International clinic week at the New York Polyclinic Medical School and Hospital during the International Surgical Congress, April, 1914 / by Alfred C. Jordan [and others].

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International Clinic Week

AT THE

New York Polyclinic Medical
School and Hospital

DURING THE

International Surgical Congress
April, 1914

Three in one, viz:—
Jordan — 12
Herlogha — 13
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New York

International Clinic Week

AT THE

New York Polyclinic Medical School and Hospital

DURING THE

International Surgical Congress April, 1914

BY

ALFRED C. JORDAN, M.D.
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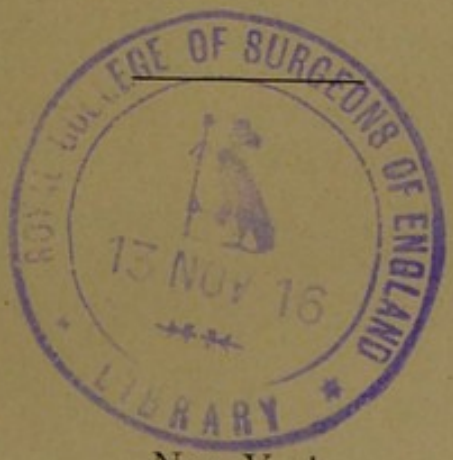

New York

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INTRODUCTORY NOTE.

In an editorial entitled, "A Notable Surgical Clinic," Dr. Thomas L. Stedman, editor of the *Medical Record,* said in part: "In the issues of the *Medical Record* for September 19 and 26 a large amount of space available for original communications has been given to papers read and reports of clinics held at the New York Polyclinic during the week of the International Surgical Congress which met in this city last spring. In honor of this Congress the Faculty of the Polyclinic requested the surgical staff of the institution to open its doors to the visiting members of the profession, and to arrange a series of lectures and clinics. Accordingly, on four days of that week the surgical lectures and demonstrations were given which have here been reproduced for the benefit of our readers.

"This was the first occasion on which the International Surgical Congress had met in this country—in fact the first time it had been held elsewhere than in Brussels, the city of its birth. One can but wonder where the next Congress will meet and when—when indeed the enmities and hatreds engendered by this European strife will have become so far dulled that any really international meeting will be possible, even of medical men. These are wont to boast in their gatherings, in the intervals of war, that science is truly international, untinged by jealousies of race and nation, but when war comes they find that after all blood is thicker than water and love of country is more compelling than love of science."

When the Surgical Staff of the New York Polyclinic Medical School and Hospital arranged for the "International Clinic Week," in honor of the members and friends of the International Surgical Congress, they had no thought that perhaps the Empire City of the Western World might witness the unforeseen ringing down of the curtain for a time upon the corporate life of this body of distinguished surgeons who, in-

dividually and collectively, have accomplished such splendid work for science and for humanity.

This, however, may be the case,—not, perhaps, because "the love of country is more compelling than the love of science," but because of the years of reconstruction, of privation, and of general disheartenment that must of necessity follow the unfortunate struggle which now engulfs the major part of the world, and which, in truth, affects deeply the whole world.

For all these reasons we are glad to respond to repeated requests for the perpetuation, in convenient form, for those who attended the International Surgical Congress and the International Clinics held at the Polyclinic, the lectures and clinics held on that occasion.

We venture, however, to express the hope and the belief that when the present conflict of nations shall have ended the medical world will find itself once more united in the earnest effort to alleviate human suffering and to render more and more impossible, through the development of a higher brotherhood, a repetition of the tragedies now being enacted.

CHRONIC INTESTINAL STASIS.

By ALFRED C. JORDAN, M.D., CAMB., M.R.C.P.,

LONDON.

I HAVE the honor to address you on one of the most fascinating and far-reaching branches of medical study; a vast realm that has been opened up by the genius and insight of our great surgeon, Sir Arbuthnot Lane.

Intestinal stasis is a chronic disease due to the absorption of poisonous substances from the alimentary canal. The disease itself may be so insidious as to escape notice, while a complication may produce severe and urgent symptoms, and compel instant attention. In this way we have hitherto lost sight of the fundamental disease—the stasis—and we have fixed our attention on the complication, regarding this as a primary disease.

The general signs and symptoms of intestinal stasis are too well known to require a detailed description. A full and most graphic account is contained in Sir Arbuthnot Lane's recent paper (*Practitioner*, March, 1914).

I am concerned with the radiological demonstration of the changes found in cases of intestinal stasis. When first I took up this work I was handicapped by the belief that the patient must be "prepared" for the bismuth meal by giving him purges and enemata, and that he must take his bismuth in the form of a "meal" while fasting. Many radiologists adhere to this mode of preparation still, but I am perfectly sure that once they have made up their minds to abandon it they will never return to it.

The best way to give the bismuth is in the form of an emulsion to be taken about an hour after an ordinary breakfast. The reasons for this were given fully by me in a recent paper (*Brit. Med. Jour.*,

November 22, 1913). In the vertical posture the emulsion is seen to pass through the esophagus rapidly, and to fall at once to the great curvature of the stomach. The patient then lies on the couch on his right side to allow the bismuth to fill the pylorus and duodenum. After a minute or so he lies on his back, and is examined with the fluorescent

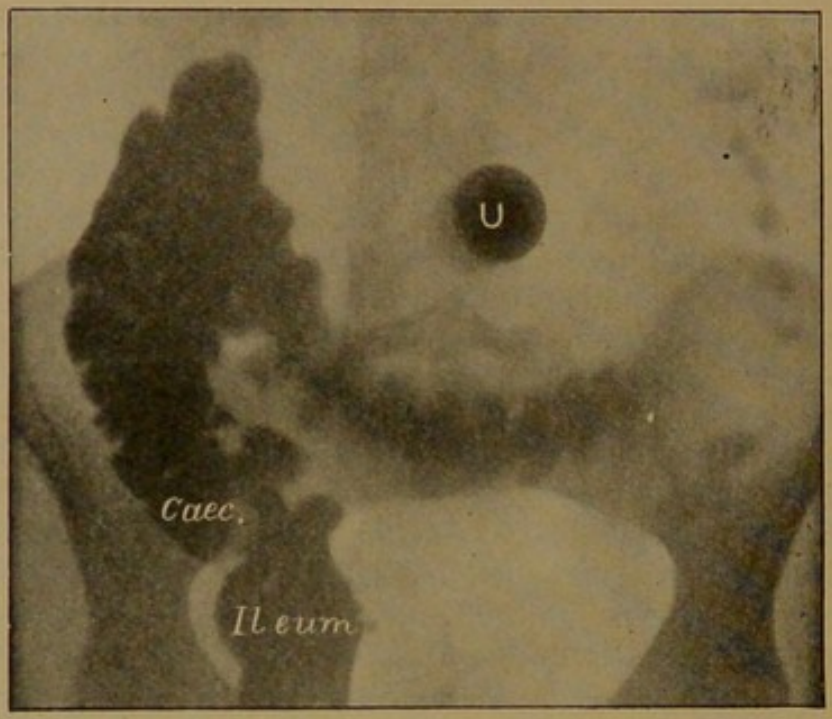

FIG. 1.—Simple ileal stasis, taken on the couch 37 hours after a bismuth meal in a woman aged 40 suffering from advanced cystic disease of the breasts.

screen. In the normal case the duodenum is small and short, its vertical portion measuring 2¾-3¼ inches; in a few seconds a duodenal peristaltic wave starts near the top of the duodenum, and carries before it the whole contents of the duodenum without delay through its four parts and on to the jejunum. At the end of three or four hours there is no longer any bismuth in the stomach or duodenum; the whole of it is in the lower abdomen, partly in the lower coils of the ileum, partly in the cecum and ascending colon. In perfectly normal cases the lower ileal coils lie above the pelvic brim, and there is never a very large collection of bismuth in these coils, the passage through the ileocecal valve being free. One of the very earliest effects of stasis is to cause the lower ileal coils to drop into the pelvis. There is then a long rise from the pelvis to the cecum. Spasm of the ileocecal valve occurs, and adds to the difficulty. The last inches

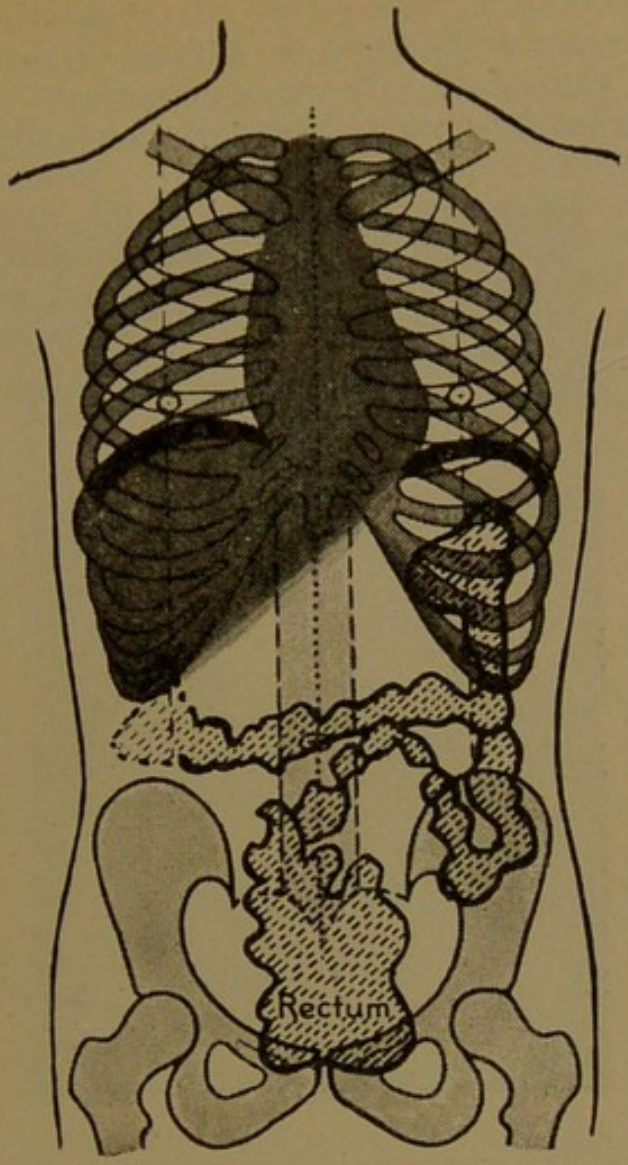

FIG. 2.—Great stasis in the large intestine, with elongation of the pelvic colon, taken on the couch 80 hours after a bismuth meal in a woman aged 46 suffering from advanced cystic disease of the breasts. There was also extreme ileal stasis, and the patient showed all the usual signs and symptoms of chronic intestinal stasis.

of the ileum become hypertrophied to a thick cord which is easily felt. In many cases the weight of the overloaded cecum and ileum causes a constant pull on the mesentery of the ileum whenever the patient is upright; a thickening then appears in the mesentery at the point where the pull is greatest. The point of greatest strain varies from case to case; often it is within an inch of the ileocecal valve (Fig. 14); sometimes it is in the right iliac fossa about four inches from the valve (Figs. 4, 5, and 19); in other cases again it is just above, or just below the pelvic brim.

It is clear, then, that the ileal kink is not the primary cause of the ileal stasis, although the kink, when present, aggravates the stasis, at any rate in the upright posture. I find there is still much mis-

understanding on this point. It is the ileal stasis which produces the kink, by pulling on the mesentery of the ileum. Some of the worst cases of ileal stagnation occur in feeble women, and in them a kink may never appear (Fig. 1). In such cases there is often extreme dropping of the large intestine, the cecum occupying the deepest part of the pelvis. The ileal contents have not then to negotiate a rise to the cecum; the difficulty must be enhanced by the ileocecal valve, which gets into a state of spasm seldom fully relaxed.

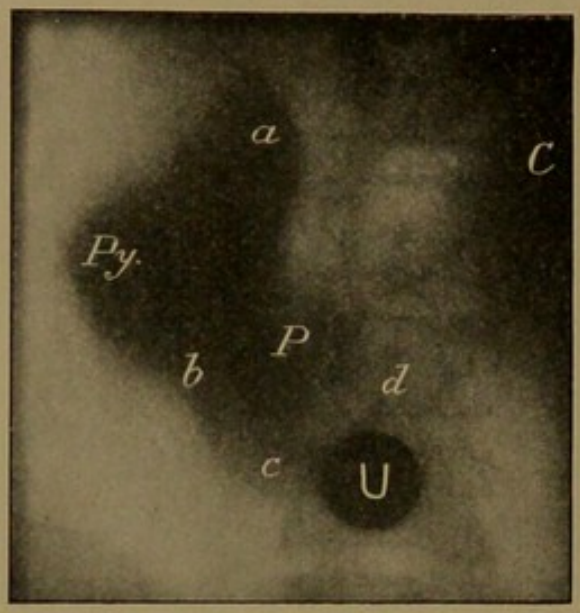

FIG. 3.—Taken on the couch after a bismuth meal in a woman aged 47, showing the duodenum and the plyoric portion of the stomach. The woman suffered from severe glycosuria; at the age of 30 she had exophthalmic goiter for six months. The screen showed active gastric peristalsis with plyoric spasm. The duodenum was dilated, and showed strong "writhing" peristalsis with repeated return of the bismuth from the third to the second part of the duodenum, only traces of bismuth having entered the jejunum at the end of 15 minutes. C, P, cardiac and pyloric portions of the stomach; Py., pylorus a, b, c, d, the four parts of the duodenum; U, the umbilicus. (See also Figs. 4, 5, and 6.)

With regard to the large intestine I propose to say little, except incidentally to explain its effect on other parts. In normal cases the bismuth begins to enter the cecum in three to four hours; in six to eight hours it has reached the middle of the transverse colon; in eight to ten hours the splenic flexure, and in eighteen to twenty-four hours the rectum. At this stage (twenty-four hours) there is usually bismuth in all parts of the large intestine, fairly evenly distributed. At the end of forty-eight hours all the bismuth should have been evacuated. The delay in the large intestine in stasis is often extreme, and after one hundred hours there may be

little bismuth beyond the splenic flexure. Undoubtedly a good deal of toxic absorption occurs from the stagnant contents of the large intestine,

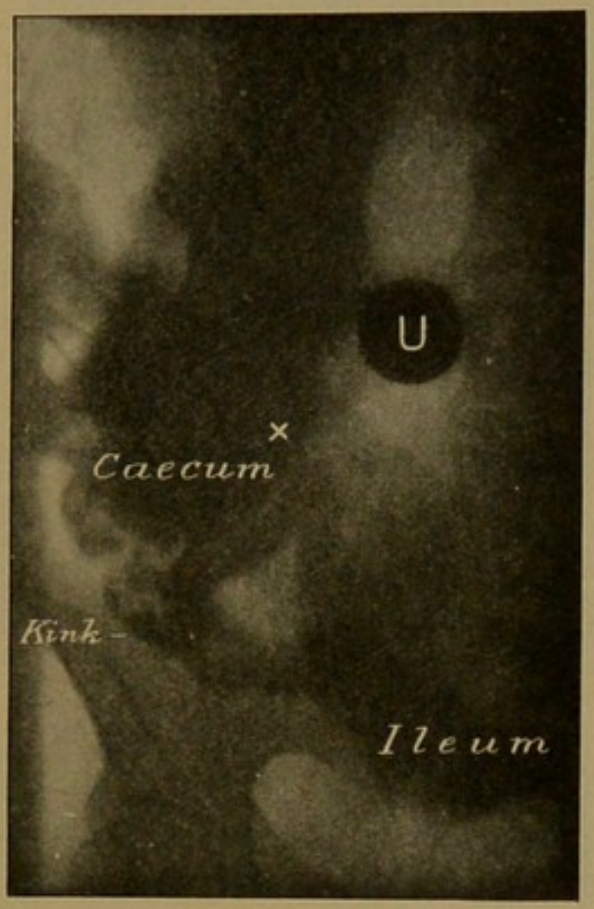

FIG. 4.—Taken on the couch 9 hours after the same bismuth meal. There was still a large amount of bismuth in the stomach—kept back by the pyloric spasm. The terminal coil of the ileum was tortuous and thick-walled; it was firmly fixed in the right iliac fossa (Lane's kink). The appendix, seen beneath the cecum, was freely movable, and appeared healthy.

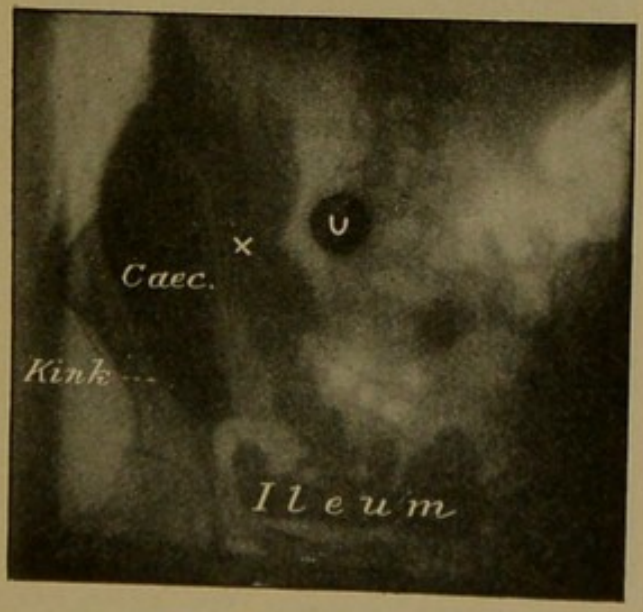

FIG. 5.—Taken 23 hours after the same bismuth meal, showing the ileal kink as before with extreme ileal stasis, the lower ileal coils being still well filled with bismuth.

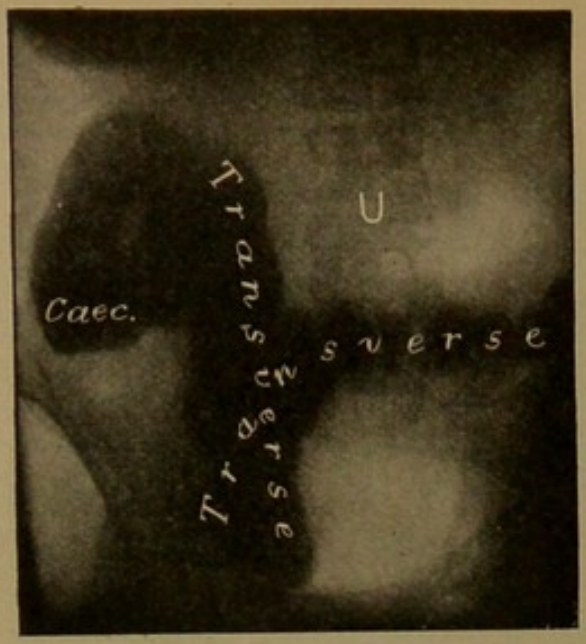

FIG. 6.—Taken on the couch 47 hours after the same bismuth meal; i.e. at a time when all the bismuth would have been evacuated in a normal case; the transverse colon dips vertically into the deepest part of the pelvis, and no bismuth has advanced beyond the middle of the transverse colon. After 95 hours no bismuth had been passed, and very little had got beyond the transverse colon. A few days later diabetic coma supervened, and the patient died.

but the greatest harm arises from the damming back of the contents of the ileum, these ileal coils becoming infected with microbes from the cecum. The ileum is sterile in health, and is not equipped by

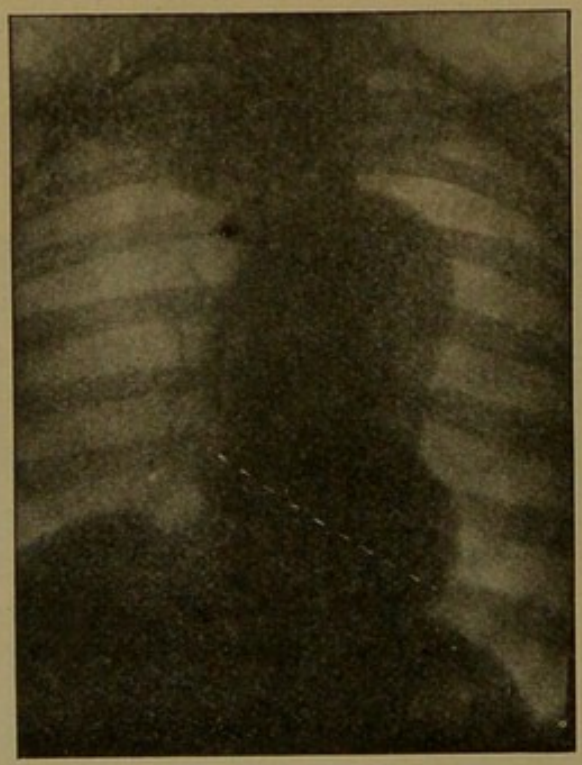

FIG. 7.—Atheromatous elongation and dilatation of the aortic arch, in a "stasis" subject. The dotted line is in the long axis of the heart, more oblique than normal in consequence of the aortic elongation. No tissue escapes the deleterious action of the intestinal toxins.

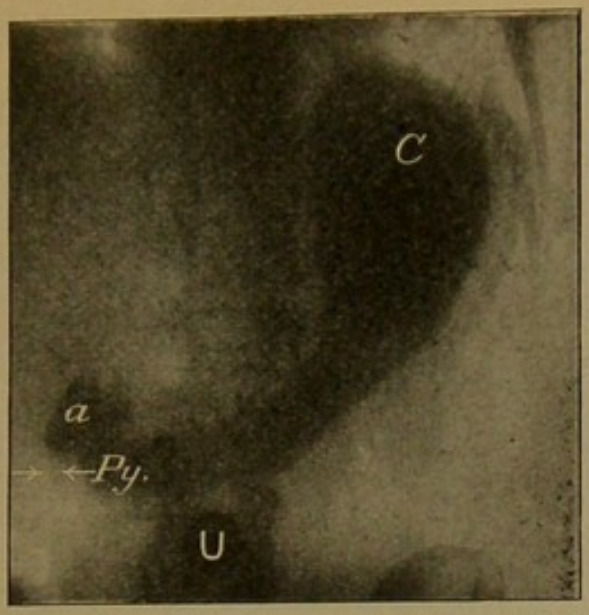

FIG. 8.—Stomach and duodenum, taken on the couch after a bismuth meal in a man aged 47, suffering from mucous colitis and a rheumatoid left hip. Both had persisted for a year. There had been no symptoms referable to the stomach or duodenum. The first part of the duodenum is much dilated and tensely filled, ending below in a blunt point beyond which no bismuth was seen to pass. The obstruction was due to spasm set up by a duodenal ulcer (in the position of the arrows). C, P, cardiac and pyloric portions of the stomach; Py., pylorus; a, the first part of the duodenum; U, the umbilicus (see also Figs. 9-12).

