees, the patient usually complains of a leucorrhea of long standing, but growing progressively worse. In young girls and nulliparas, this may be the only symptom. On examination there is a velvety feel about the os which is characteristic of ectropion. When this is present the external os assumes proportions of patulousness that are unusual in nulliparas. When the condition has not reached the stage of ectropion, one frequently finds the external os closed, but, on opening it with a forceps, a great quantity of tenacious mucus from a largely dilated, craterlike canal is obtained.

The ectropion, when present, may be slight or extensive, but rarely exceeds a ten-cent piece in size, in these cases. It is red, granular, and bleeds when cleansed.

In multiparas the process is usually much more extensive. Each pregnancy and labor adds its quota of exacerbation. The leucorrhea becomes more abundant and more troublesome during pregnancy. The patient may complain of backache, fatigue, and poor health. That

poor states of health may be due to this "cervical tonsil" is confirmed by the great improvement which follows upon effective treatment of this condition.

There are two types that are easily distinguished. In the first, the disease is confined almost exclusively to the involved mucous membrane and its ectropion, *chronic catarrhal endocervicitis.* In the second

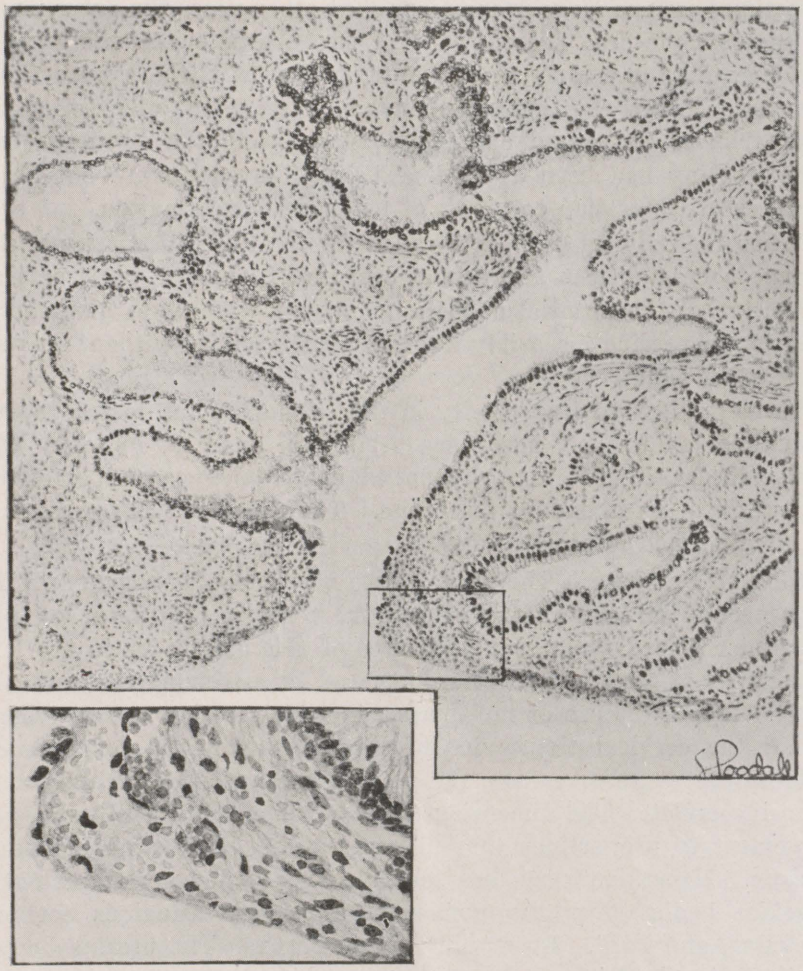


Fig. 8.—*A,* Surface erosion of the edematous, hemorrhagic type without much round-celled or leucocytic infiltration. *B,* The same magnified; note absence of covering columnar epithelium and great vascularity.

type, the cervix as a whole is involved, *chronic interstitial cervicitis.* This distinction is of paramount importance. There are intermediate cases, but generally speaking, they fall conveniently into these two big groups.

Endocervicitis with Ectropion in Nulliparas.—The cervix is more patulous than normal. There is a copious, clear, tenacious discharge;

occasionally this is white with mucous corpuscles, or even semipurulent. The mucous membrane of the canal is occasionally pale and edematous, or red and granular; sessile or pedunculated polyps may fill the visible portion of the os (Fig. 8). The area of ectropion may be extensive, usually concentric; granular and bleeding points are frequent when the surface is cleaned. Frequently a large vessel will emerge from the granular ectropion and course outwardly to the periphery. These sometimes assume large proportions. At other times the outer margin of the ectropic area presents one of two characteristic pictures. In the one, there is a blueness at the periphery as if there were venous congestion below the thin squamous margin. In the other, there is a white rim of heaped-up epithelium surrounding the ectropion as if sugar-coating had been applied. These differences arise out of differences of congestion and lack of balanced desquamation.

When the cervical canal is invaded for treatment, its irregularities, owing to enlargement and sessile overgrowth, become apparent, and dilatation of the internal os is frequently so wide as to give the impression of passing insensibly into the uterine cavity without definable limit to the canal.

These findings of irregularity in the cervical canal, owing to overgrowth of epithelium, may vary greatly in degree. In many cases the endocervical infection will spend itself chiefly in hyperfunction expressed in an abundant discharge. The difference is one inherent in infection and local resistance.

Chronic Cervicitis.—This is a disease of the parous woman. It is a sequence of an abnormal puerperium, whether full term or postabortum. The distinguishing features of this disease lie in the large dimensions of the cervix, its hardness and its associated cystic involvement. It is a disease of multiparas, in which, owing to involvement of all the cervical tissues in infection, or owing to some general debilitating cause or causes, the normal involution of the cervix has been incomplete. The dimensions vary considerably from a slight enlargement to proportions that fill the vault. Hardness is a characteristic. Nabothian cysts are the rule and frequently stand out as sagolike bodies over the portio. The cervical canal is tortuous, granular, and hard. Everything seems fibrous. The uterine body is similarly involved in a state of chronic subinvolution, as evidenced by a large globular symmetrical, usually tender corpus. This condition is frequently described in textbooks as "chronic metritis," "chronic fibrosis uteri" and "arteriosclerotic uterus." Endocervicitis and ectropion usually accompany the cervicitis but these may not be prominent.

It is most important to distinguish the cases that are dominantly endocervical from those that are dominantly cervical, because the treatment is essentially different.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of inflammatory diseases of the cervix from other conditions lies essentially between advanced cases of chronic inflammatory disease and incipient carcinoma. We know of no rule, except clinical experience and biopsy.

The insidiousness of the change from inflammatory disease to new-growth cannot be better illustrated than in the records of St. Mary's, where, in the pursuit of this work, two cases of unsuspected carcinoma of the cervix were found in the microscopic study of uteri removed by total hysterectomy by the authors, in the past year, roughly, somewhat over 2 per cent was found in 90 cases. Two others were placed in the category of precancerous hyperplasia, a rather loose term to designate overgrowth with solid masses filling the acini, but no evidences of breaking through the normal external glandular boundaries.

TREATMENT

The whole of this treatise has for its object a plan to outline a *logical* treatment of inflammatory cervical diseases. The conclusions that are about to be expressed are based upon a studious knowledge of pathology and a wide clinical experience. It is an expression of very firm convictions.

The subject may be dealt with most convincingly under three headings.

1. Nonsurgical treatment.
2. Surgical treatment.
3. The influence of a residual inflammatory cervical disease upon pelvic operations.

1. *Nonsurgical Treatment.*—There are very few forms of treatment of the inflamed cervix that offer any hope of recovery or even amelioration other than the thermocautery. Prior to its introduction, the medical profession leaned strongly to preparations of tincture of iodine, carbolic acid and other escarotics. Later, diathermia and "Elliot" enjoyed a vogue. These have been abandoned for the more rapid, more controllable and more effective electric thermocautery.

It is chiefly to the efficacy and limitations of this presently universal treatment that we wish to draw particular attention. Thermocautery is a very effective form of treatment when properly applied in suitable cases, especially if one does not wish to destroy cervical function. Where, on the other hand, one wishes to destroy function, the matter is quite another affair. Under these circumstances, cautery must be deep and thorough, or, if the cautery knife be used, it must go deep enough to destroy all the glandular tissue. Thoroughness is here the keynote.

However, in those more numerous cases where cautery is used to re-establish normal cervical function, the limits of this form of treatment

are very circumscribed. An excellent result can be anticipated only in those cases that are mild, and limited chiefly to the surface epithelium lining the cervical canal: chronic catarrhal endocervicitis. It is most effective in nulliparas, and in early treatment after each delivery. It becomes progressively less effective the more widespread the disease, the deeper it has involved the cervical stroma, and the greater the organic departure of the cervix, as a whole, from its normal size and consistence.

It has been stated, in the subject of pathology, that in the great majority of the milder cases the pathologic changes are more pronounced at the canal surface, and that the glands are more normal the deeper one recedes into the cervical parenchyma. This can be demonstrated in a very large percentage of chronic mild or recent subacute inflammatory implantations. This is just the opposite of

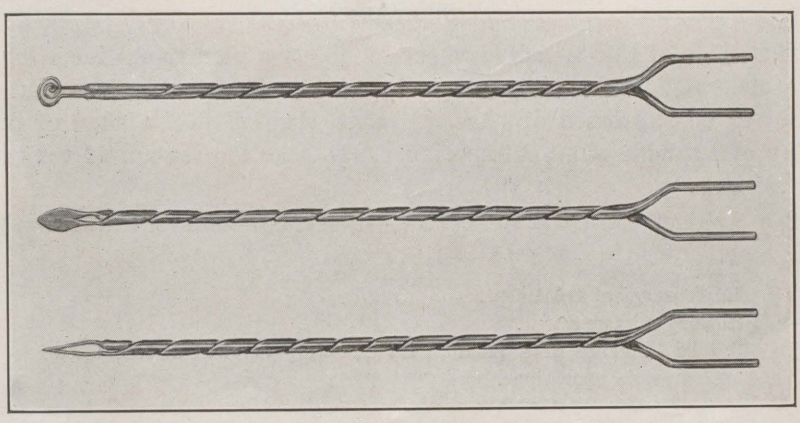


Fig. 9.—Cautery points used by authors in the cure of chronic catarrhal endocervical lesions. The instruments are heated only at the extreme one-half inch. The shank is never more than warm. Natural size.

what happens in the endometrium, where the surface sheds its diseases but allows them to become permanent in the deeper layers. The cervix, therefore, conforms to the general rule of tissues. The uterus is the exception, owing to its special function.