Nature to deal with microbic invasion, while the large intestine can cope with a considerable amount of bacteria.

The absorption of poisons into the circulation

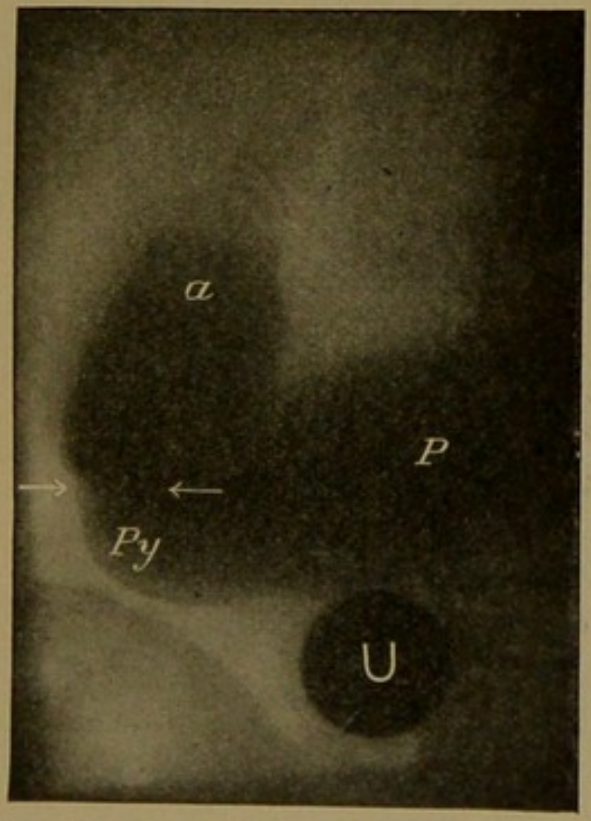

FIG. 9.—Showing the pyloric end of the stomach and the dilated first part of the duodenum in the subject of Fig. 8.

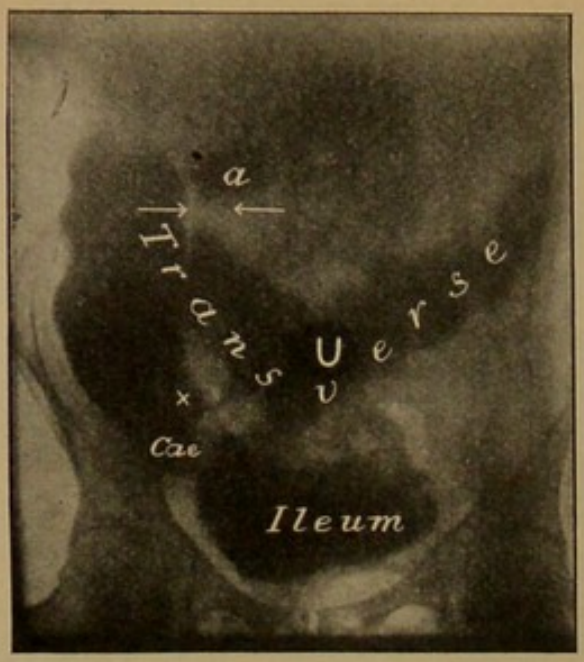

FIG. 10.—Taken on the couch 6 hours after the same bismuth meal, showing ileal stasis. There was still a little bismuth in the stomach, and the dilated first part of the duodenum (a) was again shown well filled with bismuth entrapped above the ulcer (arrows). U, umbilicus; the X marks the ileocecal entrance.

enables these poisons to gain access to every organ and tissue of the body, and no tissue escapes their baneful influence. Thus we get many of the general symptoms of stasis; the headache and depression, the aching muscles and joints, the un-

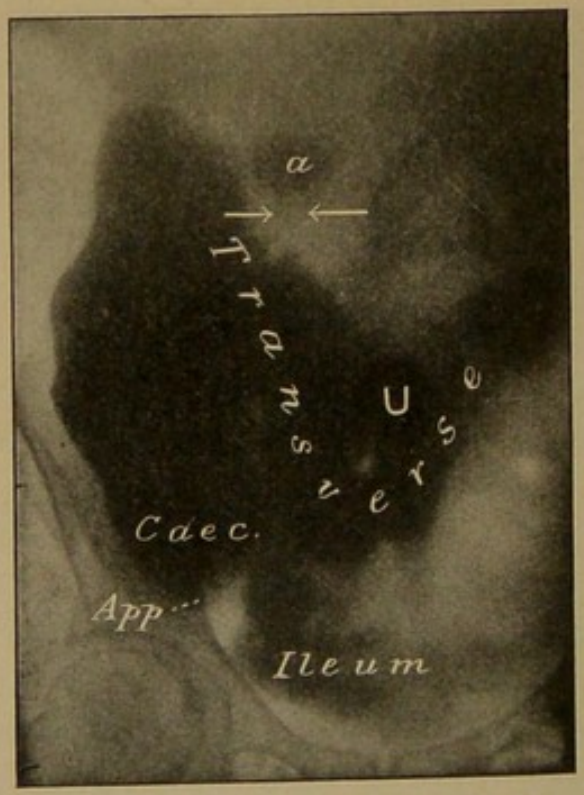

FIG. 11.—Taken 9½ hours after the same bismuth meal, showing the same conditions as Fig. 10 (*q.v.*). The appendix appeared healthy.

healthy skin, etc. The glands suffer early, one of the first changes due to stasis being in the breasts, which become nodular. The condition of the breasts is a very good index for gauging the progress of a "stasis" patient while under treatment. In neglected cases chronic cystic disease appears in the breasts, and finally they become cancerous (Figs. 1 and 2). Other glands suffer, and the pancreas is found to be hard and nodular in operations for stasis. Cancer of the head of the pancreas may be the

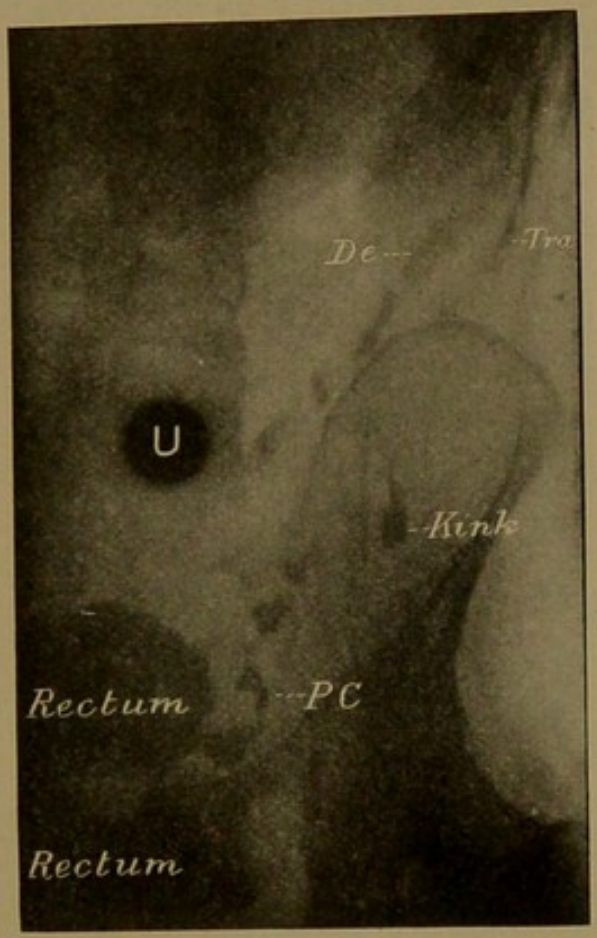

FIG. 12.—Taken 26 hours after the same bismuth meal, showing evidence of mucous colitis, the latter portions of the transverse colon, and the whole of the descending and iliac colon being in a state of tonic contraction, and holding only an irregular thin line of bismuth, mixed with mucus. The iliac colon is firmly fixed in the left iliac fossa at one point, constituting a well-marked "last kink" (Lane).

last stage in the history. The suprarenal glands probably suffer a similar change, and many cases of glycosuria result from changes in the pancreas and the suprarenals (Figs. 3-6). The pigmentation of the skin of stasis subjects is probably due to suprarenal changes. Other ductless glands are affected; the thyroid gland atrophies, or else undergoes changes leading to exophthalmic goiter. The surest proof that these diseases are due to stasis is the fact that they clear up—permanently—on the cure of the stasis, whether by operation or by treatment.

Atheromatous elongation, and later dilatation of the aortic arch occurs at an early age in the subjects of intestinal stasis; this change is easily recognized by radiology, and is capable of accurate measurement (Figs. 7 and 15). The walls of the aorta evidently share the deterioration of all the tissues. It is interesting to speculate how far changes in the pituitary gland, leading to raised blood pressure, act as the exciting cause of the dilatation of the aorta. Rheumatoid arthritis is a frequent result of stasis, and is relieved permanently on the abolition of the stasis (Figs. 8 and 24).

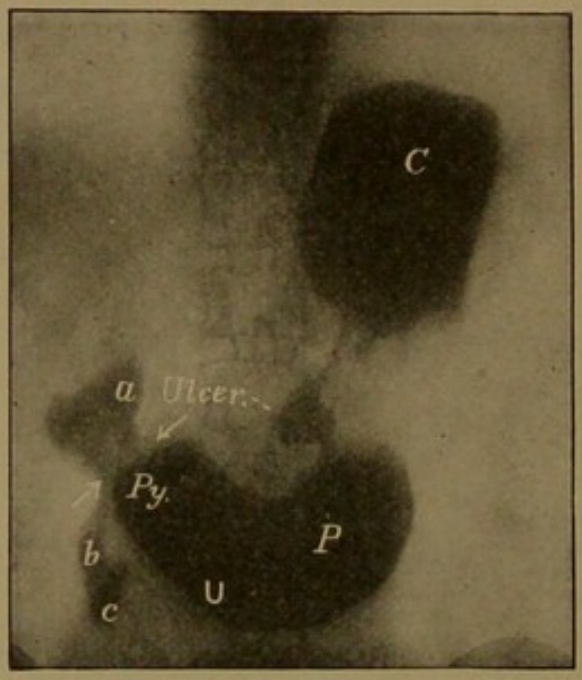

FIG. 13.—Typical chronic ulcer of the lesser curvature in a woman, aged 58, taken on the couch after a bismuth meal, showing the depressed base of the ulcer, and the tight spasmodic constriction of the circular fibers of the stomach over the ulcer. As usual there was persistent spasm at the pylorus, with dilatation and dropping of the pyloric portion of the stomach. The duodenum was elongated and dilated, especially its first part; it showed marked "writhing" contractions, with repeated regurgitation, only small amounts entering the jejunum in spite of the strong duodenal peristalsis. After 24 hours fully one-third of the bismuth was still in the pyloric portion of the stomach. C, P. cardiac and pyloric portions of the stomach; U, umbilicus; Py., pylorus; a,b,c, first, second and third parts of the duodenum. The arrows indicate the persistent pyloric spasm, resembling organic stenosis (see also Fig. 14).

The microbes which have entered the stagnant ileum from the cecum ascend to the upper reaches of the small intestine. Thus the duodenum becomes infected, and the ducts which open into it cannot escape infection. In this way we get the gall-bladder infected; it becomes distended, and gall-stones are formed in it.

This brings us to another aspect of stasis—one of the most important of all, and the one which is

still most open to doubt by the medical public, although the facts are so clear that there is no room for doubt. I refer to the effects of intestinal stasis upon the duodenum and the stomach. In studying this part of the subject radiology is able to give the greatest assistance, and the changes revealed by a bismuth meal are most striking and characteristic. I have already described the appearance and behavior of the normal duodenum. In stasis the state of affairs is quite different: the duodenum is much too large; its vertical part measures 4, 5, or even 6 inches in length instead of 2¾-3¼ inches, as in

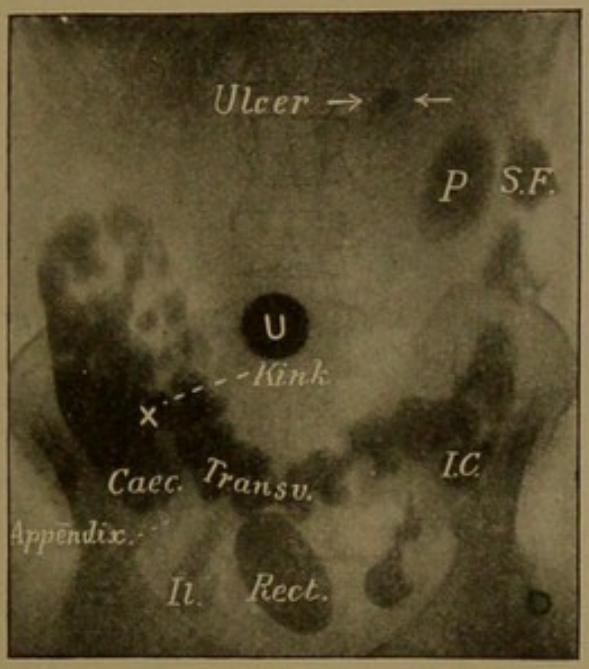

FIG. 14.—Taken on the couch 47 hours after the same bismuth meal, showing bismuth still present in the pyloric portion of the stomach and in the depressed base of the ulcer. There was an ileal kink; the appendix was normal. There was great stasis in the large intestine, and at the end of 72 hours most of the bismuth was still in the cecum, the ascending colon and the dropped transverse colon. (Confirmed by operation by Sir Arbuthnot Lane, and all the symptoms—both general and those due to the gastric ulcer—relieved by "short-circuiting" the ileum into the rectum. The stomach was not touched. Six months later she was at work, feeling quite well.) P, pyloric portion of stomach; Il., ileum; S. F., splenic flexure; I. C., iliac colon. The X marks the ileocecal entrance.

healthy subjects. These measurements are taken orthodiagraphically, so they are strictly comparable. The duodenum is also much wider than normal; often it is double the normal width. Its first part in particular is often greatly dilated. The increased size of the duodenum is not the only change, though it is a sufficiently striking one. Far more remarkable is its altered behavior. It fills well with bismuth (provided the conditions be arranged suitably), but it does not empty itself into the jejunum with a single peristaltic wave as in normal cases; the

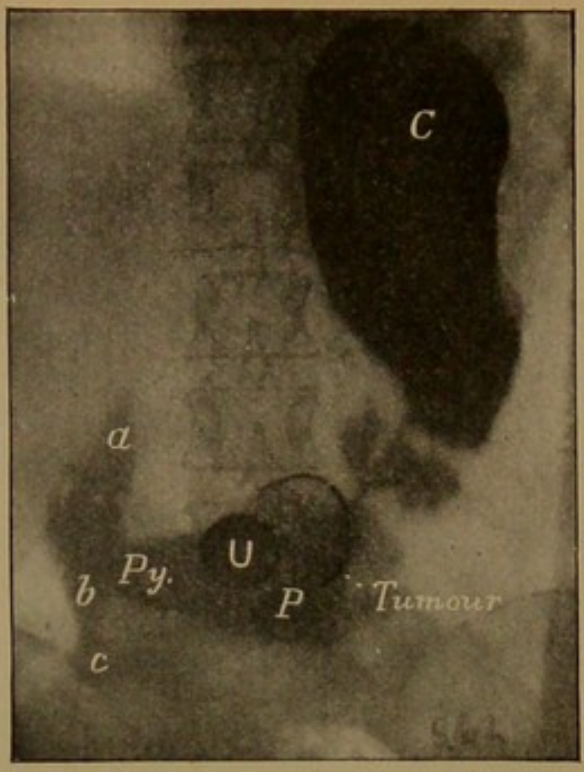

FIG. 15.—Taken on the couch after a bismuth meal in a woman aged 60, showing a carcinoma of the stomach. The *x*-ray appearances are those of chronic ulcer in caricature. The wire marks the outline of a tumor which was felt. The duodenum was dilated, and showed well-marked "writhing" contractions. The aortic arch showed considerable atheromatous elongation, and slight fusiform dilatation. C, P, cardiac and pyloric portions of the stomach; Py., pylorus; a, b, c, first, second and third parts of the duodenum; U, umbilicus. Undoubtedly the tumor originated in a chronic ulcer of the lesser curvature, though the deviation from the typical form is considerable, the tumor being a large one.

"static" duodenum shows excessively powerful peristalsis, wave after wave passing along its four

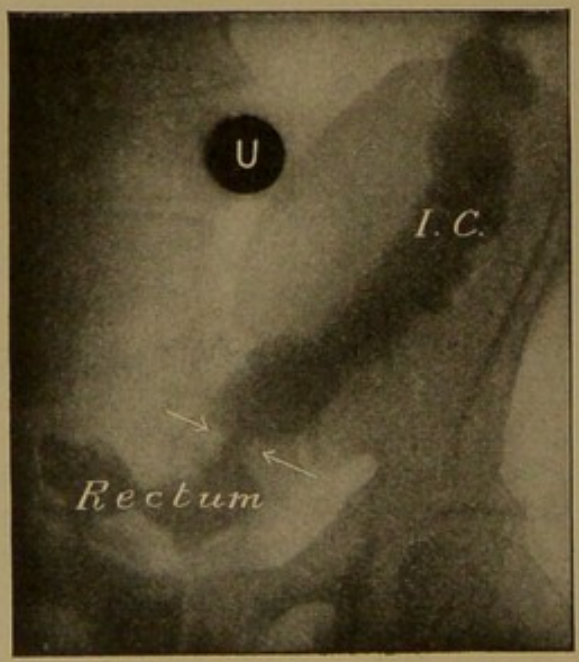

FIG. 16.—Cancer of the pelvic colon, taken on the couch 96 hours after a bismuth meal in a man aged 56. I. C., iliac colon; the arrows show the seat of obstruction in the pelvic colon.

parts; one has only to look at it to realize that it is working against an obstruction; the bismuth is driven down to the lowest part of the duodenum, and often well on into the fourth part, only to return again, time after time, to the vertical part as each wave passes over. The whole duodenum alters in form continually during the occurrence of this phenomenon, giving the appearance of "writhing." In severe cases this "writhing" peristalsis continues hour after hour, a single spurt of bismuth finding its way through to the jejunum every ten minutes or so. The duodenum seems never to tire. Of course it should be understood that all stages occur between the normal duodenum and the extremely dilated, writhing duodenum of severe stasis.

What proof have we that the static duodenum is

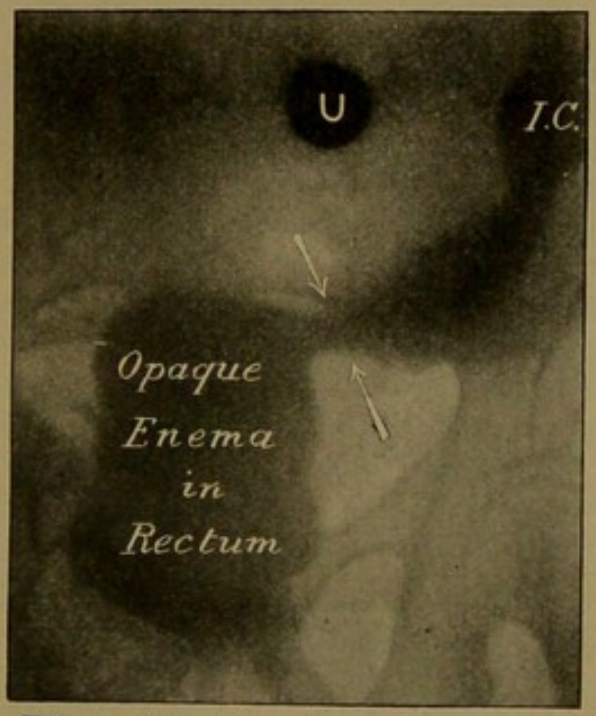

FIG. 17.—Taken after Fig. 16, the rectum having been filled with barium fluid, to show the obstruction in the pelvic colon.

due to the downward pull on the commencement of the jejunum caused by overloading of the lower ileal coils? The proof is manifold and complete. In the first place the downward pull on the jejunum can be observed at operation. The top of the jejunum, moreover, is found empty, and often it is the subject of torsion, the effect of the duodenojejunal kink being increased greatly by this torsion. The radiologist, while examining the duodenum, can actually see the bismuth forced into the fourth part of the duodenum, almost up to the junction with the jejunum; eventually some bismuth is forced through, and then the radiologist can see

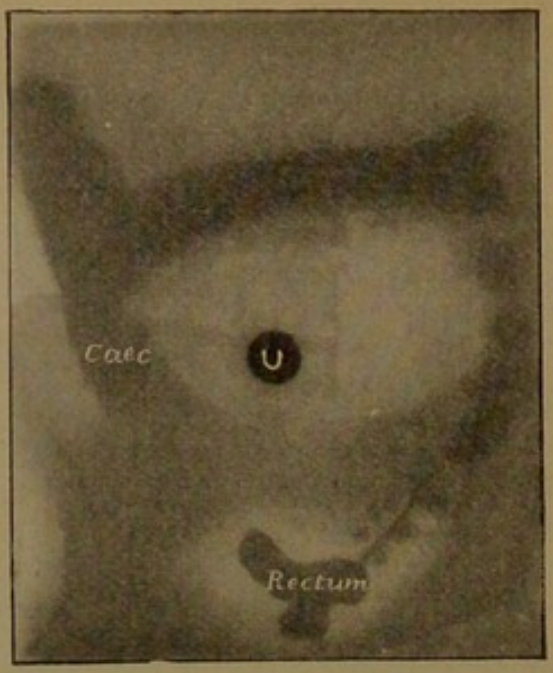

FIG. 18.—"Diverticulitis" of the pelvic colon in a man aged 54. Severe symptoms, including albuminuria and a high blood pressure, were relieved by the operation of "short-circuiting" carried out by Sir Arbuthnot Lane.

that the jejunum passes down vertically at its commencement. A few hours later he is able to convince himself that there is stasis in the lower coils of the ileum. Few things in medicine are more constant; if we find a distended duodenum we are certain to find ileal stasis, and conversely, if we find a normal duodenum, with free duodenojejunal junction we are most unlikely to find any material amount of stasis in the lower ileum. The proof is more perfect even than this: there is a definite

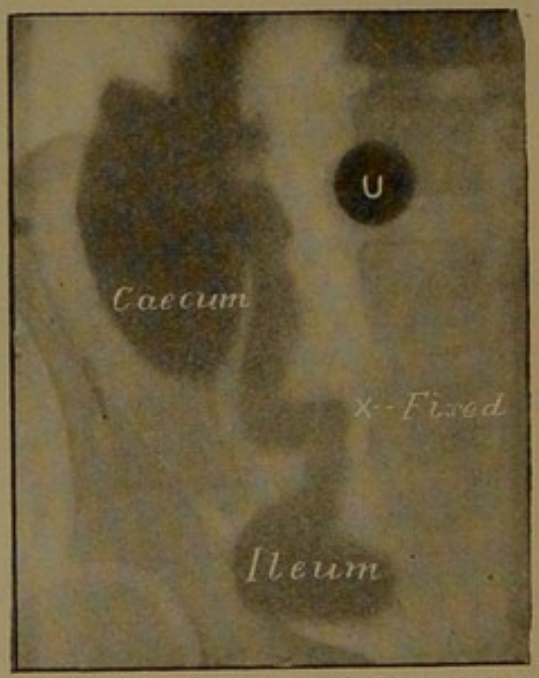

FIG. 19.—Ileal kink in a man aged 57, taken on the couch 10½ hours after a bismuth meal. (See also next Fig.)

quantitative relation between the amount of ileal stasis and the amount of duodenal distention; thus a high degree of duodenal distention will be found associated with great ileal stasis, while slight duodenal distention goes with slight ileal stasis. Of course the proportion is not mathematically accurate, but it is nearly enough so to furnish clinching proof of the dependence of duodenal distention on ileal stasis.

The distended duodenum causes no subjective symptoms, but if it is congested the patient becomes aware of its presence at once; he feels pain in this region, and there is tenderness to pressure over it; the tenderness is often attributed to the gall-bladder. If the duodenum has become infected

FIG. 20.—Taken 47 hours after the same bismuth meal, showing obstruction by bands just beyond the hepatic flexure. (Confirmed by operation.)

with microbes from the stagnant ileum we shall probably get ulceration in the duodenum (Figs. 8-12). No normal duodenum gets ulcerated; a chronic duodenal ulcer occurs only in the distended duodenum of chronic intestinal stasis. It follows that one cannot have a duodenal ulcer without ileal stasis, and this accords entirely with my experience.

The distended duodenum has a very definite effect on the pylorus, which closes tight to prevent reflux from the overfull duodenum. The pylorus gets into a state of permanent spasm (Fig. 13), its contents become too acid, and this leads to a further increase of the spasm, for physiological experiments

have proved that no acid can enter the duodenum without setting up an immediate tight closure of the pylorus. There is delay in the emptying of the stomach, and this organ is overloaded and dilates. The great curvature drops, and the transverse colon, also heavy with the weight of its stagnating contents, drops with it. Thus we get an abnormally great strain on the lesser curvature, and especially on the two "ligaments" of the stomach—the pylorus and the esophagus. A chronic ulcer is apt to appear at the pylorus, this part being accessible to the microbes which infect the "static" duo-

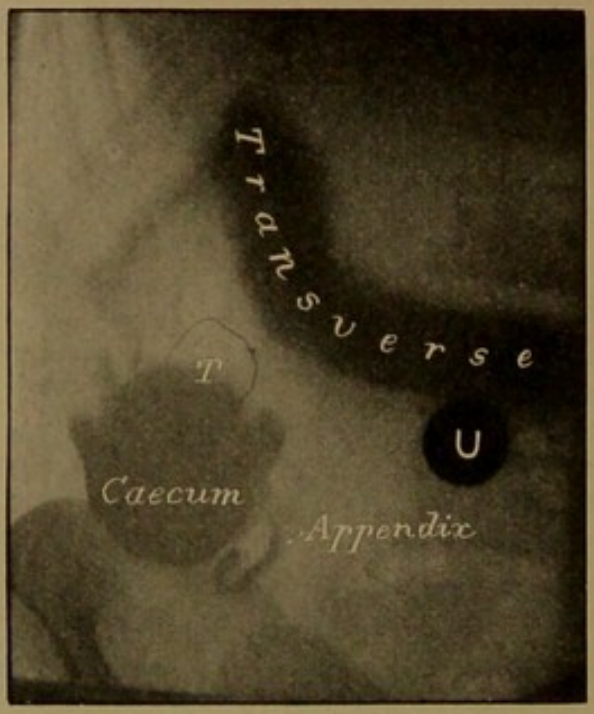

FIG. 21.—Cancer of the ascending colon in a man aged 57. There was a movable tumor in the position marked by a wire. A thin streak of bismuth ran along the outer side of the ascending colon, the tumor being attached to the inner wall. The appendix (normal) is shown well filled with bismuth.

denum, or, if the pylorus drops, the point of greatest strain is shifted toward the esophagus, and the ulcer appears at some point along the lesser curvature (Figs. 13 and 14).

With the appearance of an ulcer in the stomach the acidity of the gastric contents increases still more, and the pyloric spasm becomes so severe and constant as to simulate organic stenosis (Figs. 13 and 14), and the radiologist must be very wary in the manner of carrying out his investigations to avoid this pitfall.

The *x*-ray appearances of chronic ulcer of the lesser curvature are quite characteristic, especially on the couch. The depressed base of the ulcer fills

with bismuth; the raised margins of the ulcer are shown, and there is a tight hour-glass constriction of the stomach over the region occupied by the ulcer, so that the stomach appears divided into two parts, separated by a narrow isthmus (Fig. 13). An important point to remember is that this hour-glass constriction is permanent except during general anesthesia. The radiologist must bear this in mind, lest he diagnose a cicatricial contraction, and the surgeon must not forget it, for when operating

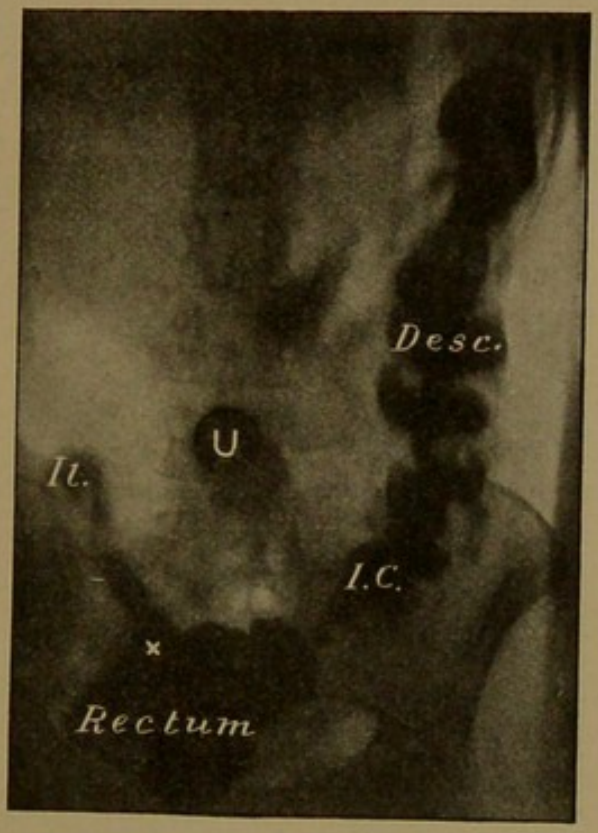

Fig. 22.—Taken 11 hours after a bismuth meal in a woman aged 35, short-circuited a year previously by Sir Arbuthnot Lane. Showing the termination of the ileum, and the rectum full of bismuth. Some bismuth had run up to the splenic flexure; the following morning this had come down again. Il., ileum; I. C., iliac colon; the X marks the union between the ileum and the rectum.

under general anesthesia he does not see a constriction, and he may not realize that the stomach will be drawn in again at the seat of the ulcer as soon as the patient has recovered from the anesthetic. I have seen a surgeon make his gastrojejunostomy opening at the level of a chronic ulcer of the lesser curvature and the result was promptly fatal.

If we were to make an artificial hour-glass constriction in a healthy stomach we should expect the pyloric portion of that stomach, beyond the constriction, to become small (for it would never be properly filled), just as the entire stomach becomes

small in cases of stricture of the esophagus. The fact is exactly the opposite in chronic ulcer of the lesser curvature; the pyloric portion of the stomach is always large and dropped (Fig. 13); in the most extreme cicatrical hour-glass stenosis of the stomach the great curvature may be in the pelvis in the upright posture. This affords the most striking confirmation of the fact that the chronic ulcer of the lesser curvature occurs, not in a healthy stomach, but in the enlarged stomach produced by constant pyloric spasm.

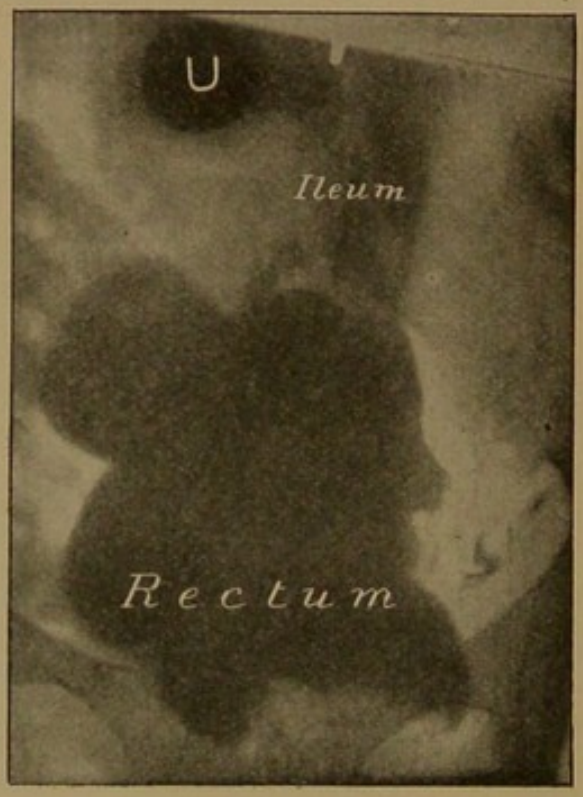

FIG. 23.—Taken 5 hours after a bismuth meal in a woman aged 26, nine months after colectomy. All the bismuth is in the rectum except traces in the last four inches of the ileum. The duodenal distension and the pyloric spasm had been relieved almost completely.

The symptoms caused by these chronic gastric ulcers are often vague and difficult to interpret clinically, and they are not often diagnosed correctly except by the *x*-ray method, when the diagnosis can be made with certainty as a rule. One of the dangers of leaving these ulcers untreated is due to their tendency to become malignant after a time, and I have a number of instances of carcinoma of the stomach exemplifying various stages in the transition from a typical chronic ulcer to an extensive malignant growth. The *x*-ray appearances are those typical of a chronic ulcer, but with more or less deviation from the typical picture due to the malignant involvement (Fig. 15).

Thus intestinal stasis is shown to be an important cause of cancer, not only in the stomach, but also in the pancreas, the liver and bile-ducts, and in the breasts. Cancers occur also in the large intestine as the result of stasis; this is readily shown. In the rectum (Figs. 16 and 17) and in the cecum (Fig. 21) the long-continued irritation of stagnant feces is responsible for the growth; in the hepatic flexure region and in the first part of the transverse colon immediately beyond the hepatic flexure fecal accumulation is often due to bands continued down from the lower surface of the

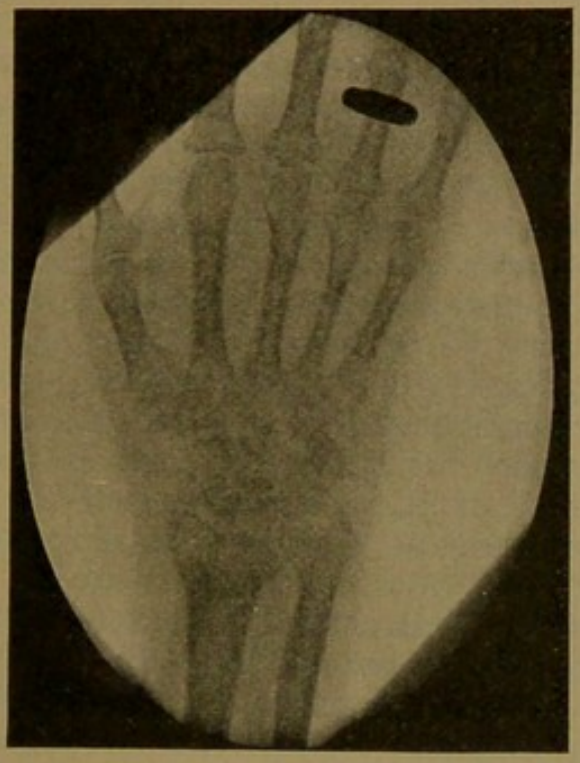

FIG. 24.—Rheumatoid arthritis; a disease frequently seen in the subjects of chronic intestinal stasis. The cancellous tissue of the bones is rarefied. The joint trouble subsides permanently on the relief of the stasis.

liver over the pylorus and gall-bladder (Fig. 20). These bands are found in a fair proportion of stasis subjects. The iliac colon, again, is often tied to the left iliac fossa by bands, these being the first to form in the body. These bands sometimes cause obstruction and lead to a condition of chronic congestion with yielding of the bowel-wall between the bands and the production of the disorder known as "diverticulitis" (Fig. 18). Following upon this long-continued obstruction we may get the appearance of a cancer in this region.

Needless to say the bearing of all this new knowledge on treatment is of the greatest importance.

I propose to say but little concerning treatment,

and only in its relation to radiology. Having ascertained the existence of stasis in a particular case, and the presence of one or more of its complications, the question will always arise—should we go to the root of the matter and deal with the stasis, or should we apply our treatment to the complication,—the end result of the stasis—*e.g.* a gastric or duodenal ulcer. The answer is simple:—leave the end result alone if you can, *i.e.* if it is not causing stenosis, or becoming malignant, or impacted in the case of gallstones. If you treat the end result (*e.g.* by performing a posterior gastro-jejunostomy for ulcer) you will give your patient temporary relief which may be great, but you will not cure his stasis. Or again, if you merely remove his appendix, which has become (secondarily) congested or kinked, no lasting benefit will result. In proof of this I would mention the very large number of patients sent to me for investigation by the *x*-rays at some period after one of these operations had been performed. They are sent to me because they are not cured; they are suffering from the general symptoms and signs of stasis; or perhaps some other complication has arisen, *e.g.* colitis.

The rational treatment of stasis is directed to the abolition of the undue retention of the contents of the lower ileum and the large intestine. In slight cases this may be done by the administration of liquid paraffin to accelerate the progress of the feces through the large intestine, and by a spring support for the lower abdomen, to prevent the dropping of the large intestine and the lower coils of the ileum, and to obviate the evil consequences of an ileal kink if there be one.

In severe cases of stasis, too advanced to gain sufficient relief from the above treatment, operative measures are needed; short-circuiting the ileum, near its lower end, into the rectum, and making a kink in the iliac colon above the short-circuit to prevent or diminish the regurgitation of the ileal contents from the rectum into the descending colon. Often there is already a well-developed kink in this region (Fig. 12), or there is a slight kink which can be made more effective at the operation. In some cases, where the large intestine is very unhealthy, and would cause flatulent distension and other trouble if left, Sir Arbuthnot Lane now removes the whole big bowel above the short-circuit opening at the time of the first operation. Formerly he would do this at a subsequent operation if it proved neces-

sary; with increased experience, however, one is now able to judge, in a particular case, whether the big bowel must be removed, or may be retained. All the details of the operations have been described by Lane, and are now well known and are practised extensively by a number of prominent surgeons throughout the world.

We come now to the results of the radical treatment of stasis from the radiological standpoint: Can we show, by radiology, that the stasis has been relieved? Certainly we can; in a successful case the whole bismuth meal is in the rectum after 6 or 8 hours (Fig. 23). The stasis has been cured. If the large intestine has been left there may be some bismuth in the descending colon for a few hours (Fig. 22), but as soon as the patient has an evacuation this comes down and is passed with the rest. Some of the cases in which the bismuth passes back, not merely into the descending colon but right back to the cecum, are those in which the subsequent removal of the big bowel becomes necessary on account of flatulent distension and tenderness due to the irritation set up in the bowel wall by the retention of lumps of secretion in the big bowel.

The question will be asked, what is the effect of the radical cure of stasis upon the distended duodenum? If the explanation ascribing the "static" duodenum to the downward pull on the upper jejunum be the true one, then the cure of the ileal stasis should abolish this downward pull, and should, therefore, relieve the duodenal distention. Does it do so? Yes. I have reinvestigated a large number of patients after short-circuiting, at varying intervals, from three weeks to several years, and the general conclusions are as follows: The duodenal obstruction, as shown by "writhing" contractions, and repeated return of the bismuth toward the pylorus, are relieved at once by the 3 or 4 weeks recumbency following the operation, and do not come again even after the patient has resumed ordinary conditions of life and work. The duodenum does not return at once to its normal size, but gradually—in the course of months—it becomes smaller and shorter, though in old, neglected cases the duodenum tends to remain permanently more "baggy" than normal; but the chief point is that the duodenum never again becomes distended; it is no longer found full of bismuth; its outlines can be sketched only by observing small quantities of bismuth as they pass through it—which they do

without delay—into the jejunum. No photograph of the "cured" duodenum can be taken, in marked contrast to the state of things before the operation, when a most striking photograph of the distended duodenum is readily obtained.

Another proof of the efficacy of the radical treatment is shown in the stomach, which loses its constant pyloric spasm, so that the rate of emptying of the stomach becomes normal, or nearly so within a few months after the operation. This was exemplified to me in a most striking way recently when I reexamined a woman six months after she had been "short-circuited" by Sir Arbuthnot Lane. Before the operation she had furnished one of my most striking instances of the distended, "writhing" duodenum, and scarcely any bismuth had left the stomach after 6½ hours. Now (6 months later) the whole of the bismuth was in the rectum after six hours, a little having run up the descending colon, and the whole had been evacuated within 24 hours. The duodenum could not be photographed, the bismuth passing through it, little by little, without difficulty, so that the duodenum was never seen filled with bismuth, in striking contrast with its behavior before the operation. Thus the patient has been relieved of all the signs and symptoms of stasis, and her stomach and duodenum have been put out of danger; the duodenum is no longer infected with microbes from the stagnant ileum, and the duodenal distension and overloading of the stomach have been rectified.

I have given details of other aspects of stasis in a contribution to the *International Journal of Surgery*, for April, 1914, with a series of skiagrams illustrating many of the changes due to the disease.

Much progress is being made on both sides of the Atlantic in our understanding of Chronic Intestinal Stasis, but there is still scope for searching inquiry by surgeons, clinicians, radiologists, pathologists, and in fact all medical workers, in the realm which has been discovered by the far-reaching observations of Sir Arbuthnot Lane.

Here, in the New York Polyclinic, I see earnest investigators at work on the stasis problem. Dr. Quimby is attacking it from the radiological side, Dr. Hayes and others from the medical and pathological standpoint, while Dr. William Seaman Bainbridge is getting brilliantly successful results from the application of surgical measures to the relief of chronic intestinal stasis.

THYROID DEFICIENCY.

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MYXEDEMA in the severe form was discovered and first studied in England. It is now a well-known pathological entity all over the world and of it I could say nothing but what you know perfectly well. But I will insist upon the mild forms because they are not so well outlined and very often escape the attention of the medical man. I called it when I described it for the first time 1899[1] mild myxedema, myxedema frustum, or benignant chronic hypothyroidism. The conception of mild myxedema was at first strongly opposed because the symptoms were so numerous, that is to say, because the symptomatological range was so great and extended to every organ of the body. Just lately Dr. Leonard Williams of London has said: "The idea of benignant chronic hypothyroidism is simple enough, but the symptomatology is a perfect maze." This is not the fact, provided we begin with a clear understanding of the lesion caused by thyroid want.

If we knew exactly the function of the thyroid gland we would no doubt be able to deduct immediately the symptom of a diminished or impoverished secretion; but we do not. However, we know something. We know that without the thyroid stimulus no cell, whatever it may be, can attain its morphological perfection—the perfection needed for good work, muscular, nervous, connective, glandular, or bone. The proof thereof is that a child born with congenital want or a child deprived completely by an operation of its thyroid gland does not grow, or grows very little. Give it a few doses of thyroid extract and it will begin to develop; stop the supply and immediately progress is stopped.

We know something more: When a cell has done its duty for some time it decays, it is no longer desirable. It must be taken to pieces and eliminated through various channels—bowels, kidneys,

(Reprint from the MEDICAL RECORD.)

lungs—especially under the form of urea. When thyroid supply is scarce the carrying away of the cellular waste matter is slow and incomplete—mucin, fat, and other principles accumulate on the spot, and there form an infiltration and edema of a special kind—hard, non-depressible—and therefrom comes the name of myxedema.

Infiltration Is the Constant Lesion of Thyroid Deficiency.—It may be obvious or it may be slight, but it is always there. If a patient, swollen with this special edema, takes thyroid extract he immediately eliminates a great quantity of urine and urea. He loses in weight, the cellular waste matter is carried away, and when he has got rid of the whole residual stock you may give him doses ten times stronger and he will lose no more. We may sum up and say, thyroid secretion is necessary to the building up and to the dismantling of our tissues—of all our tissues; and, therefore, the want of it finds an echo in all our organs without any exception.