Now, it is just in these mild chronic cervical canal diseases in nulliparas and in recent subacute infections that treatment has proved most effective. In such cases logical treatment destroys the surface layers, and allows the deeper-set normal glands to regenerate a new lining, which they will do, most effectively, if not destroyed. A minimum of scar tissue is caused by such treatment. In nulliparas, one should be careful not to cauterize the external os overvigorously where there is no ectropion. Where, on the other hand, there is ectropion, there is no such risk because the larger the area of ectropion, the more will the external os be patulous and soft. In cauterizing the canal it is not necessary to cleanse the canal of mucus

with caroid or other solvent. In fact, fluid in the canal is a great advantage, for, when it boils, it distributes the heat equally and generally over all the interstices of the canal, so that there is no necessity of bringing the cautery point into contact with the canal epithelium at all. When the canal presents a whitish or parboiled appearance, the treatment has progressed far enough. Treatment so effected will not produce a pocketed and tortuous cervical canal, because the heat is evenly distributed. Frequently, in the next ten days, a thin cast of the canal is shed or the necrotic tissue is disintegrated, leaving a pink healthy mucosa. Care not to constrict the external os is effected by using the cautery type as illustrated below (Fig. 9), where only the wire top is heated. In these cases especially, but preferably in all cases, the cautery should be introduced cold, having previously ascertained the degree of current necessary to effect a dull red heat. The objects of the cauterization are not only to destroy the diseased cervical mucosa, which is the seat of irritative hyperfunction or hyperplasia, but also to destroy the subjacent hyperanemia and hypervascularity, which can be done effectively only by a dull penetrating heat. A white heat will not only just scar the surface, but will destroy the platinum points. With proper care these delicate nasal cautery points will last for years. One minute of careless white heat melts the platinum, and destroys the whole instrument. If ectropion has developed, this should be treated by radical tracings from the os outward to, and beyond, the margin of the ectropion. This again should be done in a dull red heat, by a fine knifelike platinum point. The knife should move slowly over the surface and should penetrate only about $\frac{1}{16}$ of an inch, but slowly enough to coagulate the supernumerary subjacent blood vessels. When larger, discrete vessels emerge from, and course over, the area toward the periphery, these should be destroyed with a finer cautery point, at their point of egress. Similarly nabothian cysts should be punctured and the contents caused to boil with prolonged contact. Sterile paraffin is then applied to the cervix and vagina. Douches are not begun until twenty-four hours after cautery. Their efficacy is questionable, but they are generally used. The pressure should be minimal, and lactic acid, 2 drachms to the quart, is very effective.

Cauterization of the cervix should be done as soon as convenient after cessation of a menstrual period. By choosing this time there will be less bleeding, better healing, and less disturbance of the succeeding menstrual phases. If treatment is instituted after the middle of the intermenstrual period, the subsequent menstruation may be greatly advanced, together with an increase in duration and quantity. In cases where there is much edema of the cervical lips and an unhealthy pale appearance of the area of ectropion from uterine allergy, cardiac disease, or other cause, the efficacy of cautery may be greatly reduced.

Complete restoration of normal function should be effected, together with pearly whiteness of the ectropic area, in about thirty days. Any disease of the appendages, chronic or otherwise, is a contraindication to cautery. Vaginal and vulvar infections should receive appropriate treatment before applying cautery to the cervix.

Cautery of the cervix is contraindicated in gonorrheal disease, except where it is confined to the cervix, a rare condition and a very difficult one to determine. However, I have seen two cases of gonorrheal septicemia, and one of gonorrheal inflammation of the interosseous membrane of the forearm, cured by cautery of the cervix, where there had never been any peritoneal symptoms or palpable disease of the adnexa. Acute and subacute cervical infections may act as a tonsil, constantly infecting the pelvis with successive bouts of peritonitis and involvement of the appendages. Articular disease may not infrequently owe its origin to an infected cervix. There is a certain risk in cauterizing such cases, but it may be undertaken with caution when the metastatic state is grave, and seems to warrant it, and where the cause and effect have been established beyond doubt. Pelvic abscess after cautery, where an unrecognized appendage disease or a virulent infection is present in the vagina, is not common, but always a possibility. The writer once saw a virulent streptococcic septicemia with a rashlike scarlet fever follow upon a mild cauterization.

2. *Chronic Cervicitis.*—We wish to emphasize not only the inadequacy, but also the harmful results, that may follow deep cautery of the cervical canal with a large cautery or cautery knife, extending from ¼ to ⅓ inch in depth, in the hope of reducing a large hard cystic cervix. In the first place, pathology shows the futility of this procedure, and in the second place, it so cicatrizes the cervix that dilatation at subsequent labors may be greatly inhibited or impossible. Cautery of this heroic type tends to produce cervical cicatricial transverse bands with irregular pockets of secretion, tending to aggravate the progress of the original disease. Cicatricial disease of the internal os, where the mucosa is normally very thin, may lead to hematometra and pyometra. There is no cure for such a large fibrous cervix. Its bulk will greatly reduce after menopause, natural or artificial. Practically all types of cervical inflammatory disease rapidly improve after the menopause, with the cessation of the congestion and flow of menstruation.

3. *Surgical Treatment.*—Surgical treatment for the cure of inflammatory disease is an ostrichlike subterfuge. It removes the disease from the scanning eye, but it bottles it up in the canal. It is almost impossible to remove all the diseased mucosa by the deepest coning out during amputation. Drainage is frequently blocked and healing is frequently only by secondary intention. Amputation of the cervix,

for the cure of endocervical disease, is as dead as the dodo in the larger clinics, but is still all too frequent in the smaller centers and hospitals.

But the worst features are the two complications which so frequently follow upon the cervical operations in the presence of endocervicitis. These are (1) late septic hemorrhage and (2) lack of primary union. How frequently one sees women enter the hospital in comparatively good health for a very "simple" operation upon the cervix! On the eighth to the twelfth day, and sometimes later, septic, uncontrollable hemorrhage follows which resists packing and secondary suturing, and yields only to two or more transfusions. Before its arrest the patient is reduced to a very critical state and anxiety is written plainly on the surgeon. And following upon this or without its antecedent, septic hemorrhage, the whole wound separates and heals by granulation, frequently complicated by a thrombophlebitis. For years I have lectured against cervical amputations and cervical repairs in the presence of chronic mucosal disease. If a repair is deemed necessary, owing to tears (undue importance is attached to these) and there is an associated endocervicitis, this latter ought to be cured first by appropriate methods, and, if cure by cautery is impossible, owing to some cause described in the foregoing paragraphs, then repair should be superseded by something less dangerous and more efficacious.

THE INFLUENCE OF A RESIDUAL INFLAMMATORY CERVICAL DISEASE UPON THE RESULTS OF OTHER PELVIC OPERATIONS

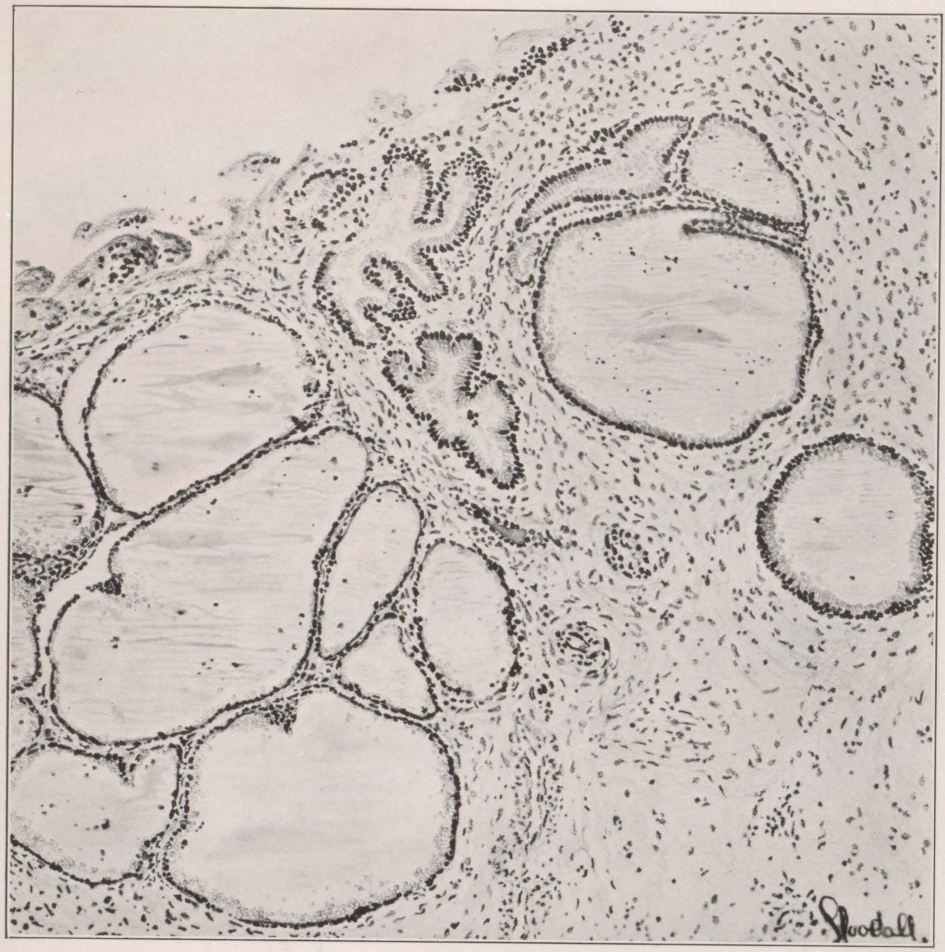
The residual cervix in these cases may act in the same manner as would a subacute or chronic gonorrheal case, only with a stronger tendency to produce postoperative septic hemorrhage and thrombophlebitic and lymphatic invasions. We are just beginning to displace the teaching that gonorrhea is a disease sui generis. It has much in common with the more common and usually more penetrating lesions of the streptococcus, especially when the latter has reduced its virulence to the level of the average gonococcal strain.

To show the trend of thought in the past few years, one has but to call to mind the general adverse reaction to leaving a uterus or cervix when dealing with gonorrheal disease of the genital organs. True, operations are infrequent in such conditions. But even today such operations have to be undertaken occasionally, for pain and economic reasons. Experience has taught us that a clean sweep is to be advocated, that is, the removal of all columnar surfaces. Why? Because the residual mucosa may be a constant infected tonsil to the pelvic system. We know today, that many low-grade ascending infections, indistinguishable from gonorrheal disease, are of streptococcic origin, whether they are postpartum or not. Thrombophlebitis is a much more

common complication of pelvic operations when the cervix remains than when it has been wholly removed, because, in the vast majority of instances, thrombophlebitis is a disease of a subacute nature arising out of a chronic or subacute mucosal disease. A little study of results will confirm this. This angle of the problem is fully dealt with in the authors' recent work *Total Versus Subtotal Hysterectomy.** Chronic inflammatory disease of the cervix usually improves after removal of both ovaries, but in conservative operations upon these appendages, followed by a subtotal hysterectomy, the diseased cervix frequently postoperatively develops a vitiated function, to the great disturbance and annoyance of the patient. In the past three months I have had to remove the cervix in three cases after supravaginal hysterectomy, where an anterior or sequent disease of the cervix set up unpleasant intolerable symptoms. These are partly due to the nutritional changes initiated chiefly by subtotal hysterectomies. Destruction of the cervix at the first operation, or its removal by a more radical procedure, would have obviated the sequelae. It is most removed from our wishes to advocate difficult operations with which the operator is not familiar or in which there resides a large element of conservative fear. But other less dangerous means should be adopted to eliminate the cervical postoperative hazard. What procedure should be advocated? Always the one that is best suited to the combined welfare of both patient and surgeon. This must always remain an individual personal question. But let us not close our eyes and ignore the hazard. It must be met, squarely and fairly in the interests of both patient and doctor. The truth is summed up by the confidential statement of a surgeon to me: "It is *very* comforting to both patient and surgeon when the cervix has been rationally dealt with and *most* comforting when that menace has been successfully removed."

1472 Sherbrooke Street, West

*Am. J. Obst. & Gynec. 23: 628, 1936.



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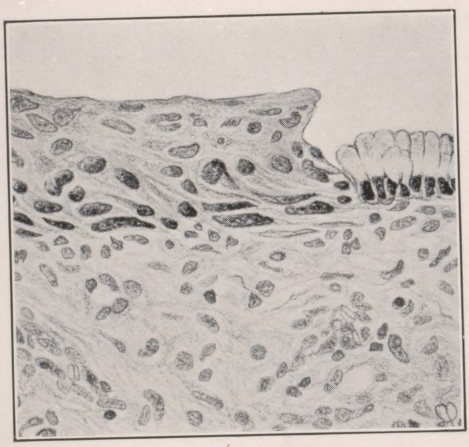

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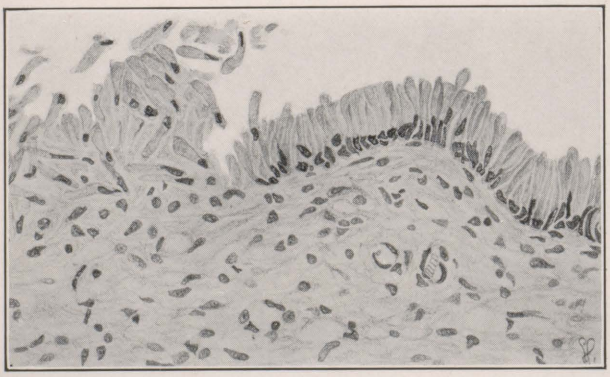

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MAX BRÖDEL, 1870–1941, DIRECTOR OF THE FIRST DEPARTMENT OF ART AS APPLIED TO MEDICINE IN THE WORLD

THOMAS S. CULLEN

Emeritus Professor of Gynecology in the Johns Hopkins University
Visiting Gynecologist to the Johns Hopkins Hospital

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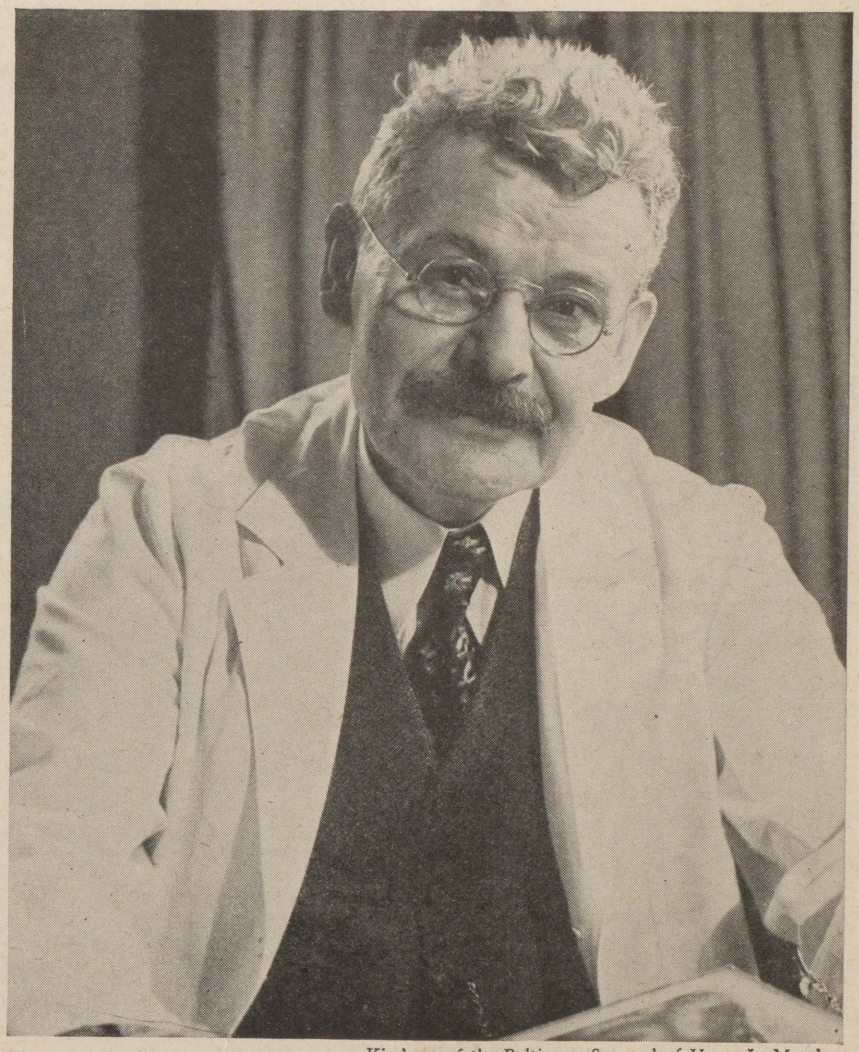

Kindness of the Baltimore *Sun* and of Henry L. Mencken

MAX BRÖDEL

Max Brödel, 1870–1941

Director of the First Department of Art as Applied to Medicine in the World

By THOMAS S. CULLEN, M.B.

AS THE YEARS roll by and this war-torn world finds itself again at peace we will gradually catch up with the things we have not attended to or have missed completely.

There was in Baltimore a kindly, curly-headed man of quiet demeanor, beloved of his friends and possessed of a passion for music. He was a born artist and during his forty-eight years in Baltimore revolutionized medical illustrating in the United States and Canada; his work has reached even the uttermost parts of the earth. No other man who has ever lived has done as much to improve the beauty and accuracy of medical illustration.

In the following pages I shall endeavor to give in some detail the picture of this man and of what he has accomplished. Less than two months before he died he gave a full account of his many years of stewardship. This was in a paper entitled "Medical Illustration," published in the *Journal of the American Medical Association,* August 30, 1941, Vol. 117, pp. 668-672. To Morris Fishbein go my warmest thanks, for I have used portions of the article wherever they could add to the proper portrayal of that Master Medical Illustrator. In after years, as he is now in many places, Max Brödel will be recognized as the greatest medical illustrator who has ever lived.

Max Brödel, Illustrator for Dr. Howard A. Kelly

Several years ago I asked Max Brödel to tell me about his early life and he jotted down the following which I have in his own handwriting:

Born in Leipzig, June 8, 1870. He was the son of Louis Brödel and Henrietta Frenzel Brödel. He went to the public schools until 1884. His father loved music and forced his son to study piano, beginning at the age of six. For several years he played nothing but scales, Czerny, and trivial sonatas. Finally he had sufficient technique to plunge into Beethoven. His teacher purposely kept him away from Beethoven until he was equal to playing his Sonata

Appassionata. Beethoven has had a powerful influence upon Brödel's entire life.

Brödel went to the Technical High School, 1884-85; then to the Leipzig Academy of Fine Arts, 1885-90. During the vacation he worked in the Anatomical Institute for His, Braune, Spalteholz, and later in the Physiological Institute for Carl Ludwig and his pupils. It was there that he met Dr. Franklin P. Mall in 1888.

Brödel was drafted into the army, November 8, 1890, to serve two years. Through the good offices of Geheimrat Carl Ludwig, Prince George of Saxony ordered that Brödel serve only one year with arms and that he devote the second year to artistic activity for the regiment.

In 1892 Brödel returned to Leipzig to free lance in fine art and anatomical and physiological illustrations.

Negotiations to come to America began in 1891 but because of misunderstandings, resulting from lost correspondence, Brödel did not arrive in Baltimore until January 18, 1894.

I can see Max as he arrived in Baltimore harbor in January, 1894. He was wearing a stiff black hat and the curly locks were welling out beneath the brim of his hat. He went to board with the Gills at 1640 E. Fayette Street where I had boarded during the Fall of 1891.

Max immediately started work with Dr. Howard A. Kelly in the Gynecological Department of the Johns Hopkins Hospital. One of the first drawings he made was of a case of prolapsus. Each operating day he would visit the operating-room and confer with the Chief; sometimes it was a specimen that Dr. Kelly wanted drawn, sometimes a new operation that was to be sketched. If the condition was not perfectly clear, Dr. Kelly, by a few judicious strokes, would clearly outline just what he wanted and Max would, at a glance, see just what Dr. Kelly desired drawn.

Max Brödel in his address at Dr. Kelly's 75th birthday dinner, on February 20, 1933, made this point very clear. I quote him verbatim:

> In the early years, Dr. Kelly's friend, Mr. Anthony Murray, made many excellent photographs, fine records of the dramatic aspect of surgery and of external lesions. But photography has its limitations and it is not hard to see where and why it fell short. I knew that it would be useless for me to compete with the camera in the realistic or imitative field. It was necessary to originate a different type of picture, one that would show far more than any photograph could ever do. To make such a picture is much more difficult. The artist must first fully comprehend the subject-matter from every standpoint: anatomical, topographical, histological, pathological, medical, and surgical. From this accumulated knowledge grows a mental picture, from which again crystallizes the plan of the future drawing. A clear and vivid mental picture always must precede the actual picture on paper. The planning of the picture, therefore, is the all important thing, not the execution.
>
> There is where we learned from Dr. Kelly. He had a way of making little

modest outline sketches when he explained his operative procedure to his illustrators. There were three of us now. Hermann Becker came in 1895, August Horn in 1898. Dr. Kelly had endless patience with us. He invented diagrams to show variations of form and relationship, motion, pressure, tension, rupture, the development of a pathological process, the sequence of operative steps, the placing of ligatures, sutures, etc.; in short, every clinical phenomenon, every operative procedure flowed in simple, eloquent lines from the end of his pencil. Few medical men can do that. What if the form was not quite correct; it did not matter, the spirit was there. We understood his diagrams; they were eloquent. In this way Dr. Kelly taught his artists the secret of the correct conception of an illustration, which is the very basis of all creative drawing. It usually was comparatively simple to build on, to give to the primitive contour correctness of form, to elaborate the plastic rendition and add surface texture. This is one great debt we owe to Dr. Kelly, and now for another: while making a drawing the conscientious artist has a way of discovering gaps in contemporary knowledge, so when knowledge was lacking and the literature silent on the subject, Dr. Kelly always permitted the artists to make original investigation to clear up the obscure point. That meant temporary cessation of illustrative output until the question could be answered. He never failed to give his consent to such digression. Few authors of medical books will do that. Without his sympathetic attitude we could not have learned our trade as we did.

It was not long before Dr. Kelly was publishing articles containing Brödel's illustrations, and these illustrations at once drew forth very favorable comments. When Dr. Kelly's two volumes on Operative Gynecology appeared in 1898 Dr. Kelly was at once recognized as the leader in American gynecology, and Brödel's illustrations in these two volumes immediately revolutionized medical illustrating.

Brödel made the majority of the gross pictures and Becker many of the histological pictures. Horn also made excellent gross and histological illustrations.

During all the years Dr. Kelly was most generous in agreeing to have Brödel make pictures for members of his staff, and every now and then, when a colleague in another department needed an illustration badly, Dr. Kelly gladly had Brödel do it for him.

Max Brödel was not only a wonderful draftsman: he had also a wide knowledge of anatomy and was a born investigator. For example, on one occasion Dr. Kelly wanted some anatomical data about the blood supply of the kidney. Brödel would go to the autopsies in the Pathological Laboratories, get a normal looking kidney, attach it by a tube to the tap, and wash out the kidney. He would then fill the arteries of the kidney with red, the veins with blue and the ureter with yellow. Next he would digest the kidney, using the digesting method he had seen Mall use in Ludwig's laboratory in Leipzig. The results he obtained were fascinating.