All the infectious diseases of early age and of later on fall heavily on the vitality of the thyroid gland. Acute rheumatism of the joints has a most nefarious influence and causes even after years the outbreak of severe forms of myxedema. And, what is more, all the great causes of pathological disturbance, tuberculosis, syphilis, paludism, alcoholism, chronic starvation, and consanguinity hit their first blow on the thyroid system, and the thyroid deficiency thus brought on comes down fatally on the offspring.

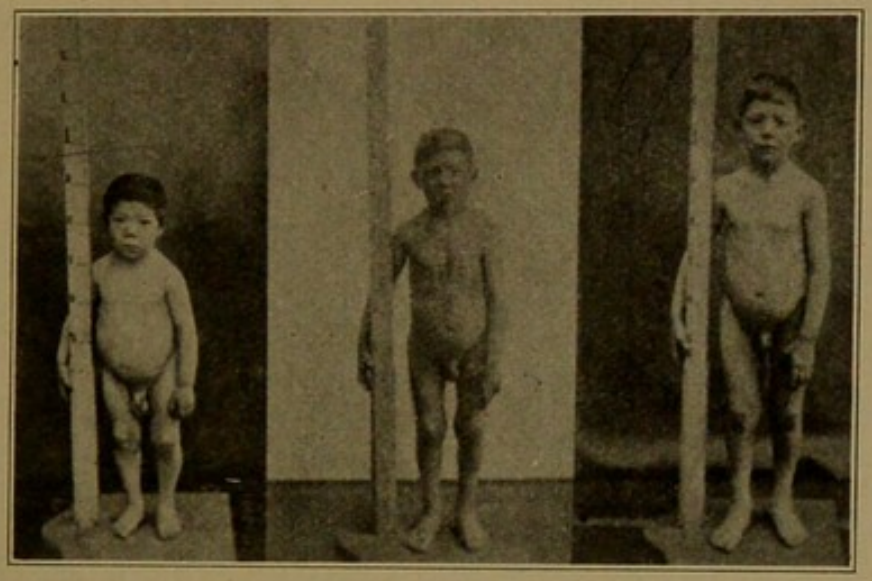
The child shown in Fig. 1 is a remarkable instance of hereditary syphilis and thyroid weakness. The father married shortly after having contracted syphilis. The result you see here—growth stopped, cretinous appearance; observe also the result of medication.

If we had seen such a patient some ten or fifteen years ago we would have no doubt diagnosed hereditary syphilis, but there would have ended our healing power—neither iodine nor mercury would have restored the child to normal growth and health. But now that we know that the growth has been stopped on account of the syphilitic toxin having dried up the thyroid well, we act in consequence and have a successful medication, that shows the usefulness of the hypothyroid notion.

The litle girl shown in Fig. 2 is a case of thyroid weakness caused by hereditary paludism. The

mother while pregnant underwent several attacks of intermittent fever. She herself had a weak thyroid. The paludic poison added to her natural disposition had the result of impairing the thyroid powers of the child, and it was born a cretin. This case looked at as merely paludic was hopeless. Seen in the light of thyroid weakness we get a good result.

The boy shown in Fig. 3 was twenty-one years old. He was a perfect dwarf, absolutely childish, and without any intellectual development. Mother healthy. Father died from pulmonary consump-



a *b* *c*

FIG. 1.—Hereditary syphilis. *a*, Arrest of growth, age 10½, height 3 ft. 2½ in.; *b* and *c*, influence of thyroid feeding after 1 and after 2 years.

tion a few days before the child was born. This may be considered as a case of hypothyroidism and tuberculous heredity.

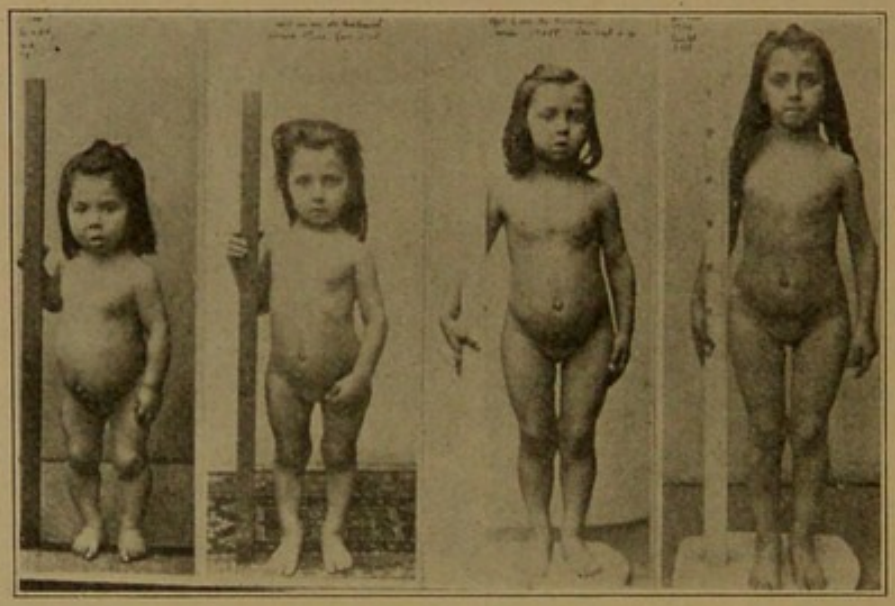
Let us bear in mind that we are inheritors of a great number of generations. Our blood is a mixture of good and bad qualities that our forefathers have left us while struggling with innumerable causes of diseases of all sorts. From all this we may conclude that thyroid weakness is very frequent, at least in a mild form.

Now I must say that thyroid weakness is not synonymous with cretinism. Incomplete forms will go very well with a fairly active and busy life; some of these patients are indeed very intelligent. I wish to draw your attention to these social forms because they are very common, and because the exact knowledge of them enables us to grasp the cause and decide the successful treatment of a

large number of pathological conditions for the occurrence and treatment of which we are otherwise unprepared.

We will now follow up the idea of infiltration which is brought on by thyroid insufficiency, and consider the consequences in various tissues of the body.

Muscles.—The cell loaded with fat and mucin is increased in size so that its contraction is delayed in onset and slow in execution. Muscular action then becomes painful, accompanied by stiffness and dread of movement. The connective tissue sheath



a *b* *c* *d*

FIG. 2.—Hereditary paludism. *a*, Arrest of growth, age 8 years, height 2 ft. 10 in. instead of 3 ft. 10 in.; *b*, *c*, *d*, influence of thyroid feeding, after 1,2, 5 years.

which supports the contractile elements and connects the muscles with the tendons, aponeuroses, and articular ligaments is equally infiltrated, and this adds to the difficulty of movement. This applies alike to non-striped and voluntary muscle.

This muscular infiltration shows itself subjectively by rheumatoid pains, which must not be confused with rheumatism, due to causes with which we are not here to be concerned. The statement made by some writers that thyroid extract cures nodular rheumatism, chronic or otherwise, should, therefore, be accepted with reserve. Thyroid extract relieves pain in the muscles, joints, and ligaments only in so far as this depends on thyroid deficiency; that is, it is due to the specific myxedematous infiltration, and it acts only by causing absorption of that infiltration. We must

remember that thyroid extract is essentially a specific and can act favorably only where there exists an inactivity or weakness of the thyroid.

Let us take another example. The nerve cell, whatever may be the degree of myxedematous cachexia, is never destroyed as in a hemorrhagic focus or embolic necrosis. Its nutrition and excretion are simply hindered. It is infiltrated and at the same time compressed by the infiltration of its supporting connecting tissues. The transmission of motor, sensory, and voluntary impulses is thereby delayed but not abolished, and the reflexes are sluggish but present.

The discomfort of the nerves shows itself by neuralgias and even by shooting pains of a neuritic character; the distinction between these two kinds of pain being well known to the patient. Cardiac pain is also present with radiations into the brachial plexus simulating attacks of angina.

The central nervous system exhibits early evidence of infiltration, however slight it may be. In well-marked myxedema, vertigo, dizziness, noises in the ears, headache, migraine, loss of memory, mental confusion, depression, melancholia, loss of consciousness, loss of balancing power, sudden falls, somnolence, attacks of coma, which may be confused with the serous apoplexy of Bright, with all the more probability that there is usually some albuminuria.

In mild myxedema the list is not so formidable. One should remember the morning headaches and tendency to migraine, vertigo, and noises in the ears.

Even the bone does not escape the consequences of thyroid defect. Every surgeon knows that in certain persons fractures unite imperfectly or not at all, and thyroid extract is given to avoid complication. If one carefully examines such patients, those at least who benefit by it, there will always be found some more symptoms of thyroid deficiency.

Cartilaginous tissue also gives clear evidence of this specific cellular infiltration. On moving the joints which are stiff and painful the application of the hand detects a peculiar sensation resembling the crackling of crushed snow which is almost pathognomonic. This is well felt in the knee joint. Sometimes the patient himself feels and hears a crepitation in the joints of his cervical vertebræ. These joint symptoms were described first by Professor Verriest in connection with a case of

myxedema shown to the Royal Academy of Medicine of Belgium in 1886.[2]

These painful affections of the joints improve very slowly and are the last symptoms to disappear. An ecclesiastic, after several months' treatment for very pronounced myxedema, still complained of the stiffness of his knees which rendered ritual genuflection very difficult, but which ultimately completely disappeared. This delay in functional restoration may perhaps be explained by the slowness of the nutritive exchanges in cartilage.

The glandular tissues which play so important

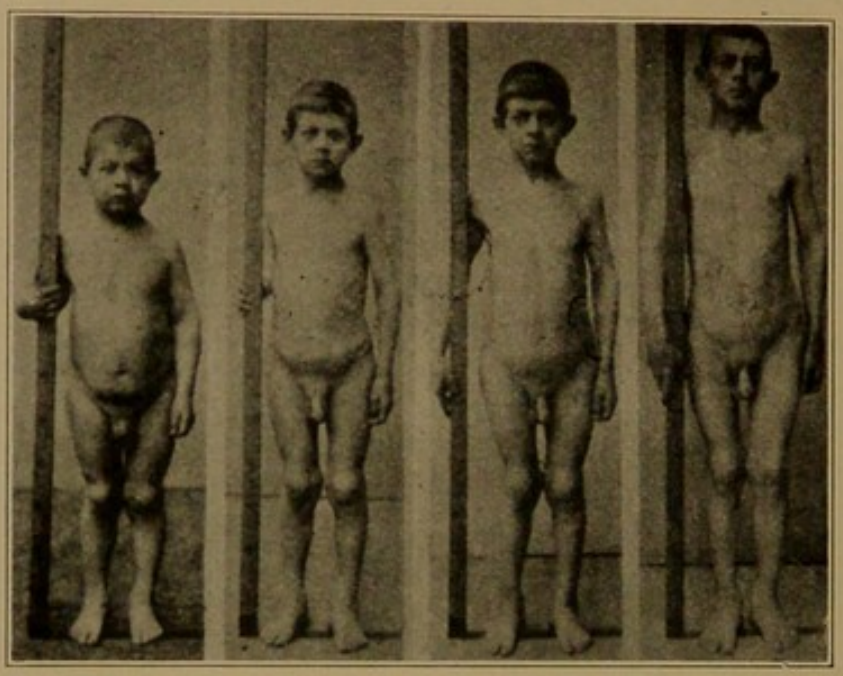


FIG. 3.—Hereditary tuberculosis, thyroid weakness. Arrest of growth; influence of thyroid feeding, after ½, 1, and 2 years.

a part in the organism also present infiltration both of their secreting elements and the supporting connective tissue. The secretion of sweat is completely abolished.

There is considerable congestion of the liver,[3] the hepatic cells secrete badly, while the canaliculi are compressed. Bile passes into the circulation, causing the characteristic amber color of the skin. Biliary calculi are also common.

The scanty intestinal secretion along with the muscular weakness of the visceral walls causes obstinate constipation, which in turn leads to fermentation with the formation of an abnormal quantity of gas, thus producing meteorism and abdominal distention with noisy eructations from the stomach.

The alterations of epidermic and epithelial coverings deserve special notice. The insufficiently

nourished epidermis undergoes early desquamation; the hair falls prematurely. In milder cases the hair is poor, brittle, and becomes early grey, while the beard is thin and straggling. The nails are striated and brittle, and in severe cases split and destroyed. The teeth are almost always in a deplorable condition, exposed, decayed, and covered with a green-

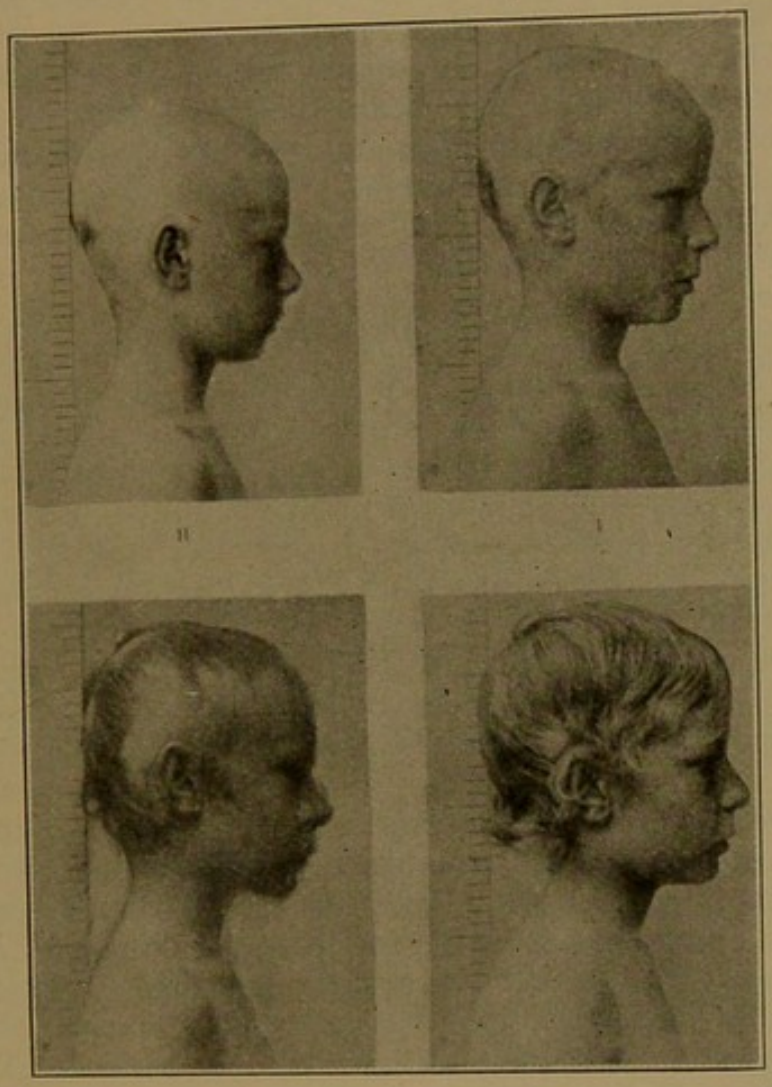


Fig. 4.—Hypothyroidic alopecia; thyroid insufficiency. Influence of thyroid feeding.

ish tartar. The gums are red and irritated, forming polypoid projections between the teeth. The eyebrows are thinned, especially in their outer thirds, giving the face a somewhat silly expression. The eyelashes are also shed, leaving the eyelids unprotected against the erosive action of the tears. This blepharitis of thyroid origin is sometimes seen in old persons. In a case which had resisted all other means of treatment, a radical cure was obtained in a few days by the local application of adrenalin and the administration of thyroid extract. The whole skin is thickened, infil-

trated, cold, and easily attacked by such affections as eczema, psoriasis, and alopecia. The eczematous condition of the scalp in young infants, known as the "milk crust," which is alike the despair of mothers and doctors, yields rapidly to a few doses of thyroidin combined with arsenic, when this affection is associated with thyroid defect, and the same can be said of psoriasis and of certain forms of alopecia.

The little boy pictured in Fig. 4 had lost all his hair. I should never have suspected thyroid deficiency in this case if the mother had not evidently been under thyroid distress. The result of thyroid treatment on growth and alopecia is well shown.'

The mucous membrane of the mouth, lips, tongue, nose, pharynx, larynx, ear and esophagus are also infiltrated.

The vaginal mucous membrane is softened and infiltrated, giving on digital examination a sensation similar to that of the commencement of labor. Similar changes produce disturbances of phonation, deglutition and hearing.

The swelling of the fauces and esophagus may actually prevent swallowing in some cases.

In very severe cases of advanced myxedema, at the approach of death, the enormously swollen tongue tends to fall backward so that the air does not enter the trachea, although the respiratory movements of the thorax continue; the condition being identical with that seen during anesthesia in similar circumstances. After five or six of such false inspirations a spasm of the tongue and pharynx occurs which again allows the entrance of air. Normal respiration is thus reestablished for a time, till again interrupted and restored as before. This condition may be mistaken by an inexperienced observer for a type of Cheyne-Stokes respiration.

Endothelial tissues share in the general feebleness. They are shed prematurely, and such cavities as the gall-bladder and also the urinary bladder are unprotected from the irritating action of their contents. The gall-bladder becomes sensitive, even painful, while the mass of desquamated epithelium may become the nucleus of a calculus. Biliary lithiasis is itself frequent in myxedema, and occurs to a less extent in the milder forms.

The bladder being constantly denuded of its epithelial lining is more than usually sensitive to the irritating action of the urine, and this alone

is responsible for many cases of nocturnal enuresis in children. An examination of the urine in these cases shows the presence of a large number of squamous epithelial cells from the bladder. When closely studied these children present signs of thyroid weakness, and their parents, especially the mother, often exhibit a more or less advanced degree of thyroid defect. As the children grow up the activity of the thyroid gland increases and these troubles disappear. In girls a trace of them frequently persists as pollakiuria or frequency of micturition.

A most constant symptom of myxedema is a lowering of the temperature. This may be due to the decreased or less active combustion of fats and

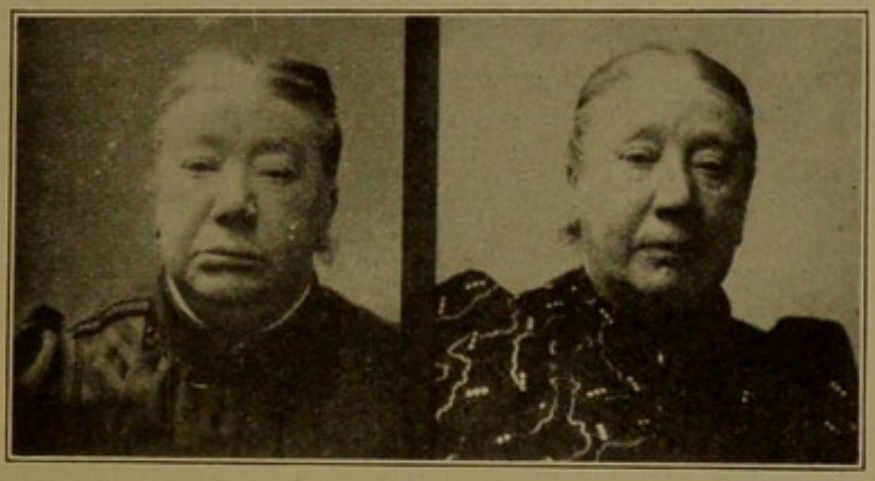


FIG. 5.—Severe myxedema before and after treatment.

mucin, or perhaps to the infiltration of the heat-regulating center in the brain. If one suddenly administers large doses of thyroidin to a myxedematous patient, at the time he has a large quantity of infiltration ready to be oxidized, one may observe a considerable elevation of temperature, which may even be mistaken for fever. This is probably due to the ultra-rapid combustion of these accumulated materials.

The lowering of the body temperature is perceived by the patient. It is subject to diurnal variations, being specially noticeable in the afternoon about 4 or 5 o'clock, in the form of violent shivering, starting with the sensation of cold water thrown on the back. These symptoms may be misinterpreted and treated by the useless administration of quinine. In slight myxedema this feeling of cold is present in a lesser degree. The patient is in a state of habitual chilliness, showing itself in women and children by constant coldness of the hands and feet, and the condition known as "dead

finger"—pallor, stiffness, and insensibility of one or more digits.

Let us consider the effects of the infiltration on a complex mechanism, such as the cardiorespiratory system, where the different elements we have just studied are associated with one another.

We have to remember the paresis of the cardiac muscle, of the external respiratory muscles, and of the diaphragm; the painful infiltration of the nervous ganglia at the base of the heart, added to the disturbances of its central innervation; the infiltration of the pulmonary tissue, the bronchial mucous membrane of the respiratory tract, and consider the consequences.

The dyspnea presented by some patients suffering from severe myxedema may surpass all description. They can with difficulty climb a short flight of stairs, even with a rest by the way; they arrive in the consulting room absolutely breathless and supporting themselves by the furniture, indicating by signs their inability to speak till they have recovered breath.

In mild cases the oppression is less marked. It may be intermittent and presented only on the occasion of unusual exertion, corresponding to an increased demand on the thyroid gland which is weak already. The condition is then readily mistaken for an attack of asthma, and many so-called asthmatics are certainly suffering from thyroid defect. Certain French writers have reported some unexpected cures of asthma by thyroid extract, and while admitting that the patients presented symptoms of thyroid weakness they persist in describing such cases by the term neuroarthritic. That is not logical; we should rejoice to see the word arthritism, which no one understands and which is so often used as a cloak for ignorance, disappear from the medical vocabulary.

Another example: Consider the gastrointestinal system—the muscular walls are slow and lazy, intestinal secretion scarce, therefrom arises constipation. Microbes find their way to the peritoneum and even kidneys; the peritoneum protects itself by false membranes which in time will grow into bands, bringing on kinks and their consequences. In hypothyroid patients these pericolitic adhesions nearly always exist and are even found in very young children, which is not to be wondered at —hypothyroid disposition being nearly always hereditary.

I would be delighted if my supposition concerning the question of intestinal stasis should prove to be correct. It would perhaps bring into a brighter light still the ideas that Sir Arbuthnot Lane in England and Professor Bainbridge in America have promoted and implanted at the price of a most daring and incessant effort.