Various portions of the kidney reminded one of branches of an apple tree, and all over these branches were minute apples—they were the glomeruli or filters of the kidney. He pointed out the avascular area in the kidney and suggested opening the kidney along this line when exploring the kidney for stone, and before finishing this kidney investigation, he developed a suture which could be used to stitch up the kidney that had fallen down—one that was prolapsed. This suture is to this day referred to as Brödel's suture; it is triangular and so placed that a piece of kidney will tear out before the suture will give way.

In 1916 I published a 680 page book, *Embryology, Anatomy, and Diseases of the Umbilicus.* The best part of that volume is the embryology. Brödel had the opportunity of studying the most extensive and best collection of human embryos in the world, a collection started and continued for years by the late Franklin P. Mall. Without it such embryological studies and such drawings would have been impossible. This collection of embryos, after Dr. Mall's death, was greatly increased by Dr. George L. Streeter, who became Director of the Carnegie Laboratory, and when Dr. Streeter retired, by his successor, Dr. George W. Corner.

Let me give you one other striking example of Brödel's investigative spirit. I had collected an interesting group of cases, in which, for some reason, there had been a sudden rupture of a rectus muscle with hemorrhage. I asked Max to make me a drawing or two of the rectus muscle. That he had long been interested in this important muscle was unknown to me. In the course of a few months he had studied the rectus muscle most carefully and had also gone into the histology of it. The drawings he made were masterpieces. I told him we would have to publish the article jointly, as I could not for a moment lay claim to these wonderful drawings. Finally, he reluctantly consented and it was published under our joint names. He was modesty itself and most self-effacing.

In 1919 I published a short sketch of Dr. Howard A. Kelly in the *Johns Hopkins Hospital Bulletin.* Accompanying this article was a relatively complete list of Dr. Kelly's publications, prepared by Miss Minnie Blogg. The vast majority of these publications were illustrated by Max Brödel or by his associates, Hermann Becker and August Horn. To speak of these many publications would carry us too far afield.

Color reproductions in the text probably reached their high-water mark in *Cancer of the Uterus,* published in 1900, and in the *Umbilicus and its Diseases,* published in 1916.

MAX BRÖDEL'S ARTIST MODEL

Most artists have models. The majority of Max's drawings had to do with tumors or with operations; consequently he needed no regular

model. On a few occasions, when he was a relatively young man, and when he was illustrating Dr. Kelly's new method of examining the bladder and ureters, he required a model from which to sketch the knee-chest posture. Every time I see the drawings he made on those occasions I cannot help smiling broadly. I was Max's model, and you may rest assured that it was easier for me to assume the knee-chest posture at that time than it is now.

Max Brödel as My German Teacher and as My Companion

Almost immediately after Brödel came to Baltimore he and I became fast friends. At that time I had charge of the laboratory of Gynecological Pathology and would watch him making sketches of the tumors that had been removed. Later on I would go to his small studio and read. Just as soon as Brödel reached Baltimore he began to talk English and I began to attempt to answer him in German. Although I had had quite a number of lessons in German, my knowledge of the language was very fragmentary. As he spoke English and I German we very frequently had temporary misunderstandings, but as our knowledge of the two languages became greater we got along perfectly. In my spare time, while he was at work on a drawing, I would read a German book aloud. In the course of a few years I had read several novels by Gustav Freitag and two large volumes on the Franco-Prussian War. Of course Max would correct my mistakes in pronunciation as we went along.

Having Max as my teacher was of inestimable value to me. I soon could read medical German with relative ease, and on one occasion the reading knowledge of German relieved me of much embarrassment. At an international congress in Rome in 1902 I gave a lantern talk on adenomyoma of the uterus. The medical amphitheatre of the university was crowded. The speaker who preceded me came from the United States and few understood what he was saying. Prof. Paul Zweifel of Leipzig, whom I knew well and who was sitting in front of me, turned round and said, "Cullen, you must talk in German; there are not four persons in the audience who can understand English." I told him that I could not, but he said, "You must."

In a moment or two I was called upon and after explaining the matter to the presiding officer, who was an Italian, I started to talk in German and kept it up for forty minutes. I made many mistakes, of course, but my pictures on the screen helped mightily. My attempt to speak in German evidently pleased the Italians, as they made me an honorary member of the Italian society the next year.

Max and I stood by our original intentions. He was always to speak

English and I German. During the nearly forty-eight years of our friendship, I spoke in all less than two hours in English to him.

In the early days Max and I would in the afternoons take long walks out into the country and after traveling a couple of miles we would sit on a fence and read German. At other times we would take the car out to Walbrook and have a long walk along the millrace. Sometimes we would journey down to Cambridge on the steamer to visit Dr. Brice W. Goldsborough and his brother, Phillips Lee Goldsborough, later Governor. On other occasions we would run up to Waynesboro, Pennsylvania, to visit our friend, Dr. A. Barr Snively. On at least one of these occasions we returned to Baltimore on our bicycles.

In the fall of 1897, just after I had completed my term as Resident Gynecologist at the Johns Hopkins Hospital, Max and I went to Groveton, New Hampshire, and started on a hunting trip with a one-armed guide who was a delightful fellow. He lost his way and we slept out in the open. It froze during the night, and next morning there were fresh bear tracks within one hundred feet of where we had slept. We started off early that morning and had to cross a small river on a fallen tree that still retained its branches. To make it more difficult, we had to carry our satchels with us. Late in the day we reached the cabin of a deserted lumber camp. That night, while we were sitting by the fire, a lynx pressed its face against a window pane to see what was going on inside. Next morning we stepped out to the stream which was not over ten feet away. As there was a little ice on each bank of the stream we had to step in very carefully in order that we would not cut our feet. The brook trout darting up and down stream gave us a real thrill. Later in the morning Max and I were sitting quietly on a log in the forest hoping a deer would come along. We really were so quiet that a squirrel came up and sat down beside us on the log. We saw no deer.

Early in 1923 I had my second gall-bladder operation and took several months off. Max and I and our old friend, Alf Raaflaub, of Pembroke, Ontario, visited the homes of my childhood. I say "homes" because my father was a Wesleyan Methodist minister and according to the church rule we moved every three years. Max made sketches of some of the old homes, of one old swimming-hole where I had spent many happy days, of several of father's old churches and of many other interesting things. We had a glorious time and often referred to the fun on that trip. All this material was gathered together at the time, over twenty years ago. It is growing mellow and may possibly some day appear under the title, "From One to Twenty-One."

Max was an expert fisherman and if anyone could catch fish, he could. Well do I remember the day when he said, "Tom, I have some fine minnows; let us go fishing." After luncheon he and I started off for Burnt Island, which belongs to Miss Olga Kelly and lies about

a mile and a half north of the Kelly camp. After dropping anchor, Max put a wonderful minnow on my hook. Within five minutes my line started out. After it had gone about thirty feet I checked it, and after playing the fish for about five minutes, cautiously reeled it in. Max gaffed it and drew it into the boat. It was a wall-eyed pickerel weighing nine pounds—my best catch. Max had made his own gaff; it was an ordinary butcher's hook on which meat is hung. The handle of this hook had been embedded in a wooden handle about eighteen inches long. It was an ideal gaff. Max also repaired his own fishing tackle.

Frequently I went over to see my youngest sister and her family on an island about eight minutes away. On the way back I would invariably drop in to say hello to Max if he were fishing on his wharf. As soon as he saw me coming he would leave his rod and slip behind his boat-house to the shore; by the time I got there he invariably had a red and green bouquet to hand me, a fine bunch of young radishes. He had a new crop of these every week or two, and this was a regular ritual with him.

On one occasion, with Chancellor James H. Kirkland of Vanderbilt University and Abraham Flexner, the distinguished Director of the Institute for Advanced Study at Princeton, Max and I went to Horn Lake after lake trout. Kirkland and Flexner were trolling along slowly when suddenly my reel slipped off and dropped into ninety feet of water. Both Flexner and Kirkland were just like two small boys and teased me unmercifully. As I started to draw in, the wire gradually unrolled from the reel until all was unwound. The reel then came to the surface. To unreel and then reel up hundreds of feet of wire without kinking any of it was certainly a feat. It was in large measure due to Max's skillful handling of the canoe that he and I were successful.

We then had luncheon on a small island which had a very steep slope into the water, which at that point was over fifty feet deep. All of us were sure-footed. I built the fire, the Chancellor boiled the potatoes, Max made the coffee and Abe Flexner fried the bacon. We had a delicious luncheon.

I have given you this glimpse of Max Brödel to show you that he was no dreamer, that he was very capable in solving small difficulties as well as large ones, and that he was an ideal and most lovable campanion. He was absolutely fearless and most unconventional. I can still see him out in his small motor boat fishing at the point at dusk. The exhaust of a motor boat ever brings back to memory that genial, curly-headed friend of mine who, when getting up to speak at the dinner given me by nearly five hundred friends of mine on my seventieth birthday, stepped up and kissed me on my bald head before beginning his speech. Nobody ever had a better or truer friend.

Max Brödel's Illustrating Apart from Medicine

Most of Max Brödel's friends thought that he made only medical drawings, but such was not the case. During his summer vacation he would every now and then find time to make a drawing from nature.

I have in my library three of these pictures. The first is a beautiful painting of our log cabin, with the solid rock in front and the blue sky and white fleecy clouds, so characteristic of this vicinity, above and in the background. I can see Max anchored out on the lake making this picture. Next to that painting is one showing the interior of our log cabin with its fireplace and with the contents of the cabin depicted in careful detail, and in front of these pictures, illuminated by a study lamp, is a view of Ahmic Lake as seen from our sleeping cabin. It shows at the end of Rhodes Island the land of my youngest sister and her husband, Mr. and Mrs. R. A. Daly of Toronto. Across the lake from Rhodes Island rises a very tall tree on the mainland, on the property of my friend, Abraham Flexner, and down our side of the lake, about half a mile from our camp, is a boathouse, belonging to Max Brödel's family; their camp is hidden by the trees.

When I come in from a busy day's work and am a little fagged, these pictures act as a strong stimulant and carry me back to the glorious times we have had on and around the lake.

When we took long trips through our woods, Max would be on the lookout for fungi. These were often a foot or a foot and a half long and were attached to the sides of the trees. They were usually brown on top, pure white on their under sides. Brödel would take these fungi home, carefully protecting the white under sides of the fungi. On reaching home he would, with a pen, a pin or some other sharp instrument, etch a charming scene on each fungus. These pictures he usually presented to his friends. They were greatly admired and treasured; I have never seen their equal.

Max Brödel modernized the seal of the Medical and Chirurgical Faculty of Maryland, providing the seal now in use. He also made bookplates for the John M. T. Finney, the John Ruhräh and the William Osler collections of books of the Medical and Chirurgical Faculty. Among the limited number of bookplates made by him was one for Dr. Lewellys F. Barker. I am the happy possessor of another, the theme for which was arranged by Mrs. Cullen and Max.

Max Brödel made the famous cartoon, "The Welch Rabbits." "Popsy" Welch is standing up with a cigar in one hand and in the other are grasped the reins leading to the younger men who are pictured as rabbits. They were men whom Dr. Welch had trained or who had been associated with him. The cartoon was made at the time a very large

dinner was given to Dr. Welch. Many copies are in existence; the original was given to Dr. Welch's niece.

Back of me as I write in my library is the original of the now famous cartoon, "The St. John's Hopkins Hospital." Dr. Osler is adorned with halo and wings; his toes are sticking out of his small shoes. In the background is the Johns Hopkins Hospital, and in the whirlwind starting at the hospital and rising heavenward is Osler. Below him are many germs; some of them are scurrying away, others just don't care a damn and are sitting down resting on their haunches. Dr. Osler sometimes addressed letters to me and to others to "The St. John's Hopkins Hospital," and it was for this reason that Max drew the cartoon.

Before entering the operating room the surgeon and his assistants take off their street clothes and put on cotton shirts and trousers with drawstrings and without buttons. If the operator is thin there is usually no trouble, but occasionally, where the surgeon is stout and where the drawstrings are at the equator or slightly below, the trousers may slip. The nurses and students were well aware that on several occasions my trousers had slipped, but that really made no difference because the operator wears a sterile gown which extends to his knees.

In January, 1923, at an operative clinic, a student handed my head nurse a long, slim package addressed to me. I at once opened it. On the cover of the box was the picture of a very pretty girl. The box contained a fine pair of suspenders with a note to me expressing the best wishes of the class and presenting these suspenders for emergencies. I thanked the students for the beautiful and valuable suspenders but reminded them that I had no buttons.

A few weeks later, on February 28, 1923, an operation revealed that I had commencing gangrene of the gall-bladder and that the gall-bladder contained many stones. Max Brödel was present at the operation and took the gall-stones to his studio, where he made a facsimile of my name with the date. He used the individual gall-stones in reproducing my name and date in color and then made a frame around the picture using the large and irregular stones for this purpose. So graphic was the picture that one of my special nurses, when she saw it, immediately put out her hand to pick off one of the gall-stones from the frame of the picture. She really thought that the large gall-stones had been glued on to the picture.

On a bitterly cold and damp morning, about six days after my operation, Max had my student group congregate on the hospital bridge near Ward D for an exhibit. First of all there was the pair of suspenders with the kind note from the students; then came the painting of my gall-stones with the following note beneath: "The reason why the

drawstrings were not drawn tight." All were greatly pleased with Max Brödel's unique and telling demonstration.

The Brödel Family

Ruth Huntington, a charming young lady from Sandusky, Ohio, had had an excellent training in anatomy and art at Smith College, where she majored in scientific subjects, especially in zoology and botany. She always loved drawing, and her laboratory books attracted the attention of her professors to such an extent that they requested her to make charts to be used in the classrooms and to illustrate some of their articles. She was recommended to Professor Bailey of Cornell to make drawings of rare plants in the very fine, endowed botanical gardens in Northampton; she also furnished fifty illustrations for his *Encyclopaedia of American Horticulture.*

In 1900 Professor Franklin P. Mall asked Miss Huntington to come to Baltimore. Here she illustrated some embryological reconstructions for Dr. Charles Bardeen. As soon as she arrived at Dr. Mall's laboratory, Max Brödel went over to pay his respects to the new artist. He showed her the drawing technic he had developed for his students and in a short time learned to his pleasure that Miss Huntington was also a musician. Max suggested that they play on the piano together occasionally.

A little later she began work on the appendix book which Dr. Howard A. Kelly and Dr. Elizabeth Hurdon were about to publish. Brödel supervised the drawings made by Miss Huntington and August Horn for this beautifully illustrated volume. Incidentally, Brödel and Miss Huntington jointly wrote the anatomical chapter for the appendix book, and in the preparation of this made frequent trips to the Surgeon General's Library to look up the literature on this subject.

Naturally, it was not long before Max thoroughly appreciated Miss Huntington's unusual ability both as an artist and as a musician. They often played on the piano together. One summer Dr. Kelly invited Ruth Huntington and Max to his summer camp on Ahmic Lake. On a memorable day Max and Ruth paddled over to Birch Island, about half a mile from the Kelly camp. They returned late that day looking very happy, and that night Dr. Kelly, around the camp fire, announced their engagement. They were married in 1902.

A few years later, the Brödels built their camp on Ahmic Lake, and in 1910 I built mine along the same shore about half a mile distant from theirs. During the years four children were born to the Brödels. Little Ruth died while still a small child. Elizabeth, Carl and Elsa survive.

Elizabeth followed in her father's and mother's footsteps and is a

most talented medical artist in New York. She illustrates chiefly for Professor H. J. Stander at the New York Hospital. Carl is a Ph.D. of the Johns Hopkins University and specializes in mining geology. Elsa also has a great deal of artistic ability, and her advice is frequently sought about the proper selection of attire. She is now Mrs. Burk Allerton.

In the early days Max and Ruth lived at 707 Carrollton Avenue, next door to the Matt Tinker family, of whom we were all very fond. Mr. and Mrs. Charles Dohme and their family also lived on Carrollton Avenue; they and Mr. Louis Dohme could not have been kinder to the Brödel family or to me. Mrs. Louise Pomplitz was invariably included in the happy group.

For many years the Brödels lived at 320 Suffolk Road, Guilford. It was there that Mrs. Cullen and I spent a very happy evening once almost every week for years, playing bridge. Max was invariably my partner. Who usually won is a secret, but one thing is certain, Max and I often lost. With Max's going, and with the major portion of the Brödel family in New York, I rarely have the heart to pass 320 Suffolk Road, the scene of so many happy occasions.

Max Brödel a Remarkable Pianist

Max Brödel, as already mentioned, began his musical career when only six years old, and for two years his teacher allowed him to play nothing but scales, Czerny, and trivial sonatinas. After Max had practiced on these for two years he was given Beethoven's "Sonata Appassionata." He has often told me that in the playing of this he was almost overcome by its beauty and by its stirring qualities. When he came to Hopkins he always had a piano; sometimes it was in a room across from the hospital, sometimes in the center front room on the third floor of the hospital.

On March 24, 1899, Max entered the Johns Hopkins Hospital on account of a serious hand and arm infection resulting from some anatomical dissections he had been making. His hand and arm were opened on numerous occasions. Finally the wound healed, but some nerves had been caught in scar tissure and this complication necessitated further operation.

Brödel made many illustrations of his left arm and hand, showing the areas of numbness that existed following the operations for the infection. Dr. Halsted urged him to publish his case, together with the most instructive drawings, but Max was too busy and never did so. I saw the illustrations a few weeks ago.

In December, 1904, shortly after Brödel's attack of typhoid, a street-

car on which he was riding gave a lurch and he was thrown to the street. The middle finger of his right hand was bent right back on the back of his hand. It looked as if he might lose this finger. Fortunately, Dr. Finney was able to save it.

Had it not been for the excellent treatment given him by Dr. Halsted in 1899 and by Dr. Finney in 1904, Max's friends would have missed the many delightful and happy musical evenings he gave us in after years.

Brödel and Henry L. Mencken were the principals in the celebrated Saturday Night Club. Max derived a great deal of pleasure from the Club and attended it religiously. Every now and then I would invite him to spend the week-end down in the country. Time and again he would reply, "I wish I could, but Henry Mencken will be away on Saturday night and I must be there."

Up on Ahmic Lake was a jolly company consisting of some of the Kellys, the R. A. Dalys, the Abraham Flexners, the Chancellor Kirklands, the Benjamin Meritts, and the Cullens. Some or all of us would on frequent Sunday evenings congregate at the Brödels. Some sat in the numerous chairs, others, by preference, sat on the floor, all in camp attire, and listened to delightful music by Max and Ruth Brödel. These were evenings that we shall never forget; our debt of gratitude to the Brödels can never be repaid.

It is not generally known, but as a boy Max made such progress with his music that the musical authorities in Leipzig urged him to give up medical illustrating and to devote his life to music. Max Brödel was a remarkable pianist.

My knowledge of music is very limited, but on one occasion I accompanied Max to the piano. He and I were visiting our friend, Dr. David Houston of Troy. The night was very warm; Mrs. Houston was away, and Dr. Houston was out all night on an obstetrical case. When he returned in the morning he found our beds empty, but our clothes still in our room. After hunting the house over he finally looked in the parlor, which had been closed for weeks; the room was cool, and Max and I were sound asleep under the piano.

A Unique Tribute to Brödel

Max Brödel was made an honorary member of the Medical and Chirurgical Faculty of Maryland in 1909. He was the only layman ever accorded this distinction.

The Establishment of the Department of Art as Applied to Medicine in the Johns Hopkins Medical School

In 1910 Max Brödel, who had illustrated so many of Dr. Howard A. Kelly's papers and books, and who had made so many pictures for my publications, received an urgent invitation to join the staff of one of the finest private clinics in America. Dr. Kelly was loath to let him go but at the time had no large publications under way. Brödel said, "I was tempted to go elsewhere, but my roots were deep in the ground and I was loath to leave Johns Hopkins."

I was worried sick at the thought of my friend's departure. One day at camp I walked down to my tent, pulled out an old corncob pipe, filled it and struck a light and dreamed. I dreamed of a department of art as applied to medicine in the Johns Hopkins Medical School. Here artists who wanted to make medical art their life work could get a training of two or three years. Medical students could be taught how to make charts and blackboard illustrations, and the spare time of the head of the department would be taken up in illustrating articles published by members of the Faculty. In ten years the leading medical schools would have competent medical artists, and in twenty years American medical illustrations would be the best in the world, adding much to the prestige of American medicine.

After many heartaches, just four days before Brödel had to give his answer I was successful in enlisting the sympathy of a hard-headed business man who had a deep interest in art. This man, Mr. Henry Walters, promised $5000 a year for three years, and on receipt of his letter, I invited President Remsen and the Trustees of the University to luncheon at the Maryland Club at forty minutes' notice, as prompt action was necessary. The letter was read and the Trustees at once accepted the generous offer. Thus the Department of Art as Applied to Medicine came into being.

Attempts were made to secure an endowment, and the correspondence shows that the W. B. Saunders Company, medical publishers of Philadelphia, agreed to give $1000 toward the endowment. Mr. J. P. Morgan, through Dr. James Markoe, promised $5000 should the full $125,000 be raised. Dr. Markoe, an outstanding New York physician, was most helpful. A short time later, when he was taking up the Sunday morning collection in one of the leading New York churches, a crazy man, who had never known him, pulled out a pistol and killed him.

Mr. Walters offered to give $15,000 should the proposed $125,000 endowment be raised. The attempts to secure the necessary endowment

were, however, unsuccessful. Mr. Walters paid the $5,000 a year for three years, and finally told his Baltimore office to pay the Johns Hopkins University $2,500 every six months until he told them to stop. After continuing his benefaction for ten years Mr. Walters agreed to endow the department to the extent of $110,000. Thus Mr. Walters contributed in all $160,000 to the only Department of Art as Applied to Medicine in the world.

I wish the Brödels' friends could have seen the happiness on Max's and Ruth's faces when it was my rare privilege and pleasure to tell them that the Department of Art as Applied to Medicine in the Johns Hopkins Medical School had been endowed. Their faces shone with happiness, but a slight moisture was visible in the eyes of both of them. Up to that time the permanence of Max's Department had ever been in doubt.

Thus was started the first Department of Art as Applied to Medicine in this or in any other country. Max Brödel and his pupils have had a profound influence on medical illustrating throughout the entire world.

On March 1 of each year, Mr. Walters had on his desk in New York a complete report of what had been accomplished in the Art Department during the preceding year: the number of students, the parts of the country from which they came, the character of the illustrations, and frequently reproductions or photographs of the most important illustrations. He received a full report each year up to the time of his death. His interest in the Department grew greater and greater, and shortly before his death he wrote me as follows:

The Breakers, Palm Beach, Fla.
March 3, 1930

Dear Dr. Cullen:

I thank you for your kind letter of February 27 regarding the increasing influence of the work done under Max Brödel's supervision, and advising me that you have sent the report of the Johns Hopkins Medical School for the year ending February 28, 1930, which has been received at my office in New York.