Sexual System.—The thyroid gland plays an im-

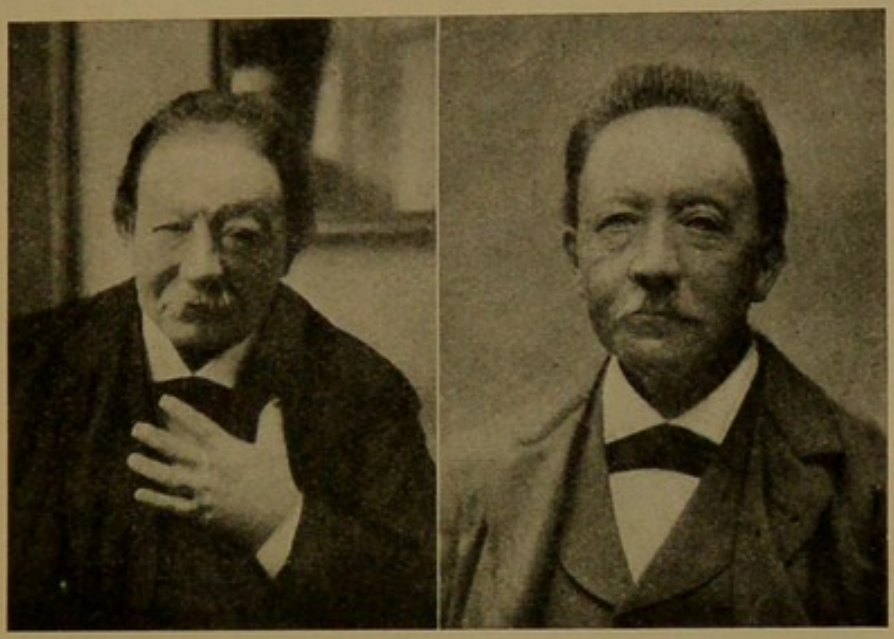


FIG. 6.—Severe myxedema before and after treatment.

portant part in the development and functional activity of the sexual organs, especially in females. It superintends the growth and general development of the sexual organs. Absolute cretins never come to puberty. In mild forms of thyroid insufficiency the uterus remains infantile and small; menstruation begins late in life. Sometimes the posterior wall of the womb does not grow as quickly as the anterior, and therefrom comes retroflexion. This is by no means a rare condition in very young girls. Profuse menstrual bleeding is often seen in such cases and is attributed to retroflexion. As a matter of fact, the excessive bleeding is caused by the infiltration of the uterine mucous lining, by the defective contractility of uterine muscular cells and by the hemophilic condition of the blood. In thyroid defect hemophilia is quite a classic symptom. Profuse oozing may be brought on by almost a mere scratch.

It is a well-recognized fact that thyroid deficiency falls, in nine-tenths of the cases, on females. The reason thereof is easily given: The thyroid has a great influence on menstruation, pregnancy,

lactation, and even uterine involution after childbirth. When the thyroid is normally active the menses are normal; when weak, menorrhagia sets in. The weaker the thyroid the greater the loss of blood. We very often come across these cases of menorrhagia even in very young girls, and we are at a loss to understand the meaning of this distressful condition. We do not know what to do to prevent it. If we can put aside such causes as fibroids and cancer, we will always think of thyroid deficiency.

A large quantity of thyroid stuff is wanted during the menstrual period, and during that time can-

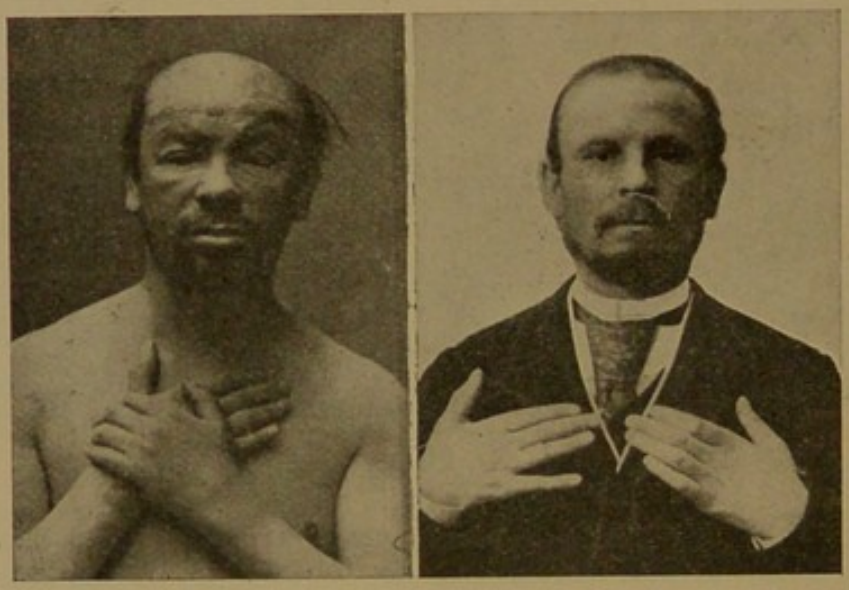


FIG. 7.—Severe myxedema before and after treatment.

not be given to the general keeping up of the body. We often see women who at ordinary times have a decent supply of thyroid secretion run short during the menses, and show then, and then only, the usual signs of thyroid defect. Headaches (migraine) are almost classic symptoms at that time. Many women have muffled voice, which is the result of temporary infiltration of the vocal chords. Extreme lassitude, pains in the back, obstinate constipation, all go along in that line of symptoms.

It is not necessary to dwell on the good effects of thyroid medication under such circumstances. A few doses of thyroid extract will act as a powerful tonic and reduce to a considerable extent the anemic influence of the menses, and reduce considerably the loss of blood.

In pregnancy the thyroid becomes enlarged and throws into the blood an unusually large quantity

of secretion, thereby suspending menstruation and protecting the fertilized ovum against the harm which would result from menstrual activity. This action of the thyroid should be remembered in cases where chronic abortion has exhausted all other forms of medication. We may assert that thyroid extract has proved in scores of cases an excellent remedy for otherwise inexplicable sterility. A great many women who have taken thyroid extract with a view to reduce their obesity have been surprised by becoming pregnant in the course of this medication, and this unexpected result is due to the thyroid inhibition of menstruation. After childbirth the maternal system is suddenly relieved; a large amount of thyroid secretion is still in store. Part of it will be given to the muscular and nervous exertion of labor itself and another part goes to the involution of the uterus. The heavy muscular walls of the womb have to degenerate into fat and be oxidized, and this cannot be done without the interference of the thyroid stimulus.

Finally, lactation claims a good deal of thyroid stuff. It has been proven by experiments on animals that thyroid extract works upon the mammary glands and increases the quantity of milk. Women who have a good lactation have also a quick involution of the womb. After weaning, the thyroid gland is now fairly exhausted. Most of mothers grow fat at that time. Menstruation takes advantage of this deficiency and comes back. Thyroid medication may be useful in such cases where lactation is scanty and where menstruation has a tendency to return before the physiological time. This is indeed the only way we have to increase the mammary secretion.

The mucous lining of the vagina is also infiltrated, and under examination gives the impresson of incipient labor. A good many patients have been curetted for hemorrhagic endometritis who were in reality deficient in their thyroid powers.

I acquainted Professor Bainbridge when he was in Antwerp with these facts. Being a gynecologist as well as a surgeon, he said he would make a careful study of hypothyroid menorrhagia. I am glad to say, and he announced it before the Academy, that he is now convinced that this is absolute truth. He has cured a number of cases already with thyroid feeding, to the astonishment of those who had previously curetted the uterus without reliev-

ing the menorrhagia. Much more might be said about thyroid secretion, indeed volumes have been written on the subject. My friend, Dr. Sajous of Philadelphia, knows about it, and his work on internal secretion has not said all.

The usefulness of thyroid medication is conspicuous in delayed development of the sexual organs, infantile uterus, infantile retroflexion and excessive menstruation. In some cases of sterility and repeated abortion it will prove to be a success-

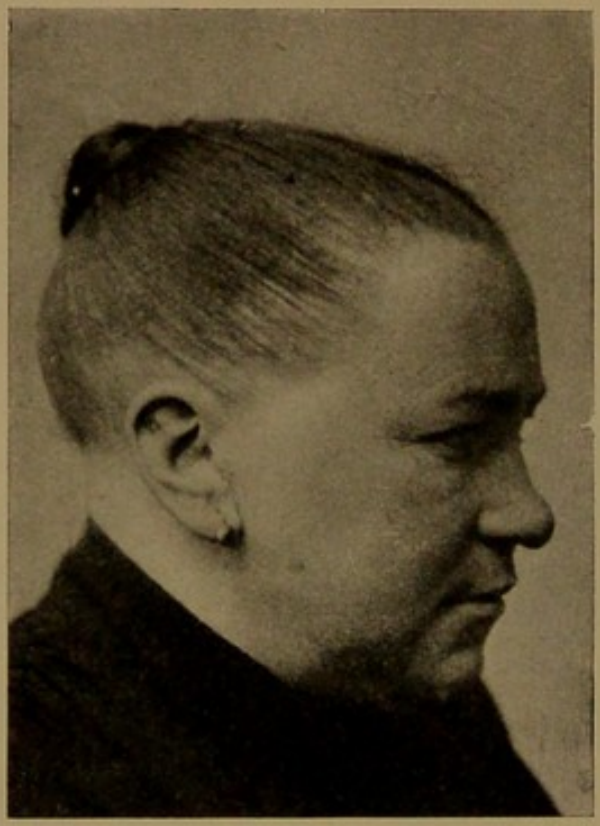


Fig. 8.—Mild myxedema, showing the loss of hair.

ful treatment. Slow involution of the uterus after childbirth and wavering lactation will also find here powerful help.

From what we have stated it is evident that the best way to understand mild myxedema is to have a clear conception of the more severe form. The more severe form, when properly treated, gradually progresses through the milder forms to become finally imperceptible even to the most experienced eye. So also, after successful treatment, a myxedematous patient, left to himself, gradually relapses, the milder symptoms being the first to return, followed by those of increasing severity, so that the disease reconstructs itself under your eyes. These symptoms of the milder form of the

disease are precisely those which should be most clearly impressed upon your memory.

The weakness of the thyroid gland is usually hereditary, and it is rare that one does not find traces of milder defect among the ascending, descending, or collateral relations of a person suffering from well-marked myxedema. One must inquire carefully into the family history; it will be found an inexhaustible source of information regarding symptoms which will render one more

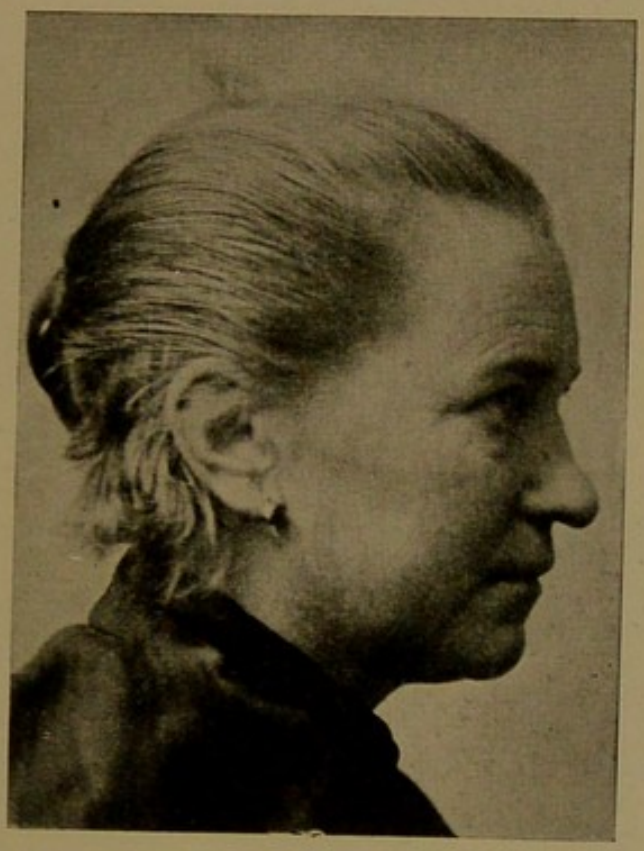


FIG. 9.—The same patient as in Fig. 8 after treatment.

familiar with the milder cases of hypothyroidism.

In doubtful cases treatment by thyroidin forms the touchstone whether for mild or severe myxedema. Many persons suffering from the milder forms do not appear stout or swollen, but may even be quite spare in body, the infiltration in such cases predominating in the internal organs. If under small doses of thyroidin such a person loses weight with simultaneous improvement in his general condition, one may assert the existence of insufficient secretion of the gland.

Let us now study some cases of advanced myxedema, and, to facilitate the task, permit me to indicate the course I myself followed at the beginning of my studies on the subject. I confess that

when I first found myself in the presence of a case of myxedema I knew nothing about it. I had neglected this part of my medical studies, believing I should never encounter what was then considered a pathological rarity. Myxedema, which meant nothing to me in 1883 when I finished my university course, interested me as little ten years later, when circumstances forced me to concern

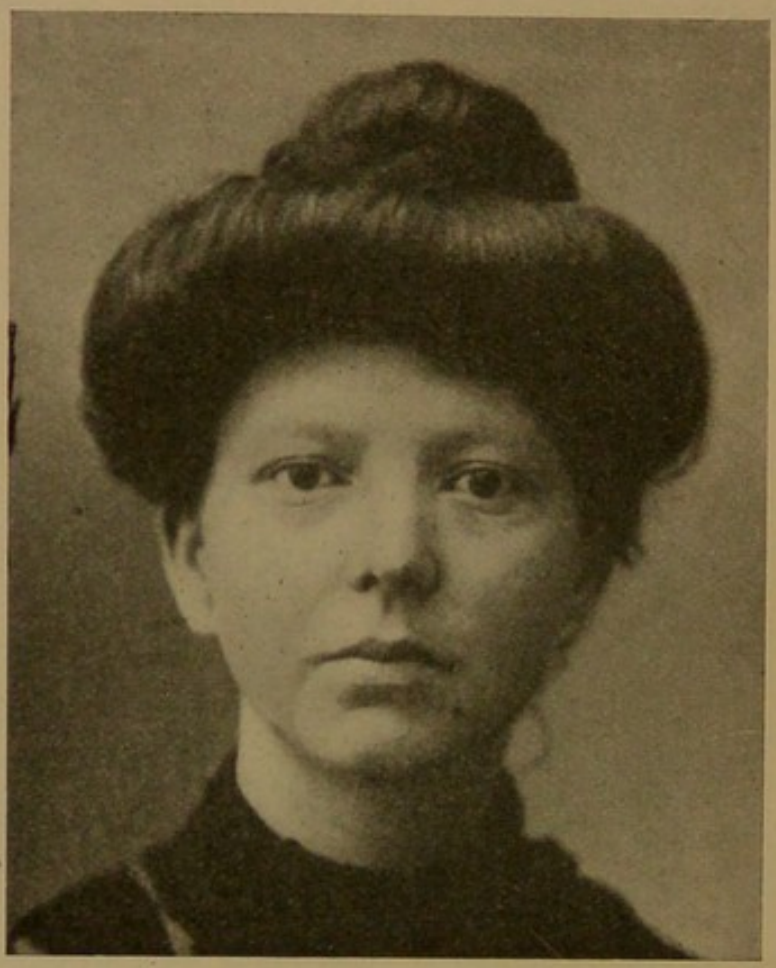


FIG. 10.—Mild myxedema—Loss of eyebrows.

myself with it, and to recognize it without having suspected its existence.

In 1894, while in attendance at the house of M. X., he confided to me that he was much concerned about the health of his wife. He informed me that she was 64 years of age, had been married at the age of 20, and had had no children. She had been delicate all her life, but recently her condition had been so much worse that he feared a fatal issue was rapidly approaching. In spite of this she absolutely refused to consult a doctor. She was indifferent to all that went on around her, this apathy being combined with an invincible disinclination to all physical exertion. Speech was laborious to her, being slow, faulty, and indistinct, so that usually despairing of making herself understood, she cut short all attempts at conversation. Her only

desire was to be left at peace, free from all emotion or anxiety.

Soon after her marriage Mme. X. had a miscarriage. Her menstruation had always been profuse and the intervals between the periods much shortened; the menstrual loss was so great that the blood soaked through the mattress to the floor. She also bled easily after

FIG. 11.—Severe myxedema with loss of hair. Before and after treatment.

FIG. 12.—Profile of the patient shown in Fig. 11.

slight injuries, a simple scratch being followed by prolonged oozing. These repeated hemorrhages had produced a state of pronounced anemia shown by the presence of marked pallor and weakness. The hemorrhages were regarded as the original cause of her condition. At the age of 35, while driving in an open carriage Mme. X. was caught in a heavy shower of rain which soaked her to the skin. Following this she took cold and suffered from a well defined attack of acute articular rheumatism, from which she recovered but had never since been free from pain. From that time her condition was attributed to the rheumatic attack by herself and her friends.

Such was the information given me by the husband, and at his request Mme. X. consented to see me. My diagnosis was instantaneous—it was evident that Mme.

X. was suffering from an attack of Bright's disease in its last stage (Fig. 5). The face was swollen, the eyelids were edematous, leaving only a narrow space between them, the lips were thick, everted, and bluish in color, the speech was slow and drawling, the voice having a strange, deep, croaking tone, while the gait was awkward with slow and indecisive movements. I communicated my opinion to her husband and intimated my fear that a fatal termination was threatening from uremia. M. X. was not surprised and told me that all the doctors previously consulted had been of the same opinion. I then examined the urine and to

FIG. 13.—Severe myxedema; baldness of the nape

my great astonishment it contained only an insignificant quantity of albumin—about 1½ grains to the ounce. There was no renal epithelium present, but squamous cells from the bladder were abundant. I was much embarrassed at finding a case of Bright's disease which had reached the stage of threatened uremia practically without change in the urine. Next day I explained to M. X. that the case was somewhat unusual, and asked his permission to keep the patient under observation for ten days in order to make a further examination, to which he willingly agreed.

I could then examine the patient at leisure. The heart presented no appreciable change, though the pulse was slow. The kidney region was painless on palpation so far as it was possible to examine it, for Mme. X., while not obese, was short and stout, weighing 204 lb. Her lower limbs did not appear notably swollen and though large were not out of proportion to her

body. Her temperature was rather low and she complained of cold although it was the end of May and a large fire made the room uncomfortably warm. The patient told me that she always felt the cold most intensely about 4 o'clock in the afternoon, at which time she felt as if cold water had been poured on her back, causing her actually to shiver. She soon became tired during my examination and even fell asleep while I talked to her, complaining of extreme lassitude and rheumatic pains throughout the whole of her body.

Having discovered all this I had advanced no further than before. That a woman 64 years of age should be

FIG. 14.—Same patient as in Fig. 13 after treatment.

sleepy, chilly, easily tired and suffer from rheumatism was neither very extraordinary nor was it pathognomonic. Five days had passed, when chance,—the Providence of doctors,—came to my assistance. M. X. informed me that he was expecting an early visit from his wife's cousin who suffered from a very troublesome goiter which at times threatened to suffocate her. He also told me that I might be consulted by her and, should alarming symptoms occur, I might even be asked to remove the tumor. Now goiter is a rarity in Antwerp and for my part I had never seen a single case and was rather worried at the prospect.

My situation was truly awkward, the old lady apparently had Bright's disease but without albuminuria and seemed to be at the point of death without presenting a single positive symptom which I could lay hold of;—and then this cousin must needs come from the other end of Austria possessed by the desire to be

Fig. 15.—Severe myxedema.

operated upon for a tumor which she would a hundred times better have had removed in her own country, where they are accustomed to similar exploits.

I returned home in a state of great anxiety and felt a real relief on finding on my table a large parcel containing the treatise on surgery in 8 volumes by Duplay and Reclus which had just been published. I turned to the article on goiter by Broca. Oh, yes! goiter could certainly be removed: it was not easy, but with de-

Fig. 16.—Same patient as in Fig. 15 after treatment.

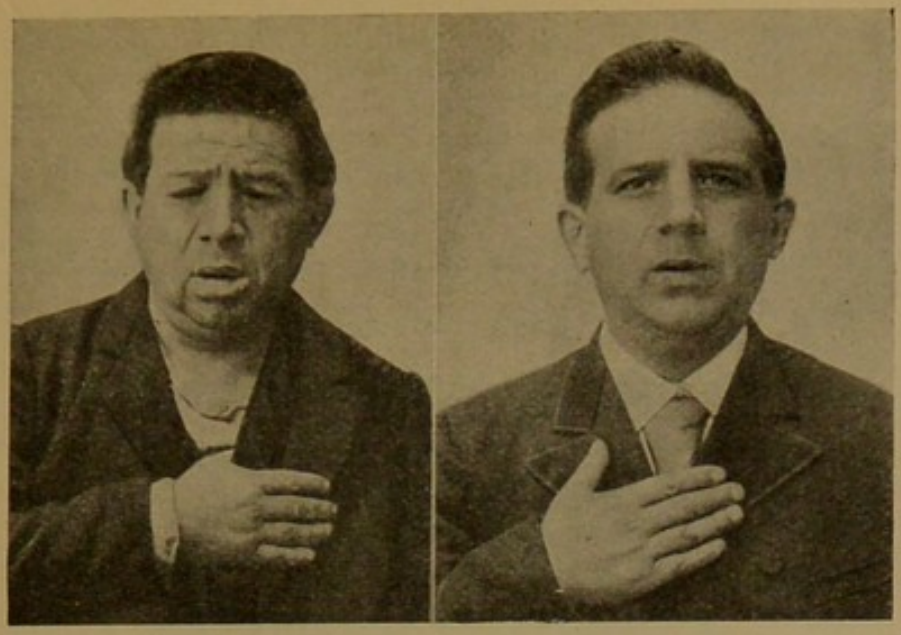


FIG. 17.—Severe myxedema, before and after treatment.

termination and a score of artery forceps it could be done. Then there were complications—one must not divide the recurrent laryngeal—must guard against hemorrhage and sepsis. And this was not all,—one must beware of removing the whole of the thyroid gland—for this was followed after a short interval by a special cachexia which Kocher had named "Cachexia strumipriva" and Reverdin had called "Post-operative myxedema." This was becoming more and more interesting.