It is a great pleasure to me to know that you led me into aiding you in establishing the Department of Art as Applied to Medicine, which has developed into so much real service to medicine and surgery.

(Signed) H. Walters.

At the time of Max Brödel's dinner, in 1938, Mrs. Walters wrote me the following letter:

Five East Sixty-First Street, New York, N.Y.
February 8, 1938

Dear Dr. Cullen:

Thank you for your kind letter of February 2. I appreciate greatly your thinking of me.

I do not think there was anything that Mr. Walters ever did, which gave him more satisfaction than the work, "Art as Applied to Medicine."

Very sincerely,

(Signed) Sarah W. Walters

The correspondence between Henry Walters and me relative to the Department of Art as Applied to Medicine in the Johns Hopkins Medical School extended over a good many years. It is intact and at the request of Mr. Morgan Marshall, Director of the Walters Art Gallery, has been deposited with the incunabula in a specially fire-proofed room in the Walters Gallery. Some one will, in due time, write a biography of Mr. Walters. His correspondence with me reveals a delightful side of him, a side that few knew anything about.

Presentation of a Portrait of Max Brödel by W. B. Saunders and Company, Medical Publishers of Philadelphia, to the Johns Hopkins University

In the Spring of 1937 Mr. R. W. Greene, vice-president of the W. B. Saunders Company, medical publishers of Philadelphia who had published many of the books brought out by Dr. Howard A. Kelly's department, dropped in to see me; he said that their company would celebrate its fiftieth anniversary on March 4, 1938 and that they wanted on that occasion to honor Max Brödel who had done so much for medical illustrating. He asked my advice as to the best plan to pursue.

In June I went down to Atlantic City to attend a meeting of the Board of Trustees of the American Medical Association a couple of days before the annual session. Mr. Greene took dinner with me on the night of my arrival. We finally came to the conclusion that probably the most appropriate thing would be for the Saunders Company to have Brödel's portrait painted, give a dinner to Max in Philadelphia, and present the portrait to the Johns Hopkins University where Max Brödel had done such wonderful work. The company at once decided on this plan of procedure.

The only difficulty was in persuading Max to fall in with these plans. I well knew his retiring nature and fully appreciated that the task ahead of me was not easy. On June 28, 1937, I wrote Henry Mencken telling him that just as soon as I reached camp I would take Max out in a boat where he could not possibly get away and then tell him what was in store for him.

On September 10, 1937, I again wrote Henry Mencken saying, "I wish you had seen Max the evening of the day he arrived at camp. He and Ruth took dinner with Mrs. Cullen and me, and after dinner I unfolded what is to take place on the 4th of March. His eyes bulged, he shook his head, he could not do it. As the evening progressed and as he

smoked a very delicious imported cigar he became more docile and in due time expressed appreciation for the honor in store for him. We shall have no trouble."

Mr. Thomas Corner, one of the country's outstanding artists, although very busy at the time, agreed to paint the portrait. He gave us a striking picture of Max.

The late afternoon of March 4, 1938 arrived and a full carload of Brödel's friends, lay and medical, left Mt. Royal Station, Baltimore, for the Barclay Hotel in Philadelphia. There they were joined by the leaders in medicine and surgery from all over the United States. It was a joyous company, as the great majority of those present were old friends of the distinguished guest of the evening. The members of the W. B. Saunders Company asked that they be allowed to stay in the background. Mr. R. W. Greene introduced me as the presiding officer and toastmaster. Max's and my old and beloved Chief, Dr. Kelly, slipped up to me and said, "Tom, may I say grace?" Of course he could; he did, and the dinner got under way.

After the dinner Dr. Howard A. Kelly spoke of the remarkable work Max had done at the Johns Hopkins Hospital and later in the Medical School. Morris Fishbein, the distinguished Editor of the *Journal of the American Medical Association,* told us of the profound influence Max Brödel had had on medical illustrating in periodicals and books. Henry L. Mencken described the celebrated Saturday Night Club which he and Max had started in 1910, just twenty-eight years before; this address, under the title "Max Brödel as a Pianist," appeared in "Tonics and Sedatives" of the *Journal of the American Medical Association* for March 26, 1938. Mr. Lawrence Saunders then presented the Corner picture of Max Brödel to the Johns Hopkins University. Because of the illness of President Isaiah Bowman, Dean Berry, an old friend of Max, accepted the portrait on behalf of the University, and we had finally a most appreciative and touching speech from our honored guest and beloved friend, Max Brödel.

We had had a delicious repast. The speeches were short and snappy. We returned to Baltimore the same evening. Everybody, including Max Brödel and Mr. Thomas Corner, the artist, had had a lovely time, and on the way home to Baltimore all were in a very happy frame of mind. On March 9, 1938, I had a short note from Morris Fishbein from Chicago. He said: "The dinner was marvelous and I shall always remember it as one of the big events of my life." The W. B. Saunders Company could not have done a finer thing for Max Brödel; as outstanding medical publishers they knew full well what this celebrated medical artist had done for the improvement of medical illustrating. A full report of the dinner is given in the *Journal of the American Medical Association* for

March 12, 1938, page 823. A reproduction of Thomas Corner's portrait of Max Brödel appears on the same page of the Journal.

"Medical Illustration," by Max Brödel

A number of years ago Morris Fishbein told me that he would like to have an article by Max Brödel giving a description of the Department of Art as Applied to Medicine. The paper, entitled "Medical Illustration," appeared in the *Journal of the American Medical Association* on August 30, 1941. We were fortunate, as Max left us on October 26, less than two months later.

In the paper Brödel gives us a panoramic view of how his department was run. It is of such interest and importance that I am going to give the highlights in his own words. In a few places there is a little duplication of what I have already written, but the repetition only serves to emphasize the various points. He says:

> I believe that the illustrator will learn to regard the photographer not as a rival to be feared but as a helpful friend.
>
> A medical picture may even be entirely synthetic and yet be drawn with convincing realism. To make such a picture the artist must know his subject so thoroughly that he can shut his eyes and coax into existence a mental picture of great clarity, complete in every respect. He also must be fully equipped to put this imaginary picture on paper, swiftly, accurately and, if necessary, with convincing realism. This is medical illustrating at its best.
>
> The technic is a matter of choice—half-tone, water-color, oil, lead pencil, simple or elaborate pen and ink, or a combination of these. It should be remembered, however, that technic, artistic feeling, accurate draftsmanship, neatness and speed are all relatively unimportant. The planning of the picture and the registration of the scientific facts are what give it its value, not the execution.
>
> As a rule a simple outline drawing is harder to make than an elaborate plastic picture. It is perhaps the most eloquent and useful type of medical illustration.
>
> Much information, explanation and analysis can be crowded into a diagram. It may be stripped entirely of all form and structure, relying for its message solely on well chosen key words, figures and numbers, connected by lines and rendered more expressive by the addition of symbols, such as loops, rings, arrows and the like. Even the time element can be graphically shown, also cause and effect, sequence of stages in a disease or an operation. Most instructive pictures can be made that way.
>
> For more than fifty years it has been my privilege to make medical illustrations in all the branches of the field.
>
> Since March 1911 I have also taught medical illustrating at Johns Hopkins University School of Medicine. Horn had died, Becker's health failed and I was left alone. So it became my privilege to organize and develop this school from 1911 until 1940, a period of thirty years, very happy years to me.

The experience gained during this long period may be of interest to those wishing to become medical illustrators. I shall try to show that the making of a medical picture is an intricate process, requiring much specialized knowledge and skill. It takes years of preparation to become an artist good enough to serve the medical profession.

How I Became a Medical Illustrator

The Art Department of the Johns Hopkins Medical School was the first of its kind in existence. I did not plan it. It came into being through a series of fortunate circumstances which I shall briefly describe.

1. Fifty years ago most medical pictures in textbooks and journals were done by untrained, self-taught artists who knew very little about medicine and less about art. There were no others available, and as a consequence the pictures in the medical literature were of poor quality, far beneath the illustrations in non-medical publications such as magazines and storybooks. The draftsmanship of medical pictures was amateurish, the object ineffectually posed and illuminated, sometimes inaccurate in regard to its anatomy and topography. The object evidently was not properly understood by the artist, and the author was unable to help. He probably knew that something was wrong with the picture, but his attempt to suggest corrections usually made it worse. It must be admitted that the atlases were notable exceptions. They were costly tomes, mostly from an earlier period, magnificently illustrated with elaborate copper and steel engravings, later on by lithography, often in gorgeous colors. Much of their beauty was due to the exquisite technic of the engraver or lithographer, not to the artist who made the originals. The same was true of the really fine wood engravings of the early textbooks.

The photomechanical method of reproduction ended this period and the cheap reproductions and poorly made drawings began.

This was the state of medical illustrating when I entered the service of Dr. Howard A. Kelly on January 18, 1894.

2. I had been fortunate in having received my art training in an academy where meticulous draftsmanship was insisted on and where the graphic arts were included in the curriculum. Both were of great help to me in medical art.

It was also lucky for me to be poor, for I had to seek work during the summer vacations and other free hours throughout the year. I came under the eye of Prof. Carl Ludwig, the great physiologist, and was permitted to illustrate his research and that of his famous pupils. In the course of this work I met Dr. F. P. Mall and Dr. William H. Welch.

3. Luck pursued me. Through Dr. Mall I came to know and work for Dr. H. A. Kelly, whose brilliant work in gynecology marked the beginning of a new era in that field. He chose me to make the pictures for his first large publication, "Operative Gynecology." That was in 1894. I worked hard but with little success. Photography was called in to aid in holding the elusive steps in an operation, to produce a clinical picture or show a pathologic specimen. I was urged to accept the help of the camera and obediently did so for a while but soon abandoned its aid, realizing that mere copying of a medical

object is really not medical illustrating at all, which, as every medical man knows, goes much deeper than that. Moreover, an artist feels degraded when he copies or uses a photograph as a basis for his drawing.

4. It was fortunate for me that Dr. Kelly was not only a kind and patient chief but also an excellent teacher. He could see that my ignorance in medical matters was a handicap to me. I felt sure that I could draw what I understood but found it exceedingly hard to plan a picture so that any one, even a layman, could understand it. It was difficult for me to select the most suitable view, to determine what to show and how to show it, what to emphasize and what to subdue or leave out. This is where I hestitated and wasted time, as every novice does. It was lucky for me that Dr. Kelly had the remarkable gift of explaining with sketches. In a few simple but graphic lines he could show all the new ideas in connection with his operative work. There is no question that Dr. Kelly's genius for visualization and for sketching paved the way for his illustrators. He made it clear that the conception of a picture is the all important thing, not the plastic elaboration, the realism or the technical finish.

5. Another lucky factor was that Dr. Kelly let me study while working for him. Few employers would permit that. They want pictures for their money. I dissected and injected the pelvic and abdominal organs many times. No drawing was made by me without original study by injection, dissection, frozen section or reconstruction. When variations in adult forms puzzled the eye, the study of embryology gave the key. Many embryos and fetuses were injected, dissected, sectioned and studied. Had these studies been made for me by some one else, in order to save time, I would have benefited little; the finished dissection, injection or reconstruction would still in part have been an enigma to me. The eye and hand must work together to obtain the priceless information that automatically crystallizes into a mental image, which is the forerunner of the subsequent picture on paper. There is no other way, at least for the beginner.

If the artist has made sure of his ground, his drawing shows it. It is a truer, bolder, better picture and is done with greater speed.

6. Dr. Kelly also permitted me to help other illustrators while in his employ. He even encouraged this digression. This paved the way for subsequent teaching of professional illustrators, of art students, medical students and members of the faculty doing research work. There is no better way to learn a subject thoroughly than by teaching it to others. My first pupils, of course, were my friends Becker (1895) and Horn (1898), who came to assist me in the work for Dr. Kelly and his staff. I tried to teach them the fundamental principles of gynecologic illustrating but had little to give in those days. The job was not of my making; it was wished on me. The truth is, we learned together. I was rarely more than a jump or two ahead of them. Confession is good for the soul.

7. Thousands of pictures were made by the three of us to illustrate the various books and articles written by Dr. Kelly and his associates. Each book marked an advance in our method of approach and technic.

It should be stated here that Dr. Kelly also permitted us to make illustrations for other departments—those of anatomy, embryology, physiology,

pathology, surgery with its many sub-divisions, and obstetrics. This outside work retarded our regular illustrating, but Dr. Kelly invariably presented the pictures to his colleagues. This unselfishness broadened my field and ultimately led to the creation of the Art Department in March 1911. When Dr. Kelly's work ceased, I was tempted to go elsewhere. But my roots were deep in the ground and I was loath to leave Johns Hopkins.

8. This is where my friend Dr. Thomas S. Cullen came in. He had other plans. He had faith in our work, ideals and technic and wanted to keep them at Johns Hopkins. His dream was to create an art department in which the methods and technic which we had evolved during the wonderful Kelly period could be handed down to new generations of medical illustrators and spare them the years of trial and disappointment of their self-taught predecessors. . . .

I have described elsewhere in this paper how the Department of Art as Applied to Medicine came into being. Let me quote Max Brödel further:

The Art Department, 1911-1940

During the thirty years of its existence the art department has trained nearly two hundred medical illustrators, carefully chosen from thousands of applicants. There are no two alike. Each has his or her own individual style. I can nearly always tell who made the picture without seeing the signature.

Those who have taken the course are employed in the important medical schools and clinics of the United States and Canada. A few of them are abroad. Their work can be found in the medical literature, exhibits, hospital records, lantern slides and the like.

The size of the class varied from four to twenty-three regular students a year; the average was ten. Every year a few professional illustrators were admitted as post-graduate students for intensive study in a special branch, mostly technic. Members of the faculty and a limited number of medical students received instructions in drawing—the former as an aid in their research, the latter because of their interest in and talent for drawing and its obvious benefit in the study of medicine.

The instructions of the regular art students, the beginners, were as follows:

Since no illustration can be made without anatomic and histologic knowledge, the student begins in the dissecting room, doing the work with his (or her) own hands, slowly and thoroughly. Because each student can have only a few bodies to study from, while the range of variations is legion, daily lectures and demonstrations accompany and augment the dissection. A large transparent ground-glass plate covers life sized drawings of a skeleton, front, back and side views; also sections, properly co-ordinated. With charcoal and colored chalk these pictures can be altered before the student's eye to show variations in size, form and proportion due to sex, age or race; also physiologic, pathologic and postural changes, diseases and their course and operations; in brief, everything that concerns the illustrator. Each alteration requires

only a few strokes, which are erased when the alteration is demonstrated and another type drawn—and so on.

While the student dissects and studies the skin, the ground-glass plate explains its phenomena in a multitude of variations. Then are taken up the bony landmarks, fat deposits, vessels, nerves, lymphatics and their variations and significance; muscles, tendons, ligaments and fasciae. Then come the internal organs first in toto, then each organ separately with its variations and pathologic alterations.

Every structure and every organ is studied at the same time microscopically under low, medium and high power to give a complete picture.

The most important part of the student's work in the dissecting room, however, is sketching and drawing. Everything revealed by the student's eye and hand is put on paper immediately, while the impression is fresh. The sketches are made boldly and rapidly in black and colored crayons. Thousands of such sketches are made. They are the test that the student has grasped the subject.

They are valuable to him as forerunners of medical and surgical pictures. He has learned to study the subject in the form of pictures, not in words.

At the end of the year a recapitulation of the topography of the viscera is made by the study of frozen sections, sagittal and transverse.

Fresh material is employed, whenever necessary, to augment the studies on the cadaver and to correct post-mortem phenomena, alterations of form, consistency and color.

Many accessory methods are used to teach the student the correct approach to a problem in illustrating. It is always done in the form of pictures, either on paper, or on the ground-glass plate or on the blackboard; occasionally by graphic description, made alive by expressive gestures by the hands. Nearly every discussion terminates with a diagram; a question asked by an alert pupil may lead to the creation of a helpful topographic sketch sometimes startling in its originality. It is a stimulating habit to show the beginning, course and end result of a disease in an eloquent diagram. The medical literature can use more such pictures.

The student must also learn to make accurate, realistic pictures of objects placed before him. He must know how to pose and illuminate them so that their plastic rendition is simplified.

Much time in the class is given to this part of the training. No artist can create a picture unless he has first learned to imitate nature. Many pictures are made of fresh or hardened specimens from an operation or an autopsy, and most important of all, realistic pictures of operative steps.

Paralleling these studies are complete instructions in accurate draftsmanship and perfect technic. All important technics must be mastered in order to make the original drawing or painting acceptable to the author, to the engraver and to the publisher. Several new technics were worked out in this department and are taught to the students. The pen and ink technic is by far the most useful, because a line drawing costs less to reproduce and is not easily ruined by inferior press work.

Being thus equipped, the student is ready independently to plan and make

finished pictures for publication. After many failures a moderate degree of success may be attained; occasionally there is a gem, even a little masterpiece.

The superior work of a talented student is quickly noticed by instructor and fellow-students, then by members of the medical and surgical staff and then by visiting physicians. The student is asked to make drawings for them, which may start him on the way to getting a job.

Applicants

It is generally accepted that medical illustrating is an intricate, highly specialized form of art requiring for its mastery systematic full time study over a period of from two to four years or even longer, according to the talent, speed and preliminary training of the applicants.

They come from colleges, art schools and high schools. I have found that good material may come from any of these.

1. Some applicants are born artists with exquisite technic and a poetic temperament but entirely ignorant of the basic branches of medical science and for some reason quite unteachable.

2. Others have a college background with a fine grasp of the medical aspects of their studies but are awkward in drawing; their eyes and hands do not work together.

3. A rare group of applicants is gifted in science as well as in art.

4. The last group is without talent in either. The applicants of this group assert that they are fascinated by medicine and its dramatic service to humanity and want to help, but mere interest in the subject is not enough. However, there are even here notable exceptions.

The applicant may be a man or a woman. The best age is between twenty and twenty-four.

The qualifications in order of their importance are:

1. Keen interest in science, in nature and in all living things.

2. Ability to study intelligently, to observe accurately and to doubt the statements of authority.

3. Ability to draw and paint from nature free hand and with artistic charm.

4. Ability to visualize, to imagine a picture based on previous study and then give it reality on paper, either in contour alone or with convincing plasticity.

5. Technical skill in drawing, a trustworthy eye guiding an obedient hand, preferably the right.

6. Ability to stick to a task with tenacity and to be resourceful in the face of obstacles.

7. Good general health and normal vision.

Every medical artist worthy of the name realizes that he has to know the entire human body and the entire field of medical illustrating before he can discover the branch for which his special talent and his chief interest and inclination fit him best. A medical student does the same and for the same reason.

Moreover, in many institutions an illustrator has to serve several departments and be prepared to draw all types of pictures in every known technic.

An Evaluation of Max Brödel and His Amazing Work

Good illustrations are to a fine medical article what show-windows are to an outstanding department store. People walking along the street stop and look at the window display and then enter the store. So medical readers are attracted to an article by the fine pictures and then linger to scan the medical treatise.

When Mr. Henry Walters and I were considering the founding of the Department of Art as Applied to Medicine he wrote and said, "Tell me what you want." I promptly told him of what van Calcar, the famous artist, had done for medicine when he illustrated Vesalius' atlas on anatomy, and I pointed out what Brödel might do for the medicine of this generation.

I knew then of Max's wonderful qualifications but I had little idea that he would be the potent factor in the advancement of medicine in our generation that he later became.

The last seventy-five years have shown marvelous strides in inventions and in making things easier for mankind. We have the telephone, the radio, and recently the instruments helping those who are hard of hearing. Our trains are infinitely more speedy and more comfortable. Automobiles enable one to travel to all parts of the country quickly, and the airplane can take us to Europe in less than a day. I could mention many other important improvements but you know what they are as well as I do.

Let us see what has happened in medicine and surgery. We in the medical profession have, I am proud to say, kept pace with the steady advance in the other branches of human endeavor. Typhoid has, in large measure, disappeared. The deaths from pulmonary tuberculosis have been greatly reduced. Lockjaw when taken early has lost its horror; diphtheria, which caused so many tragedies in years gone by and which would frequently take two or three children out of a family within a week, can now be avoided if proper inoculation is carried out. The threat of smallpox can be eliminated where people have the good sense to be vaccinated, and most of them have.

Let us turn for a moment to what surgery has done in this period. Many tumors are successfully removed from the brain. Tumors of the neck are frequently taken away. The chest was formerly a forbidden area except where pus was to be withdrawn; now the chest wall is collapsed in cases of tuberculosis. A part of or an entire lung can be removed for malignancy, and in some cases the heart has been sutured

so that not only has the life of the patient been saved but also that of the person responsible for the stabbing. You are all familiar with the operations on the stomach, the gallbladder, the intestines and the appendix. The waterways in men and women have been carefully studied; kidneys can be removed, tumors of the bladder taken out, and untold thousands of American men who formerly became slaves to the use of the catheter, with its frequent inflammation of the bladder, can now have their prostates removed when they are in good condition and can go on living comfortably for many years. Then we have the youngsters with crooked arms and legs who become useful citizens when these members are straightened.

The time was ripe for Max. He came just as medicine and surgery were making greater strides than they had done in centuries and when many new illustrations were necessary. Max came to us fifty years ago. It seems but yesterday and I cannot help thinking of the Biblical quotation, "For a thousand years in Thy sight are but as yesterday when it is past."

Let us consider Max Brödel's qualifications when they had become fully developed.

1. He was a wonderful artist.
2. He was a fine anatomist.
3. He was a remarkable investigator.
4. He was an excellent teacher.
5. He was a lovable character, so much so that it was always a pleasure to grant his requests.
6. He was one of the country's outstanding pianists. I mention this because music was balm of Gilead to his soul.

Let us consider for a moment Brödel's friends. Howard A. Kelly was for years his Chief. Dr. Kelly during his lifetime did more for the advancement of gynecology than any other man who has ever lived. Franklin P. Mall was Professor of Anatomy and had the largest collection of human embryos in the world. Max had made drawings for Mall in Carl Ludwig's laboratory in Leipzig, and it was through Mall that he came to Dr. Kelly. Naturally, Brödel was given every facility in teaching his students anatomy in Mall's dissecting-room, and when Brödel wanted to study any embryological problem he was welcome to use this material which had been sectioned and was in excellent condition for study. Dr. William H. Welch had known Brödel in Leipzig and if Max wanted to do anything in the autopsy room he was at once given the opportunity.

Max, when he came to Baltimore, had a rather meagre knowledge of the English language, but he soon remedied that. In due time he grew to know Henry Mencken, one of America's greatest English scholars—a

man who can handle the English language just as skillfully as one of the best surgeons can wield a scalpel. For no one did Max have a deeper respect or affection than for Henry L. Mencken.