Thus when the patient had escaped from the dangers of the operation, hemorrhage, and sepsis, just when we might expect him to enter upon convalescence, a very strange condition gradually disclosed itself, characterized by the following symptoms:—*lassitude, feebleness, clumsiness, heaviness of the limbs, pain in the arms, legs, neck, and shoulders, swelling of the*

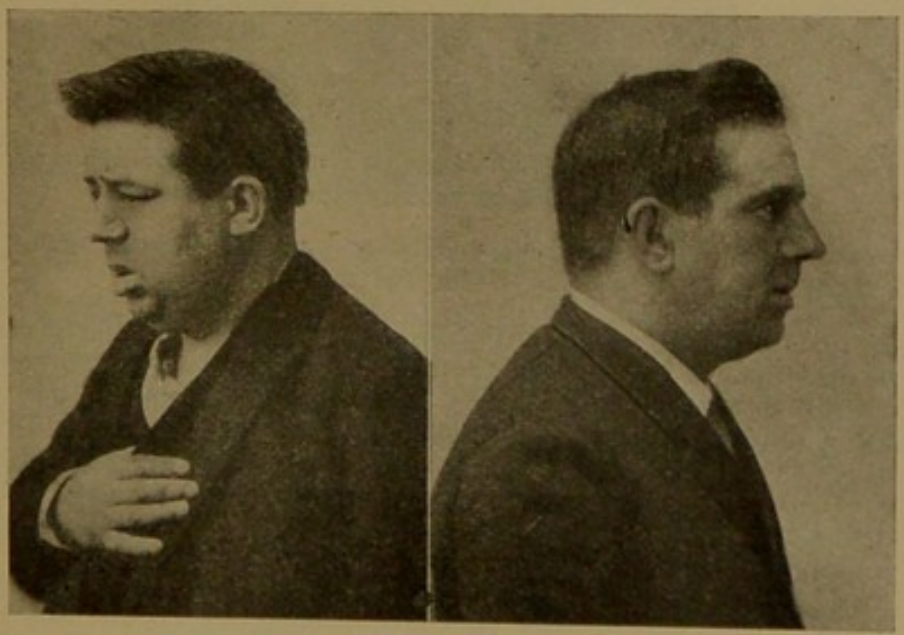


FIG. 18.—Profile view of the same patient shown in Fig. 17.

face and puffiness of the eyelids. The eyes become sunken, the activity of the brain diminished and mental effort dulled. Then there came an extraordinary phase: *"The urine is almost always normal, to the astonishment of the early observers who expected from the appearance of the face to discover albumin.* And again another phrase:—*one of the most remarkable phenomena is a sensation of cold, which is almost constant.*

Truly Mme. X. presented a complete picture of these

FIG. 19.—Severe myxedema with predominance of rheumatoid pains before treatment.

symptoms. She had the false suggestion of albuminuria without the presence of albumin, and the general swelling of the body. Like the patients operated upon by Kocher, Reverdin, and Bruns, she had the drawling voice, the sluggish attitude of body, the thinned hair and eyebrows, the swollen mucous membranes, and the difficulty in swallowing. Her tongue appeared too large for her mouth, the floor of which was swollen and raised till it suggested a double ranula. Even her ocular conjunctiva was edematous and prolapsed while her complexion was amber-yellow with patches of red on the cheeks. She had also the low temperature with

subjective sensations of cold—but indeed Mme. X. presented all the symptoms described in post-operative myxedema and if so she must suffer from spontaneous myxedema.

At last my diagnosis was made. On the following day I verified the presence of the hard, nondepressible edema extending over the whole body and some other symptoms which I had not previously recognized. I then informed her husband of what I had discovered and that I was prepared to commence treatment at

Fig. 20.—Same patient as shown in Fig. 19, after treatment.

once. M. X. was too polite to say that he did not believe me, though his face plainly showed his incredulity, but he followed my instructions to the letter.

The result exceeded my hopes. After three weeks treatment the bodily and mental transformation was so complete that she would no longer have been recognized as the same woman (Fig. 5). The edema of the tongue, of the lips, and of the eyelids disappeared as if by enchantment and the face assumed an intelligent expression. The patient then went to the country where the treatment was continued by the local doctor, who gave me valuable assistance. I did not see her

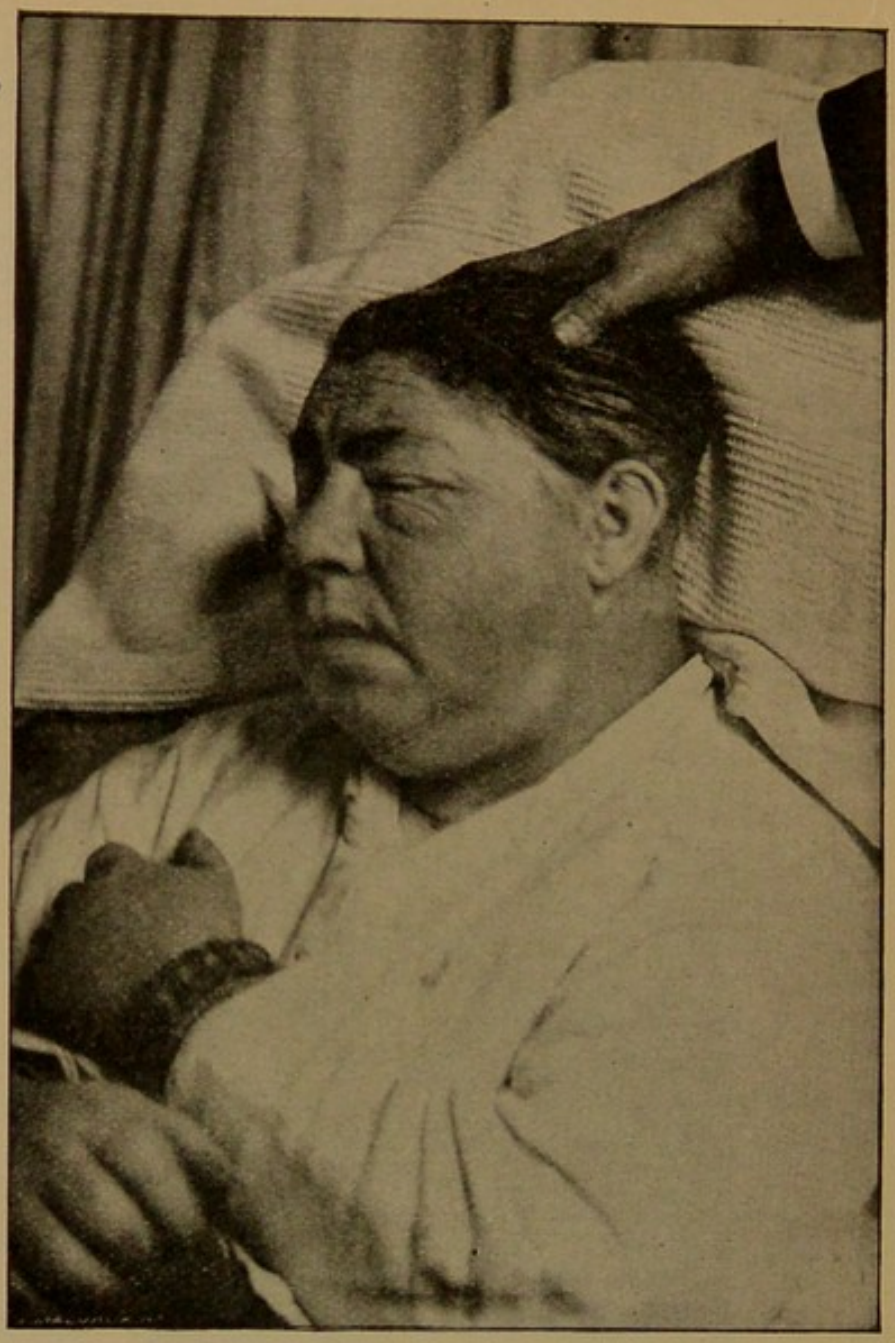


FIG. 21.—Severe myxedema, cachectic stage, three days before death.

again for over two months by which time she had completely recovered, that is to say the absorption of the infiltration had been complete.

If you now ask me what we have to learn from this observation as regards mild myxedema, and on what point it helps our knowledge of this condition, I would first direct your attention to the metrorrhagia which is presented by the history of the patient. As a rule thyroid weakness shows itself by the presence of metrorrhagia which is sometimes appalling in its amount. The administration of thyroidin moderates these losses, and if large doses are given one may even completely

suppress menstruation, as in the same way complete amenorrhea is not infrequently present in cases of exophthalmic goiter. Women with feeble thyroids conceive readily, but abort as readily in consequence of the onset of profuse bleeding which carries away the fertilized ovum. I do not mean to say that a woman suffering from thyroid weakness cannot go to full term, as from the beginning of pregnancy the thyroid gland undergoes hypertrophy with increase in size and in the amount of its secretion. In fortunate cases this increased activity is maintained throughout pregnancy and forms an effectual protection to the embryo against the menstrual return. Such women tell you that their health is better when pregnant. The increased activity of the gland continues during lactation, and such patients instinctively prolong suckling beyond the physiological period. After weaning, the symptoms of thyroid weakness reappear and certain authors have even discovered in prolonged lactation a cause of myxedema. *Thus when you encounter cases of profuse menstruation in which you can exclude such ordinary causes as fibroids, cancer, or placental remains,* think of possible thyroid defect and search for other symptoms of this condition.

Think of it also in those disheartening cases of repeated abortion, in which the administration of thyroidin will often permit a pregnancy to go to term when all other rational means of treatment have failed.

I would in the second place direct your attention to the rheumatic pains from which this patient suffered. In almost all cases of severe myxedema one finds that the patient at some period of life has passed through an attack of acute articular rheumatism. This affection can itself cause grave disturbances of the thyroid gland, the congestion of which in the course of acute articular rheumatism is a classic symptom. I believe, however, that its influence is usually limited to producing an aggravation of a pre-existing thyroid weakness, thus bringing to light symptoms hitherto unnoticed. The metrorrhagia from which Mme. X. had suffered before the occurrence of her acute rheumatic attack supports this view. The chronic rheumatic or rheumatoid condition is almost always associated with mild myxedema, and its occurrence should be carefully inquired into, especially when metrorrhagia is also present. These pains, which are

often complicated by neuralgia, tend to assume a characteristic form and course, to which I will now refer. The most frequent rheumatoid pain experienced in mild myxedema is that affecting the back between the shoulder blades, and is most severe in the morning on rising, after the chilling and inanition of the night. Driven from bed by the pain, these patients rise absolutely worn out, as if they had slept on a hard uneven mattress. The pains subside gradually during the day, owing to the warmth produced by food and exercise, and disappear completely in the evening after a good meal with plenty of wine. Such patients are strongly attracted to the use of alcoholic stimulants.

I have already stated my opinion as to the lowered temperature and the subjective feeling of cold, so I need not insist further on this point. The hard, cold hand of a patient suffering from severe myxedema is very characteristic, and in the milder forms coldness of the extremities is usually present, though to a less degree, as I have already stated.

Among the symptoms presented by Mme. X., one of the more interesting was the special character of the voice and speech. The voice was deep, rough, and croaking, with an indescribable quality which when once heard could not be forgotten. This symptom is caused by the infiltration of the vocal chords and the pharyngeal mucous membrane, and is present in a less marked degree in milder forms of hypothyroidism. In women with feeble thyroids the voice is slightly hollow or muffled. Sometimes this is only occasionally present, as during menstruation, when a considerable part of the thyroid resources is employed in the inhibition of the menstrual function. We will not consider further the case of Mme. X., though interesting from the standpoint of our subject, but will bear in mind as leading symptoms the *metrorrhagia*, the *recurring abortions*, the *rheumatoid pains*, the *feeling of chilliness, and the alteration of the voice.*

After my attendance on Mme. X., I reproached myself for having so long neglected to inform myself as to the diseased conditions of the thyroid gland. During my ten years of practice I must have already met with these cases of apparent Bright's disease; I had treated them, and they had disappeared and been forgotten. Surely I had heard somewhere a voice similar to that of Mme. X., but when? and where? On racking my brains, I remembered that one day I had been summoned as an expert before

FIG. 22.—Myxedema, severe form, before and after treatment.

the Civil Pensions Commission, the administrative body to which the servants of the State apply when on account of infirmity they wish to claim their pension before the retiring age or when it is necessary to retire them. It decides in accordance with the advice of the medical experts.

A professor of solfeggio in the Antwerp Conservatoire had appeared before us. His condition was so lamentable, so profoundly cachectic, that my colleague and I requested the President to send some one home with him lest he should fall in the street. The strange, hoarse, croaking voice of this man whose occupation was the teaching of singing to children, was so irresistibly comic that commissioners and doctors alike had great difficulty in keeping their gravity. The medical examination did not take long and the verdict was explicit—Bright's disease in its last stage with uremia, and he was at once retired on pension.

I searched for and found the singing master who was still alive, and whom I now recognized to be suffering from myxedema. I show here his photograph before and after treatment (Fig. 6). I informed the Minister of the error, who sharply rebuked the Commission for a mistake made through no fault of theirs. The board then had the patient examined afresh by other doctors, who discovered nothing wrong with him as he no longer presented the least sign of myxedema. The proposal to retire him was withdrawn, and his voice having now recovered, he was able to resume his class with a brilliancy to which he had been long a stranger. He was at this time 55 years of age. Before treatment was begun his whole face was swollen, especially the eyelids, the space between which was reduced to a mere slit. He also suffered from rheumatic pains, a constant feeling of cold, and continual dyspnea. The skin was dry, thin, and scaly, the epidermis covered with fine lozenge-shaped wrinkles. The whole body was heavy, clumsy, and infiltrated with a firm resistant edema. He complained of invincible somnolence, depression, and weariness of life. The infiltration was rapidly absorbed under treatment, his weight falling from 169 lb. to 136 lb. in two months, at the end of which period the second photograph was taken.

Here for the first time dyspnea appears as a leading symptom of myxedema. The patient had suffered from it for a long time, and in my former notes of his condition I found it occupied an important place. His walk was slow and difficult, and he required to support himself by the furniture, making signs that he could not speak for want of breath. In mild myxedema this breathlessness is constantly present, though to a less degree, only showing itself on walking rapidly or uphill, and is usually accompanied by palpitation. Sometimes the breathlessness is intermittent, like the insufficiency of the thyroid secretion on which it depends, and is then liable to be mistaken for an attack of asthma.

The gratitude of this singing master was all the greater that he had been restored to his duties and escaped the misery of a premature pension. He told me that one of his friends, a police agent, who had been put upon the retired list ten years before, suffered from the same condition as himself. This was rather too much—to have myxedema diagnosed by a patient who had only just recovered from the disease.

I called on the police agent whose appearance before and after treatment is shown in the photographs (Fig. 7). He was suffering from very advanced myxedema and though only 42 he appeared much older. The photograph shows the degree of cachexia at which he had arrived. His whole face was infiltrated, the eye-

FIG. 23.—The patient shown in Fig. 2 after 14 years of treatment.

lids being so swollen that the eyes could be opened only with the greatest difficulty, while the thickened lips resembled those of a negro. The complexion was amber yellow with bright red patches on the cheeks. The trophic changes in the hair were striking, the forehead presented a band of brown pigmentation and there were similar marks on each side of the neck. The head was too heavy for the infiltrated muscles and ligaments of the neck so that it fell forward and the patient could raise it only by throwing the trunk backwards. He complained of rheumatism and a perpetual feeling of cold, huddling himself night and day under thick bedclothes. The breath was offensive, the teeth bad, the gums red and inflamed. Speech was much impaired. On replying to a question he opened his mouth widely, so that the motions of his tongue could be plainly seen although he uttered no sound, and it was only after a lengthy effort that the words were slowly formed. His weight was 152 lb. of which he

lost 22 lb. under treatment with the simultaneous improvement of all his symptoms. The loss of weight in this case amounted to 18, 28, and even 35 ounces a day. His hair grew again rapidly, muscular contractility was regained so that he could again raise his head, and he was delighted to find his self-confidence return, complete recovery being attained in about two months.

This case directs our attention to the trophic changes of the hair, teeth, and gums. In the milder type of myxedema the hair may also be shed early, though more frequently it becomes prematurely grey. The destruction of the teeth and the chronic alteration of the gums is also observed, though to a less extent.

FIG. 24.—Mild myxedema in the mother of the patient pictured in Figs. 2 and 23.

Whatever the type of myxedema, the baldness very constantly presents a special distribution, the hair being first shed in the frontal region, then on the nape of the neck, giving the appearance which is well shown in Figs 8 and 9. The loss of the hair in the eyebrows is early and constant, even in mild myxedema, and has been called the eyebrow sign. It is not so easily concealed as the baldness of the scalp, and it gives to the patient's face an air of perpetual astonishment.

Fig. 10 is the portrait of a woman aged 24 who had suffered from uterine hemorrhage since her confinement, four months previously, for which she had been packed and curetted several times. The absence of the eyebrows is striking, and though the frontal baldness had been artfully concealed, she at once suggested to me a case of insufficient thyroid secretion. She

suffered much from migraine and also from occipital headaches; she was always cold, while her menses were always very profuse. Under thyroid treatment the hemorrhage ceased in a fortnight.

A very interesting case of myxedema is shown in figures 11 and 12. There was marked loss of hair which in addition to the usual situations had affected the sagittal line, but the baldness was concealed by the presence of black crusts which covered the whole scalp, the eyebrows were also markedly affected. This patient might be classed as an advanced case of myxedema and the result of treatment was very striking. She had always been tired, constipated, and somnolent. She was never free from rheumatic pains and became breathless on the slighest exertion. Her face was infiltrated, the eyelids and lips being specially swollen. Under thyroid extract she rapidly lost 22 lb. in weight and after four months treatment, was completely changed both morally and physically, having regained

Fig. 25.—Mild myxedema, before and after treatment.

courage, strength, and cheerfulness. She even became coquettish and would not believe that her appearance had been as in the first photograph.

The patient shown in Figs. 13 and 14 is a good example of the effects of thyroid weakness on the hair and eyebrows. She was only 39 years of age, but appeared much older. The face was swollen, amber yellow in color, with patches of red on the cheeks. The trophic changes had chiefly affected the hair and teeth, the nape of the neck being almost bald. She complained of a constant sensation of cold, obstinate constipation, and pains all over the body, which she attributed to rheumatism. She presented also the drawling voice with its characteristic intonation. Her weight was 154 lb., of which she lost 22 lb. in the course of treatment.

The patient presented in Figs. 15 and 16 was 42 years of age. Fourteen years before, as the result of a chill, she suffered from swelling of the hands and feet with albuminuria. She was treated for Bright's disease, but did not recover her health, dragging on in a weak, exhausted, somnolent condition, with pains in all her limbs. Becoming pregnant she suddenly im-

proved considerably, due to the stimulation of the thyroid gland by her condition. This improvement lasted throughout pregnancy and was maintained during lactation, although she nursed her infant for two years. After weaning him she gradually relapsed into her previous condition, again becoming apathetic and depressed while the rheumatic pains returned with increased severity. The eyebrows were very thin, the features swollen, while the pale yellow complexion recalled that usually associated with albuminuria. She suffered from constipation and headache, her teeth were decayed, and she presented attacks of shivering during the evening which were attributed to fever.

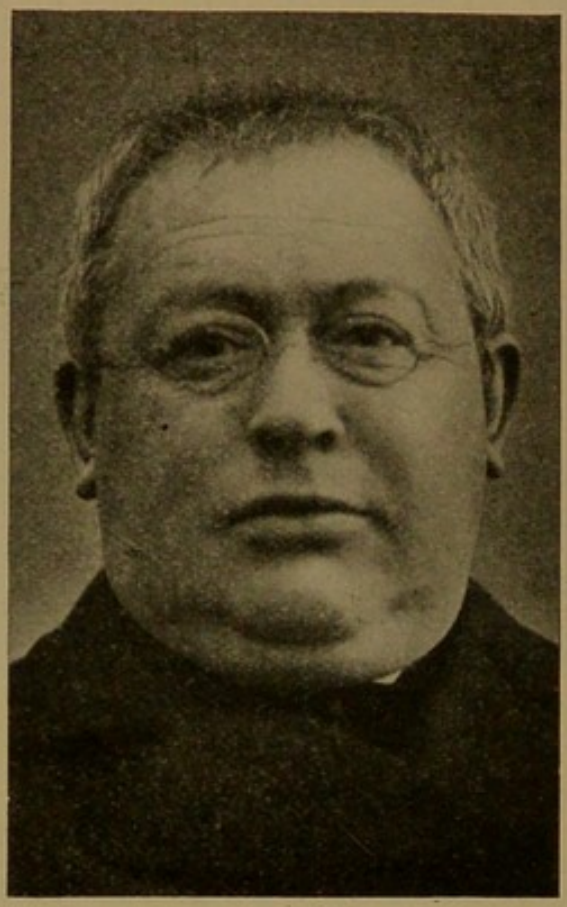


FIG. 26.—Severe myxedema before treatment.

All these symptoms improved under treatment, her weight at the same time falling from 143 to 138 lb.

The patient shown in Figs. 17 and 18 was only 39 years of age. He was a cigar maker, who had been unable to work for six years, owing to his fingers having become stiff and clumsy. He had taken to drink and he and his family had fallen into extreme poverty. He was positively ugly, and the boys of the district, who knew him under the name of Ravachol, followed him on the street. I literally picked him out of the gutter and kept him under observation for a week before commencing treatment. His appearance is well shown in the illustrations. He complained of pain in the spine, a constant feeling of cold and insuperable fatigue, and was melancholic, depressed, and extremely miserable. His hair was unaffected, except

in the occipital region, which was bald. During the week before treatment was commenced he excreted on an average 323.4 grains of urea per day, which increased under thyroidin to an average of 477.4 grains per day, during the first week, with a further increase during the second week to 677.6 grains per day. In consequence of the absorption of the infiltration his weight fell from 167 lb. to 138 lb. His urine contained an extraordinary number of spermatozoa, this symptom persisting for a whole month, when it suddenly ceased. He rapidly recovered as shown by the illustration, and he not only regained his health but also his self-confidence, so that he resumed his work, at which

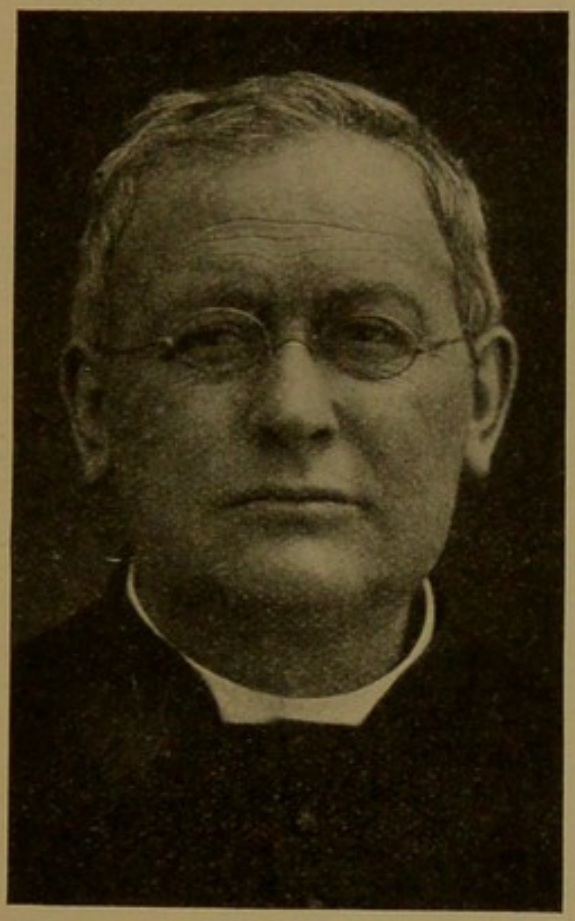


FIG. 27.—The same patient as in Fig. 26, after two months' treatment.

he was expert, and after some months sailed for America in quest of a better situation. He obtained work at once, and is now himself an employer, and has made money. Every two or three years he returns to Europe to express his gratitude to me.

Soon after the cure of the singing master and the police agent I remembered that at the beginning of my career I had treated a woman of whose peculiar voice I was reminded by that of these two patients. She had left town, but at last I discovered her. In those days she had suffered from rheumatism involving all the body, the muscles being stiff, hard, and painful. The joints were also swollen, the gait

stiff and awkward, while the pains in the back were very severe. I had formerly treated her by every means I could think of, without benefit. On her reappearance I found that she was really suffering from myxedema—as you will see by the photographs before and after treatment (Figs. 19 and 20). The puffiness of the face, the swelling of the lips, especially the lower one, the loss of the hair of the scalp and eyebrows, the redness of the cheeks on a yellow skin, the drawling, croaking voice, the dry wrinkled skin, and the desquamation of the scalp

FIG. 28.—Severe myxedema.

left no doubt as to the diagnosis. Under thyroid treatment the infiltration rapidly disappeared. The general pain and stiffness also disappeared steadily, though slowly, along with marked breathlessness, to which she had always been subject, and which I had formerly attributed partly to obesity and partly to pulmonary emphysema. Finally all these symptoms were completely abolished, but on the patient ceasing treatment the pains gradually returned, to cease again on the administration of thyroidin. This woman had suffered from these rheumatic symptoms for so many years that she had become resigned to them, and sought advice only when the

pain in the back or the swelling in the wrists or knees became unusually severe. *The pain was then, as later, simply a manifestation of defective thyroid secretion.*

Let me now record a case illustrating the disorders of the liver associated with defect in the thyroid gland. The patient shown in Fig. 21 was a women suffering from myxedema in its last stage. She died three days after admission to my ward without it being possible to begin thyroid treatment. On post-mortem examination the gall-bladder was

Fig. 29.—The same patient as in Fig. 28, after treatment.

found much enlarged, distended to the bursting point, with marked thinning at its upper pole. It contained a large gallstone, which permitted the entrance but not the escape of the bile. I was much impressed by this observation, and later in cases of myxedema, whether mild or severe, I examined for tenderness of the liver and especially of the gall-bladder. Very frequently, not to say always, a painful point was found in this region, which disappeared during treatment. Although the presence of a calculus in the gall-bladder is a common occurrence in the post-mortem room, I have dwelt on it and shown this photograph in order to emphasize

the frequent presence of disorders of the liver in myxedema, of whatever type. In this affection one should always think of congestion of the liver and the possible presence of calculi or biliary sand in the gall-bladder. The amber yellow coloration of the skin, so characteristic of myxedema, is only an attenuated jaundice and depends on biliary disturbances.

Fig. 22 is that of a woman 40 years of age, suffering from well-marked myxedema. Her father, who was dead, had suffered from rheumatism and al-

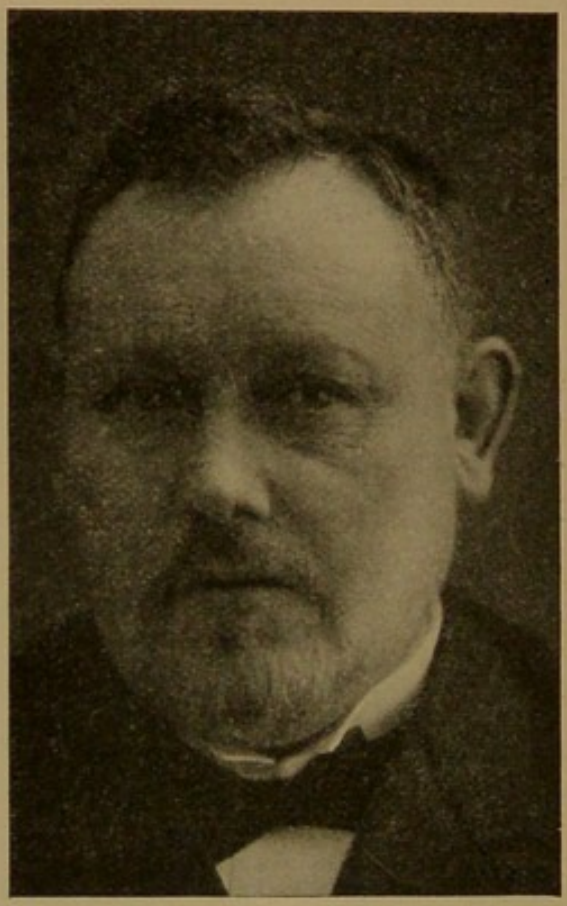


FIG. 30.—Mild myxedema with atrophy of the optic nerves.

buminuria. She had six children; the eldest daughter was subject to metrorrhagia, while two of the sons had suffered very severely from acute articular rheumatism. Her appetite had completely gone, she suffered from menorrhagia, with headaches and frequent attacks of shivering. Her temperature taken in the mouth was only 96.2° F. There was great swelling of the face, but the hair was well preserved and the teeth in good condition. She suffered from melancholia and apathy with difficulty of thought, speech, and action. After six weeks' treatment at the Institute she was discharged so changed in appearance that she would not have been recognized. Since then she had continued her treatment, but only very irregularly. When she neglected it too long the first symptoms of relapse were a feeling of weight at the stomach, diffi-

culty in stooping, tenderness over the liver, and a distaste for meat. After an unusually long period of neglected treatment she sent for me in haste one night and was found suffering from biliary colic. The urgent symptoms yielded to oil and morphine and later under thyroidin she made a complete recovery.

I have said that an excellent means of becoming acquainted with the slighter degrees of myxedema is to study the morbid characteristics of the ascending, descending, or collateral relations of patients suffering from the more severe type, the weakness of the thyroid gland being essentially hereditary.

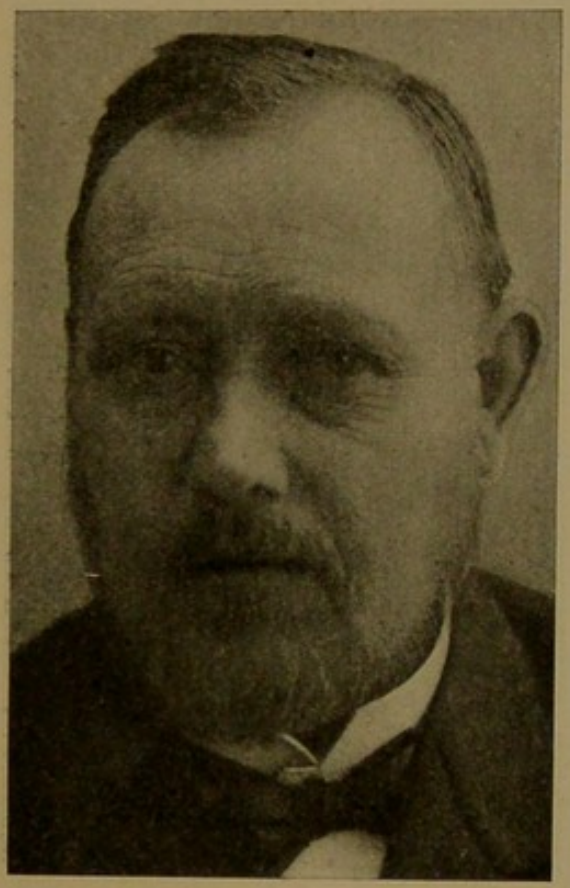


FIG. 31.—Same patient as in Fig. 30, after treatment.

I have shown in Fig. 2 four photographs of a child who suffered from severe myxedema, and presented marked delay in her physical and intellectual development. When first brought to me in November, 1896, she was 8 years of age, and she measured only 2 ft. 10 in. in height, instead of 3 ft. 10 in., the normal average for her age, though she was comparatively heavy, her weight being 33 pounds. The expression of the face was that of well-marked myxedema; the face itself was swollen with the characteristic red cheeks on an amber yellow skin.

The dystrophy had not involved all the tissues to the same extent, those of epidermic origin being relatively intact. The hair was black and thick, the

eyebrows were well marked, while the teeth were healthy, an exceptional event in myxedema. The bones, on the other hand, were severely affected, the femora were curved, the tibiæ and fibulæ showed advanced signs of rickets, and the feet were short and flat. The belly was large—a characteristic of this affection—and presented an umbilical hernia. The false ribs had been carried outwards by the distention of the abdomen—a change more apparent in the second photograph, where the belly has been

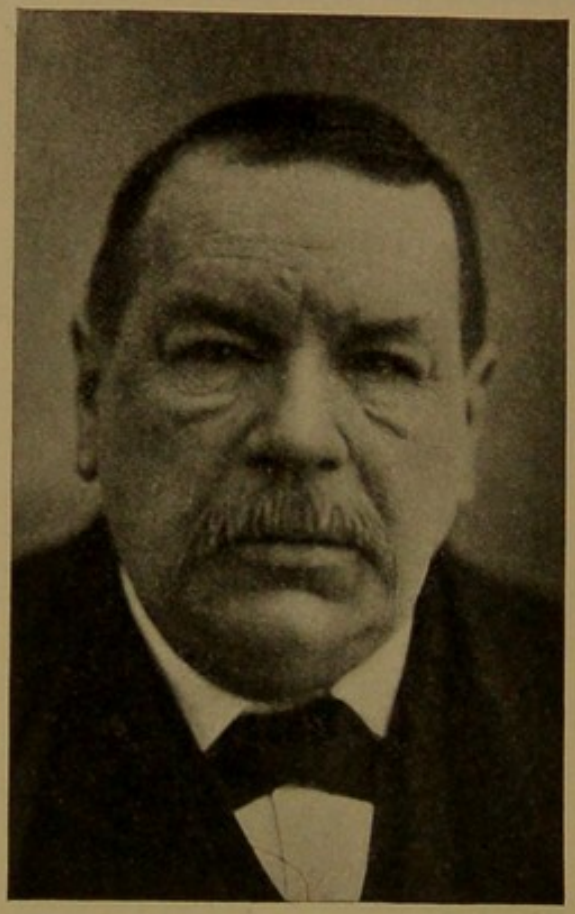


Fig. 32.—M. A., severe myxedema; before treatment.

reduced by a year's treatment. Her intelligence was but slightly developed. Her temperature was very low, even in summer, and in winter it became extremely so, there being great difficulty in keeping her warm. She throve badly, and was extremely constipated. There could be no doubt as to the cause of the condition—a congenital weakness of the thyroid gland.

The patient's mother had suffered from several severe attacks of malarial fever in the course of her pregnancy, and had been obliged to leave the low-lying village where she lived and come into town for her confinement. Four years previously, before she lived in this low-lying district, she had borne a

son, now 12 years of age, who was slender and delicate. A second child had died of some abdominal condition at the age of five months, the third being the patient with whom we are now concerned.

I have often observed that for the production of a severe case of congenital myxedema the association of at least two grave defects in the parents is necessary, as, for example, the coexistence of syphilis and tuberculosis; gout or diabetes, complicated with tuberculosis or syphilis; or, again, the

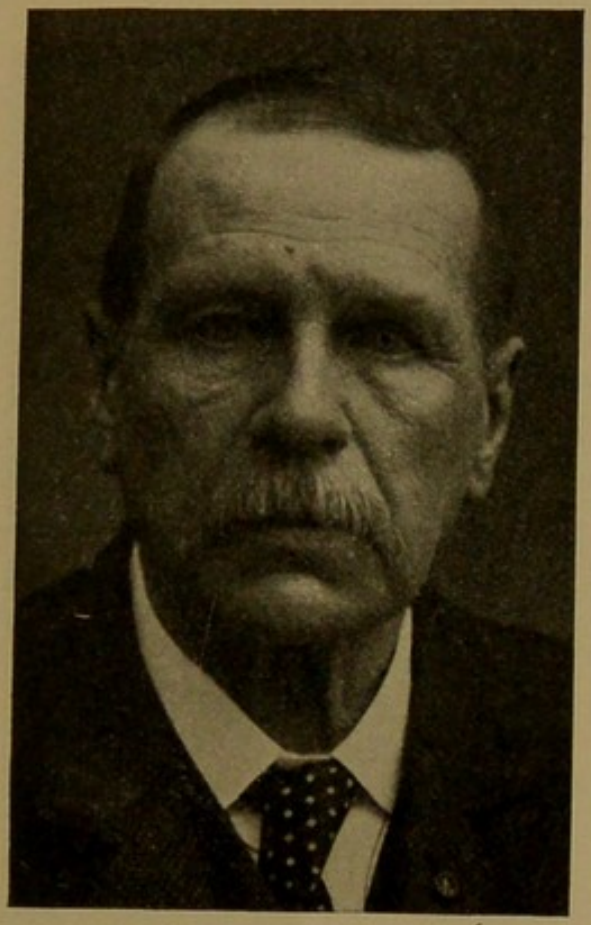


Fig. 33.—M. A. after two months' treatment

coincidence of alcoholism and syphilis. The mother was therefore carefully examined to discover the defect which, joined to malaria, had produced such a complete degeneration in the fetus. She suffered from severe migraine, was very constipated, her liver and gall-bladder were tender, and her menstrual loss was excessive. She also complained of transient rheumatoid pains in her muscles, which became aggravated during the cold season. She was dull, depressed, almost melancholic, and presented a low temperature, frequent breathlessness and palpitation. Her complexion was pale yellow, like that of the child. Vertigo, tinnitus, and muscæ volitantes were also present.

The clinical picture was that of the simpler forms of thyroid defect. Without being myxedematous she evidently suffered from thyroid weakness. The malarial poison had done the rest, and the mother, unable to furnish the fetus with the necessary amount of thyroid secretion, gave birth to a cretin.

The little patient quickly responded to treatment by thyroidin. After a year her appearance had much improved, as seen in the second photograph (Fig. 2*b*). At the beginning of treatment the thyroid extract appeared to disagree with the

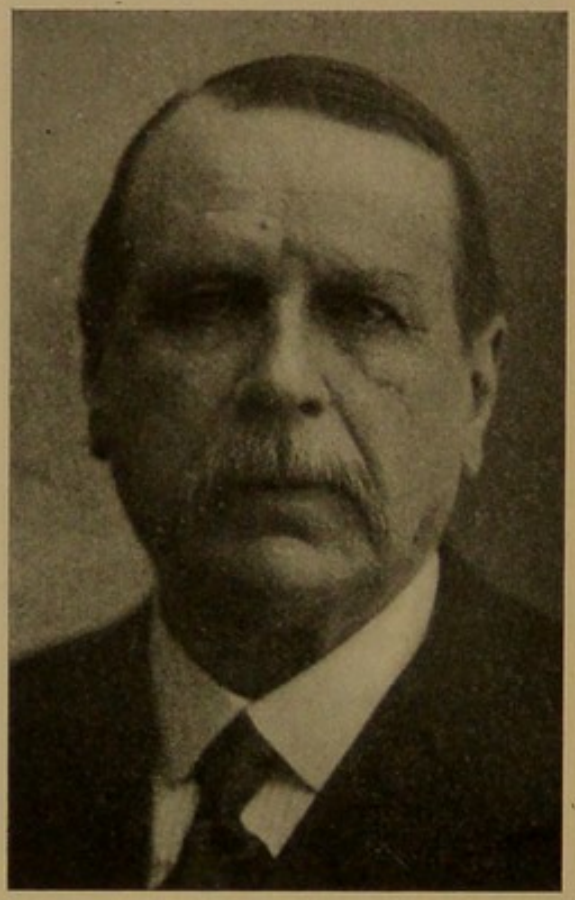


Fig. 34.—M. A. after 14 months' treatment.

patient, causing vomiting in the morning, before breakfast, but in spite of this its administration was continued. During the first year she gained 5½ inches in height, while the rachitic deformities greatly improved but did not entirely disappear. The distended abdomen assumed more natural proportions, the umbilical ring closed, and the neck became more slender. The expression of her face became more inquiring and thoughtful. Her progress during the second year was less satisfactory, as she unfortunately suffered severely from whooping-cough, which lasted five months. The increase in height amounted to only 2¼ in., but all traces

of rickets had disappeared from the limbs. The third photograph (*c*) shows her appearance at the end of the second year.

The action of infectious diseases on the activity of the thyroid gland has been well known since the work of Marcel Garnier.[5] He states in connection with a case of whooping-cough which proved fatal by bronchopneumonia: "The thyroid gland was completely transformed. (on post-mortem examination). The coloring matter was almost entirely absent; the vesicles were empty; the thyroid cells,

FIG. 35.—Sister of M. A.—Myxedema.

more or less raised towards the interior of the cavity, did not fill it, and there was no cellular proliferation, so that the thyroid tissue appeared like a fine network between the meshes of the connective tissue, which were closer than usual. There was in this case an arrest of the colloid secretion, a state of *athyroidism.*" Garnier attributes these lesions to the bronchopneumonia rather than to the whooping-cough, but we may remark that whooping-cough is almost constantly complicated by bronchopneumonia, and it certainly was so in the case of our little patient. One is not then astonished at the slight progress made during the second year,

and it is worthy of note that whooping-cough showed itself able to lower the secretory activity, even in this child, apparently destitute of an active thyroid gland. I believe that however degenerate it may be, the thyroid gland, even in the most confirmed cretinism, never loses entirely its secretory power, as the complete absence of thyroid secretion from the blood causes death in a short time.

In the course of the second year the parents became much alarmed at the appearance of a defect in speech, which they attributed to the treatment. I have seen several similar cases in analogous circumstances. The child had great difficulty in articulation, the first syllable being specially hard to utter, so that she panted, gasped, and twisted herself about. Soon, however, this disturbance ceased spontaneously. During the third year the patient gained 2½ inches in height.

The progress in the fourth year was also unsatisfactory, the treatment being irregularly followed and even interrupted for some months. There was, however, a gain of 1½ inches. After a serious admonition the parents became more attentive, and in the course of the fifth year the child's growth showed a distinct improvement, the increase in height amounting to 3½ inches. The fourth photograph (Fig. 2*d*) shows the patient as she appeared after five completed years of thyroid treatment. During that period she had gained 15½ inches in height, her lower limbs were straight and well formed, and the most trained eye could no longer detect the slightest indication of thyroid defect.

This case confirms the ideas which we have long expressed on the etiological unity of several morbid states which have been attributed to very diverse causes. Such conditions as infantile obesity, rickets, the slender type of infantilism, anangioplasia, chondrodystrophy, myxedema, the arrested growth in congenital syphilis, the arrest and delay of growth associated with alcoholism, tobacco poisoning, malaria, and tuberculosis, are all due in their last analysis to the same cause—a lesion of the thyroid gland. Syphilis, tuberculosis, alcoholism, chronic malnutrition, and consanguineous marriages alike deal their first blow at the thyroid gland and alter its secretion in various directions. Does not the perfect recovery from the rachitic changes in our little patient show us that this condition is due to an alteration in the thyroid gland? Thyroid extract is essentially a specific and can

benefit only the lesions which come within its sphere of influence.

The different morbid influences which we have just enumerated do not all affect the thyroid secretion in the same manner. This secretion is very complex in its composition, containing nucleins associated with phosphorus, iodine (Baumann), arsenic (A. Gautier), and even bromine. The thyroid gland is liable to injury from several directions, and from this results the great variety of trophic disorders which are the echo of its impairment.

To return to the patient, I may state that she continued to grow till 21 years of age, when she measured 4 feet 9 inches in height. Her appearance at the age of 24 is shown in Fig. 23.

Her mother's portrait is shown in Fig. 24, her hair is silvery white, but she now finds herself healthier than fourteen years ago. Since the menopause. the thyroid secretion, which during her reproductive life was devoted to the inhibition of menstruation is now fully available for the general nutrition of the body. She still suffers, however, from breathlessness and rheumatic pains, and when these symptoms become troublesome she takes thyroidin for a time with beneficial effect.

Let us now take an example of mild myxedema occurring in a descendant of a patient presenting a more severe type of myxedema.