With the outstanding qualifications possessed by Max Brödel, with every possible help from the leaders in surgery, anatomy, embryology, pathology and English at his disposal, and with the advances in medicine and surgery coming along so rapidly, what else could one expect from Brödel but a phenomenal career? He was never idle, and when he played he really played.

If one wishes to see just what Max Brödel and his students have done, let him go to any of the leading medical libraries, pick out the outstanding medical journals and books of from fifty to one hundred years ago and put them in one pile, and then pick out the leading medical publications during the last fifty years and compare the two. This procedure will show the startling advances made in medical illustrating during the last fifty years. Max was largely responsible for this epoch-making improvement. He established a fine technic and set a very high standard.

Max Brödel was born on June 8, 1870 and died on October 26, 1941. During his span of life he revolutionized medical illustrating and placed it on a very high plane. His pioneer work in medical illustrating has already been of inestimable value to medicine and surgery, and the appreciation of his remarkable contribution will grow greater and greater as the years go by.

Although he left us in 1941 he still lives in his many drawings, in his many students, and in a personality that will be remembered as long as any of his old friends live.

great that in America a school - the first of its kind - was started where artists would receive proper training, and that because of the high flight which modern medicine has taken the practical application of art in medicine is still in the beginning. It calls this a beautiful and interesting field for those to whom this work appeals, and notes that although only a handful of artists is busy along these lines, three years ago an international organization of medical illustrators was started.

H.L.

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BETHKE, EMIL G., Eye Institute, Col.Presbyterian Medical Center, New York 32, N.Y.

BOOTH, DOROTHY, Art Section, Mayo Clinic, Rochester, Minn. (Associate to Russell Drake)

BOSSELMAN, FRED, American College of Surgeons, 40 E. Erie St., Chicago 11, Ill.

BRÖDEL, ELIZABETH, M-712 Lying-In Hospital, New York 21, N.Y. (Staff artist)

BROWNING, BETTY JANE (Mrs.S.B.Andrus), 459 Beacon St. Boston 16, Mass. (Free-lance illustrator)

BURGESS, ANNETTE, Wilmer Institute, Johns Hopkins Medical School, Baltimore 5, Md. (Artist)

BURR-THOMAS, PAULINE (neé Larivière), 1105 Berwyn St.,Chicago 40,Ill. (Free-lance artist and illustrator)

CLARK, ELON H., Med.Illust.Div., Research and Education Service, Veterans Administration,Central Office, Washington 25,D.C.(Chief, Medical Art and Photographic Section)

CODDING, MILDRED, Peter Bent Brigham Hospital, Boston 15, Mass. (Artist and photographer, Dept. Surgery, Harvard Medical School and Peter Bent Brigham Hospital)

COLEMAN, RUTH, Illustration Studios, University of Illinois College of Medicine, Chicago 12, Ill. (Asst.Prof. Medical & Dental Illust.)

CONRATH, PHILIP A., St. Louis U. School of Medicine, St. Louis 4, Mo.

CUZZORT, ELIZABETH (Mrs.E. Singer), Hoagland Lab., Long Island College of Medicine, Brooklyn 2, N.Y. (Artist, Pathology Dept.)

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DIDUSCH, WILLIAM P., Studio, 910 E. Biddle St.,Baltimore 2, Md.

DIEDRICK, MELFORD D., Univ. Buffalo Medical School, Buffalo 3,N.Y. (Director Medical Illust.)

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DRAKE, RUSSELL L., Art Section, Mayo Clinic, Rochester, Minn. (Head of Art Section)

DRESKIN, JEANET S., Dept. Anatomy, Univ. of Chicago Med. School, Chicago 37, Ill.(Illustrator)

DUNLAP, JEAN THOMPSON, Med. Illustration Dept., Sanford Medical School, Sacramento & Webster Sts., San Francisco, Cal. (Illustrator)

ERICKSON, EVELYN J., Wayne University College of Medicine, Detroit 26, Mich. (Illustrator)

FEINBERG, ALFRED, Dept. Pathology,Coll. Phys. & Surgeons, Columbia U., New York 32,N.Y. (Instructor of Medical Illustration)

FOSTER, CYNTHIA E., The Army Institute of Pathology, Washington 25, D.C. (Illustrator)

FOSTER, DOROTHY (Mrs.S.Chubb), 732 Avenue Road, Toronto 12, Canada (Free-lance illustrator)

HEMBERGER, ARMIN, Bureau, Medical illustration, Yale U.School of Med., New Haven,Conn. (Artist)

HILL, EDNA, Harrison Surg. Research Dept.,University of Pennsylvania,Philadelphia 4,Pa.(Assoc.)

HIRSCH, JEAN E., Medical Art Shop, Univ. Minnesota, Minneapolis 14, Minn. (Director)

HISER, ERNEST F., Dept. Med. Illustration, University of Oklahoma School of Medicine, Oklahoma 4, Okla. (Asst. Prof. and Chairman)

HOLT, CHARLOTTE S., Room 292, 1853 W.Polk St., Chicago 12, Ill. (Staff illustrator, Illinois Dept.Public Health, Div.Public Education, & U. Illinois Coll.Med., Dept.Obst. & Gynec.)

HYDE, KAY, Med.Illustration Laboratory, U.S.V.A. Hospital, San Francisco 21, Cal. (Illustrator)

INGRAM, CHARLES F., W.C.Campbell Clinic, Memphis 3, Tenn. (Illustrator and photographer and registered X-ray technician)

INNES, LUCILLE CASSELL, 1218 Myra Ave., Los Angeles 27, Cal. (Free-lance illustrator)

JACOBS, NATT C., Anatomy Dept., U. Rochester School of Medicine and Dentistry, Rochester 7,N.Y. (Instructor, School of Med. Illustration)

JONES, THOMAS S., Illustration Studios, University of Illinois Coll.of Medicine, Chicago 12, Ill. (Prof. Medical and Dental Illustration)

KOHRING, MRS. WILLIAM J. (Marion Mason)..... Holland

KRAUSE, LISBETH, Dept. Zoology, O.Z.L., Yale University, New Haven 11, Conn. (Illustrator)

LINDEN, CARL T., Illustration Dept., U.Illinois College of Medicine, Chicago 12,Ill.(Instructor

LORRAINE, HELEN, Studio, 5212 Sylvan Road, Richmond 24, Va. (Free-lance illustrator)

LOUD, MRS. NEWTON G. (Helen Lewis), 40 Elm Street, Wellesley Hills 82, Mass.

MACIEL, MARY, School of Medical Illustration, University Cincinnati College of Medicine, Cincinnati 29, Ohio (Asst. Prof. & Director)

MACKAY, KATHLEEN, Department Medical Illustration, Emory University Hospital, Emory University Atlanta, Georgia. (Director)

MALONE, PAUL D., Lahey Clinic, Boston 15, Mass., (Director of Medical Art)

MC CONNELL, JEAN, Medical Art Dept., Northwestern U. Medical School, Chicago 11, Ill. (Artist)

MC HUGH, GLADYS, Dept. Obst. & Gynec., University of Chicago, Chicago 37, Ill. (Illustrator)

MC NETT, ELIZABETH VARDELL (Mrs.W.B.McNett), Lankenau Hospital, Corinthian & Girard Aves., Philadelphia, Pa. (Staff artist)

MC NETT, WILLIAM BROWN, Art Department, The Blakiston Co., Philadelphia 5, Pa. (Art editor)

MILES, MARY WICK, Youngstown Hospital Assn., Youngstown 4, Ohio. (Director of photography and illustration)

MILLER, MURIEL MC LATCHIE, School of Medical Illustration, Massachusetts General Hospital, Boston 14, Mass. (Director)

MOREL, VERA, Surgical Dept., Tulane U. School of Medicine, New Orleans 12, La. (Illustrator)

PADGET, DORCAS HAGER (Mrs. Paul Padget), Dept. of Art as Applied to Medicine, Johns Hopkins Medical School, Baltimore 5, Md. (Associate)

PARKES, ORVILLE A., Div.Med.Art & Illustration, Duke U. School of Med., Durham, N.C. (Director)

PAXSON, ELEANOR M.(Mrs. W.B.Keighton, Jr.) 311 Cedar Lane, Swarthmore, Penna. (Scientific Medical Artist, free-lance, and Senior Illust. with Fish and Wild Life, U.S.Dept.Agriculture, Bureau of Animal Industry)

PERKINS, EMILY FRERET (Mrs.G.R.Perkins), Pascock Road Spring Valley,N.Y. (Free-lance illustrator)

PHILLIPS, JESSIE W., Am.Coll.Surgeons,Chicago 11,Ill.

PHILLIPS, KENNETH G., Dept. Med. Illustration, Scott and White Hospital, Temple,Texas.(Director)

PHILLIPS, MARGARET WHITING (Mrs.K.G.Phillips), Dept. Medical Illustration, Scott and White Hospital,Temple,Texas (Artist)

REYNOLDS, ROSE M., Univ.Nebraska College of Medicine, Omaha 5, Neb. (Technical artist, Anatomy Dept. and Med. illustrator, Clinical Dept.)

ROSS, E.HOPPER, 124 Summit Drive,Toronto,Ontario,Can.

SANDERS, RUTH M. (Mrs.O.Sanders),Dept.Medical Art and Visual Education,Southwestern Medical College, Dallas 4, Texas (Asst. Professor)

SCHLOSSBERG, LEON, Art Dept., Johns Hopkins Medical School, Baltimore 5, Md.

SHACKELFORD, ELLA MAY (Mrs.F.C.Breckenridge), Dept. Visual Education, Baylor U. Coll. Medicine, Box 156, Houston, Texas. (Staff artist)

SHEPARD, WILLARD C., % W.B.Saunders Co.,W. Washington Square, Philadelphia 5, Pa. (Art editor)

SHULTZ, FRANCES, Johns Hopkins Medical School and 1909 McElderry St., Baltimore 5, Md. (Free-lance illustrator)

STEVIC, WANEETA (Mrs.F.A.Althoff), U.Colorado Medical Center, Denver 7, Colorado

STEWART, W.BRANKS, Dept. Medical Illus., Louisiana State U.School of Medicine, New Orleans 12, La. (Associate Professor)

SUMMERS, IVAN, Dept. Plastic Surgery, Presbyterian Hospital, New York 32, N.Y. (Artist)

SWEET, RALPH, Dept. Medical Illustration, University of California Medical School, San Francisco 22, Cal.

SWEEZEY, ELEANOR A.,Queen Mary Veterans Hospital,4565 Queen Mary Road, Montreal 26,Quebec,Canada. (Adviser in Med. Art and Director Dept. Art & Photography, Department Veterans Affairs)

VELICK, BERNADETTE S.(Mrs.S.F.Velick),6908 Millbrook Blvd.,St.Louis 5,Mo.(Free-lance illustrator

WATERS, LEWIS B.,Dept.Medical Art & Visual Education, Southwestern Medical College, Dallas 4, Texas (Prof. Art as Applied to Medicine)

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WILSON, JACK, University of Georgia School of Medicine, Augusta, Ga.,(Assoc. Prof. Art as Applied to Med. and Director of Illustration)

WINTER, DUNCAN KENNETH, Med.Illus.Dept.,Veterans Adm. Hospital, Hines, Illinois (Illustrator)

WISHART, MARIA T., Dept. Art as Applied to Medicine, University of Toronto, Ontario 5, Canada. (Assistant Professor and Director)

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