The patient shown in Fig. 25 was the daughter of the singing master whose case I have previously described. The woman was 40 years of age. At all times her menstruation had been profuse and exhausting and she suffered much from migraine and dullness in the head, while her hair had come out freely. When closely examined her face presented a slight degree of swelling, she was tired, worn out, somnolent, with an urgent desire to sleep. At the time she complained of sciatic pains in the left leg. Her hands were large, cold, bluish red in color and were covered with chilblains in winter. Under very small doses of thyroidin the swelling of the face disappeared, and the other symptoms ceased, including the shooting pains in the left sciatic. This case forms a good example of mild myxedema.

Finally, let me give an example where the study of the collateral relations of the patient has been of great value.

The venerable ecclesiastic shown in Figs. 26 and 27 suffered from well-marked myxedema. He was the priest of a parish in the neighborhood of Antwerp, and having visited his church, which was of archeological interest, I heard him preach. He stood by the steps of

the altar painfully supporting himself with both hands on the communion rail. His speech was so defective that I could not understand a word of his discourse. Then he mounted the steps of the altar, very slowly, and with great difficulty, leaning on the shoulder of the server. There was no doubt as to the diagnosis, he was suffering from well-marked myxedema. I had the satisfaction of quickly restoring him to health, as may be seen in the photograph. This patient, as you may imagine, had been previously treated in various ways in the course of a disease which had lasted ten years. He was much troubled by great thickening of the nasal mucous membrane, for which he consulted a specialist, who nimbly removed a portion of his turbinals, at the price of an alarming hemorrhage. Myxedematous patients are very hemophilic. The operation had no beneficial result for the patient.

I inquired into the state of health of his brothers and sisters. One of the latter lived with him, she was very thin and had a pronounced nasal voice with marked hypertrophy of the mucous membrane of the nose. This was in her the only symptom of thyroid defect. Another sister was married and presented the appearance shown in Fig. 28. She was as myxedematous as her brother the abbé. Under treatment she quickly and completely recovered her health. (Fig. 29.) But this was not all.

The abbé had a brother aged 54, who for ten years had suffered from an obscure condition characterized by weakness, anemia, and progressive exhaustion (Fig. 30). Along with the loss of strength his vision had gradually failed which had been attributed by well-known specialists to white atrophy of the optic nerves. Three years before, on his return from an exhausting journey, he suffered severely from headache, then suddenly collapsed and became unconscious with complete loss of motion and sensation. This state of coma lasted three days and on recovery he remembered nothing that had taken place, but presented no loss of functional power. He continued to suffer from weakness and exhaustion and his eyesight became steadily worse, so that when I saw him two years later he was almost blind. White atrophy of the optic nerves has been recorded several times in the course of myxedema, but I believe that this is purely a coincidence, and that there is no causal relation between these two affections. If the alteration of the optic nerves was caused by the specific myxedematous infiltration, vision would return under the influence of thyroid treatment, as we have seen that the most pronounced changes of the nervous system due to this cause are capable of complete restoration. The attack of coma which the patient had presented had placed his medical attendants in a serious difficulty. As there was no history of syphilis, the diagnosis appeared to lie between a tumor, hemorrhage, and embolism of the brain, serous apoplexy being excluded by the absence of albuminuria. I confess, if I had not previously seen the abbé and his sister, I would not have suspected thyroid weakness in this case, but once my attention was directed to this possibility it was easy to confirm the diagnosis. The low temperature, the yellowish pallor and slight swelling of the face, the fine and scanty

beard, the breathlessness, and extreme weakness all pointed to this conclusion. Even the attack of coma, hitherto so difficult to explain, fitted in with this opinion in a very simple manner. As the result of an harassing journey there was produced an abnormal exhaustion of the resources of the thyroid gland in a patient in whom they were already very restricted. This had caused a severe and sudden infiltration of the nervous centers, producing an attack of coma. The immobility secured by the unconscious condition of the patient permitted the recovery of the secretory activity of the gland, and the consequent absorption of the infiltration, so that the coma passed off without leaving any paralysis or loss of function. On being submitted to thyroid treatment, this man regained strength, color and self-confidence, his appearance being shown in Fig. 31. The anemia completely disappeared, but his eyesight did not improve nor could it be expected to do so.

A last example showing the utility of an inquiry into the family history is the case of M. A., whose photograph is shown in Figs. 32 to 34. This is the most severe case of myxedema which I have yet met with, and also that which I have most closely studied and the treatment of which I followed from day to day. When I first saw him, the patient was 63 years of age. As you see by his photograph, he was much infiltrated and weighed 221 lb., although he ate very little, having a distaste for all food, especially meat. He suffered much from breathlessness and was incapable of the slightest exertion. Three years previously he had had an attack of epistaxis, which lasted five days and threatened to prove fatal, when the bleeding was arrested by an injection of antidiphtheritic serum. Soon after this he became suddenly comatose for a period of eight hours. As he had long been known to suffer from albuminuria, and had been treated for Bright's disease, the coma and epistaxis were naturally attributed to the renal affection and regarded as signs of uremia, while the dyspnea, intellectual dullness, headaches, vertigo, and noises in the ears were believed to be due to the same cause. This interpretation must be admitted to be both scientific and logical. He also suffered from nocturnal incontinence of urine which caused him great distress. He rapidly improved under treatment, his appearance after two months being shown in Fig. 33. At this stage he was still weak and his features were somewhat haggard, but the fierce expression they had assumed was quite misleading as the man was amiability itself. His appearance after fourteen months' treatment is shown in Fig. 34. He has become a little stouter, but is in complete possession of all his faculties and his face well reflects his character as he shows extraordinary energy in the management of his affairs. His recovery was absolutely complete. Some idea of the extent of the myxedematous infiltration may be obtained by a study of the loss of weight presented by this patient. His original weight was 221 lb., which was reduced in the course of two months' treatment to 163 lb. He thus lost 48 lb., or about 23 per cent. of his total weight in sixty days, being at the average rate of nearly 13 ounces a day.

I naturally inquired into the family history and found that about ten years previously the patient's sister had died in a state of coma following great fatigue on the occasion of a removal. She had formerly had an attack of coma and had for some years presented albuminuria and been treated for Bright's disease. A photograph of this sister is reproduced in Fig. 35. You will observe that it has been carefully retouched in order to supply the absence of the eyebrows and that the photographer has unsuccessfully endeavored to reduce the swelling of the face and neck. It is certain that this woman suffered from myxedema and that the apparent Bright's disease, including the fatal attack of coma was due to myxedematous infiltration. Suitable treatment might have saved her as it saved her brother.

I need not further multiply examples, but may sum up by saying:

When you encounter the association of one or more of the following symptoms: Trophic changes in hair, eyebrows, eyelashes, teeth, or gums; an habitual chilliness, biliary disturbances with lithiasis, dyspnea with asthmatic attacks; menorrhagia, recurring abortion, hemophilia; melancholia, depression, weariness of life, migraine, vertigo, sudden loss of consciousness, noises in the ears; somnolence, rheumatoid changes in the muscles, ligaments, or aponeuroses; nocturnal incontinence of urine, pollakiuria, loss of appetite and obstinate constipation—think of a possible deficiency of the thyroid secretion.

Treatment.—I will conclude with some practical indications as to treatment. The theory of thyroid defect which I have submitted to you is based on the undoubted existence of an infiltration, the amount of which varies with the degree of deficiency, but which is always present. This theory agrees with all the known facts and is strongly supported by the complete restoration of the body as the result of treatment. There is no destruction of even the most delicate tissues, since all of them are capable of resuming their functions after the administration of thyroidin. This theory also assists us in judging the progress of the patient and explains the various incidents which may be produced in the course of treatment. *Thus, the too rapid absorption of the infiltration from the muscular, nervous, connective, or osseous tissue causes*

painful phenomena exactly similar to, but more acute than, those experienced during the primary distention. These pains are easily explained by the too sudden shrinking of the walls of the cells, and perhaps also by the increased oxidation of their contents. Too intensive a treatment produces violent headaches, neuralgia, anginiform cardiac symptoms which are apt to alarm both the patient and the doctor. Very acute rheumatoid pains also develop in the muscles, tendons, and joints, especially affecting the anterior muscles of the leg, the extensor tendons of the foot, and the joints of the toes. I have already spoken of the rise in temperature which occurs in the course of too rapid treatment.

In the treatment of myxedema, whether mild or severe, let us have the courage to be patient and proceed slowly, as it is unnecessary to cause the absorption of more than 3½ to 5 ounces of infiltration per day.

In adults a dose of 5 grains of thyroidin, corresponding to an English tabloid of thyroid extract, is quite sufficient, and even this small dose often provokes disagreeable symptoms in connection with the heart, muscles, and joints. Absorption of infiltration should be controlled by daily weighing of the patient. It is found that children tolerate thyroidin better than adults.

This fundamental treatment should be assisted by the observation of certain dietetic rules. Wine, beer, alcohol in every form, and also tobacco, should be prohibited, because these toxic agents diminish the activity of the thyroid gland, and it is important to spare that of the patient, however much degenerated it may be.

Complete rest in bed is also useful during the first period of treatment, because when in bed the thyroid secretion of the patient is not expended in useless muscular action. Certain forms of mild thyroid defect derive benefit from the administration of small doses of arsenic, iodine, or bromine, because these substances form part of the thyroid secretion. Arsenic is of value in the forms associated with migraine, while a combination of iodine and bromine benefits the cases of incontinence of urine. A few doses of an active purgative may be required to clear the viscera when loaded after long-continued constipation.

I need hardly say that I absolutely forbid all cold bathing, but hot baths on the contrary are both useful and pleasant to the patient.

When the infiltration has been absorbed, that is to say, when the patient no longer loses weight, one must endeavor to fix the dose of thyroidin necessary to maintain him in health. This varies from 1 to 6 tabloids a week, the amount being slightly increased in winter and decreased in summer. When once the patient is cured one should not be alarmed to see him increase slightly in weight, as with the better assimilation an increase of stoutness is to be expected.

Finally, it may happen that a case of myxedema comes under observation so late that treatment seems hopeless, the patient having sunk into a state of complete coma, and death seems imminent. I recently saw a woman in whom at times false respiratory movements occurred, giving the impression of Cheyne-Stokes breathing in a dying person. For several days two nurses took turns at her bedside in order to support her chin. A metal tube was inserted between her teeth and connected with a supply of ogygen, and at the same time thyroidin was administered by hypodermic injection. The symptoms rapidly improved, the periods of suspended respiration became less frequent, the somnolence disappeared, and the temperature rose considerably—an evident sign of the oxidation of the infiltration. I consider this patient was saved by the hypodermic injections of thyroidin.

In a case of prolonged coma one would be justified in performing lumbar puncture for the relief of the nervous centers.

I have now sketched the leading features of thyroid defect in its various degrees. I have not been able even to enter on a number of interesting points, but I have said enough to enable you to observe around you facts which you have perhaps hitherto not suspected. It is for you to complete these notions, and in order to do so it suffices to think of them. Trousseau has well said that the life of a physician should be a long meditation.

Just as we search all our patients for tuberculosis, syphilis, alcoholism, a day will come when a systematic examination will also be made to ascertain their thyroid powers and defects. The question of the internal secretions is coming more and more to the foreground. Innumerable possibilities may arise from it. I must now conclude and thank you for the attention and time you have so liberally given me.

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INTRATRACHEAL INSUFFLATION.

By BENJAMIN MERRILL RICKETTS, M.D.,
CINCINNATI, O.

As early as the middle of the sixteenth century Vesalius I. recognized the possibility of aerating the blood, after the chest had been opened, by passing a continuous current of air through the lungs; while in 1667 Hook read before the Royal Society a paper entitled, "An Account of an Experiment Made by Mr. Hook by Preserving Animals Alive by Blowing Through Their Lungs with Bellows."

In his experiment, Hook, having laid open the entire thorax of a dog and removed the pericardium, sustained the animal's life, first by reciprocal inflation and deflation of the lungs, in imitation of normal respiration, and then by means of a constant current of fresh air under such pressure that all respiratory movements of the lungs themselves were suspended. Both methods were successful in continuing animation and the pulmonary circulation.

While mechanical respiration remained a valuable adjunct of laboratory experiment for almost 250 years, its failure as a human resuscitative measure in the practice of such surgeons as LeRoy of France, and Monroe and Dalrymple of England, forced the scientist, John Erichson, in 1845 to conclude that, "In spite of all the improvement and modifications of the technique and the methods of inflation by bellows, mechanical respiration never again came into favor and was speedily forgotten when the postural methods came into use."

Marshall Hall was especially severe in his condemnation of forced mechanical respiration by means of bellows, and until 1887 the postural resuscitative methods of Hall, Sylvester, Schäfer, and Howard were extensively employed.

On July 23, 1887, Dr. George Edward Fell of Buffalo, N. Y., after all the postural methods of resuscitation had been tried and failed, by means of forced respiration saved the life of a patient who had taken twenty grains of morphine and some chloral hydrate, even after the pupils had dilated

in the last stage of asphyxia. Within about three months following, Prof. Dr. Boehm of the Allgemeines Krankenhaus in Vienna saved the life of Dr. Langer by the same method.

Other successful cases of a similar character followed, and established a new epoch of artificial respiration. In practice Dr. Fell used an especially devised face mask and a trachectomy tube, and later an intubation tube, the invention of Dr. O'Dwyer, connected to a single bellows and provided with a coronet piston exit valve, by the manipulation of which inflation and deflation of the lungs could be made to duplicate normal breathing. In one instance, the case of Dr. Williams, reciprocal respiration was continued intermittently during four days, with recovery of the patient, and without any untoward pulmonary laryngeal or systemic complications.

It was through the incentive given to artificial respiration by the introduction of Fell's method of "forced respiration," and his adaptation of etherization to the technique, that the surgery of the thorax, through its utilization became a possibility. F. W. Parham of New Orleans was the first to successfully remove a sarcomatous growth from the walls of the thorax, using "forced respiration anesthesia" by the Fell-O'Dwyer method, for purposes of the narcosis and combating pneumothorax. The operation was performed under reciprocal breathing, and the chest walls were closed with the lungs fully inflated. Complete recovery of the patient ensued.

About 1896 the French surgeon, Tuffier, in association with Hallion, after exhaustive intrathoracic experimentation under continuous insufflation, concluded that, "the success of this method on animals justified its use in man." In 1902 Matas turned his attention to a solution of the surgical problems involved in pneumothorax.

Kuhn of Cassel, who had made intubation a specialty since 1895, in 1905 came out with a positive differential method for compensating pneumothorax, based on insufflation by intubation. He at first closed the mouth tight, but in 1908 improved his previous technique by introducing two tubes in one, a narrow tube for inbound compressed air and a wider one for the exhaust. After many experiments on the human being, Kuhn found it advisable to keep the tube out of the trachea, and to stop it just below the larynx. While Kuhn demon-

strated the pulmonary application of anesthesia by intubation to Czerny of Heidelberg, Trendelenburg of Leipsic, Angerer of Munich, and Lotsch of Berlin visiting each of these surgeons personally, nevertheless the method was not favorably received for use in intrathoracic surgery on the human subject.

In 1908 Robinson of Boston intubated through the mouth to the bifurcation of the trachea, sending in air under pressure through a cannula, and letting the exhaust escape by way of the remaining lumen of the trachea. Later Robinson investigated the Volhard-Sollman method of oxygen insufflation, by means of which, during animal experimentation, life could be sustained without distention of the lungs; but he was distracted from perfecting this or the air-insufflation technique by becoming interested in progressive improvements which he made in both the Brauer and Sauerbruch methods.

Mikulicz, however, must be credited with the first systematic investigation of the physiological problems besetting intrathoracic surgery. At his suggestion in 1903 Sauerbruch began a series of experimental researches, and in 1904 completed cabinets for intrathoracic surgery, using respectively positive or negative pressure.

While Sauerbruch soon discarded his hyperatmospheric apparatus in favor of his negative-pressure cabinet, Brauer, working independently, developed the first positive-pressure chamber to come into general use. The apparatus devised by Karewski resembled closely that of Bauer, as did also the initial cabinets of Janeway and Green. The devices of Tiegel and Brat-Schmieden were essentially of the emergency or laboratory type, although an effort was made to adapt them to the more exacting requirements of intrathoracic surgery on the human subject.

What Carrel has termed the "classical type" (cabinet) of apparatus has found its apotheosis in the new intrathoracic surgical pavilion of Willy Meyer at the German Hospital, New York, in which it is possible to operate differential, positive, or negative pressure at will.

Until the present popularization of intratracheal insufflation by the Meltzer-Auer technique, whatever real progress has been made in intrathoracic surgery on the human subject must be credited to the "classical style" of apparatus.

In 1908 Meltzer saw Sauerbruch doing intra-

thoracic surgery, and later witnessed some of Willy Meyer's operations at the Rockefeller Institute, and seeing both pleural cavities wide open and the animals continuing to breathe, but not trusting the evidence of his own eyes he went into his laboratory to verify the experiments. A year later, in association with Dr. Auer, he published an article on "Continuous Respiration without Respiratory Movements." After Meltzer had perfected the technique of intratracheal insufflation in his laboratory, Elsberg, with the assistance of Yankauer, developed an apparatus for its use on the human subject, and personally administered anesthesia by this method for a successful thoracotomy performed by Dr. Lilienthal at Mt. Sinai Hospital in 1910.

Meanwhile, Morriston Davies, after a thorough investigation of all the intrathoracic apparatus and methods in vogue, perfected and used a hyperatmosheric device, combining both the advantages of the cabinet and intratracheal insufflation techniques, and to Elsberg, and Peck (New York) and Davies (London) belong the credit, not only of adapting intratracheal insufflation to the requirements of intrathoracic surgery, but also of demonstrating its value as a technique of narcosis in the surgery of the head and neck.

While the "classical type" of the cabinet and intratracheal style of apparatus are in apparent competition for supremacy, still each has its possibilities and its limitations, and further clinical experience alone will determine their respective utility in certain definite intrathoracic surgical procedures.

Physiological Considerations.—Progress in intrathoracic surgery has depended absolutely on the mechanical control of pneumothorax during the operative procedure. This mechanical control has varied in different methods, from that of Hook, now represented by the Meltzer-Auer technique, in which the lungs for certain periods have been so distended by a constant current of air as to preclude more or less any respiratory movements, to that of Volhard-Sollman, in which the lungs have been allowed to collapse, while a current of oxygen sustained life.

However, for all practical purposes, reciprocal breathing, under manometrically controlled positive, negative, or differental pressure, has proven the only safe method of compensating pneumothorax during intrathoracic operations upon the human subject.

Any consideration of pneumothorax involves an understanding of some elementary facts of the respiratory mechanism. Respiration is made possible in the thorax of the human subjects by a partial vacuum existing in the pleural space after the contraction of the lungs during expiration. This vacuum is represented by a varying negative pressure of from 4 to 10 mm. of mercury. Consequently thoracotomy requires either positive-pressure insufflation, with or without respiratory movements, or autorespiration in the negative or differential pressure cabinet, to prevent the collapse of the lung resulting in dyspnea, displacement of the thoracic viscera, shock, and death.

With the body of the patient within the cabinet in which the air has been rarefied to approximately the negative pressure in the pleural cavity (4 to 10 mm.) and the head of the patient is outside the chamber, permitting the respiring of normal pressure atmosphere, Sauerbruch, by varying the negative pressure as required, has been able to conduct intrathoracic operations with almost the same confidence as in abdominal surgery. In the Meyer cabinet differential pressure allows the surgeon to use positive, negative, or combinations of both pressures as needed. The great distinction between the cabinet control of pneumothorax and intratracheal insufflation is that in the former autorespiration is depended on to conserve life, and already overtaxed and weakened nerve centers are called upon to formulate respiratory impulses, while during intratracheal insufflation these centers can be made to remain passive, thereby adding a determining factor between success and failure. Moreover, in some operative procedures those muscles which participate in respiration are rendered inactive, thereby making the use of intratracheal insufflation all the more imperative. This demand is accentuated whenever during the course of any intrathoracic procedure dislocation of the posterior lobes of the lungs inhibits the usual respiratory movements.

Respiratory movements are not only concerned in the aeration of the lungs, but also contribute a factor essential to the normal maintenance of the pulmonary circulation and of considerable importance to that of the systemic. Thus the pulmonary circulation may be regarded as of double function. the ventricular, impelling the blood onward with rapid pulsations of the cardiac rhythm, while the

lungs contribute the slower but more voluminous impulses of the respiratory cycle. (Davies.)

Hence it is that during intracheal insufflation Meltzer has found it inadvisable to use a pressure which altogether suspends respiratory movements, resulting in apnea and CO_2 asphyxia, and suggests that, in so far as it is possible or necessary, the lungs be periodically deflated five or six times to secure not only a more satisfactory diffusion of the air and anesthetic in the smaller bronchi and alveoli, but also to eliminate CO_2 accumulation, and further to preserve the stimulus of respiratory movements upon the pulmonary and systemic circulations. In cases of open pneumothorax in which the respiratory mechanism is not paralyzed, spontaneous respirations offer the required aid to continuous intratracheal insufflation.

However, it is always advisable to arrange for occasional interruptions of the continuous insufflation, especially in operations in which the thorax has to be laid wide open and the posterior and inferior portions of the lungs have to be dislocated, in which condition spontaneous respirations are of no avail. The occasional deflation of the lungs insures the continuance and efficiency of the pulmonary ventilation under all circumstances. It is essential, however, that this deflation be not allowed to result in a complete collapse of the lung, for reinflation under the circumstances may leave portions of the lung atelectatio.

Moreover, such deflation is valuable as a diagnostic factor in differentiating healthy from pathological lung tissue by the change of color and the presence or absence of proper distention. Again partial deflation is useful in left-sided pneumothorax, particularly in suturing the wounds of the heart, under which circumstances hemorrhage from the heart wound, according to Friedrich, diminishes in proportion as the lungs are allowed to collapse.

The partial pneumothorax thus becomes the regulator of the hemorrhage and is allowed to persist until suture of the heart wound has been completed, after which the pericardium and pleura are closed with the lungs properly reinflated.

Extensive laboratory experiments, which have been verified by post-operative results in the human subject, prove conclusively that so far as respiratory complications are concerned intratracheal insufflation is an innocuous procedure even in the presence of the lobar pneumonia. Also it has been

found in practice that the recurrent air-stream through the trachea precludes the possibility of aspirating vomited material or hemorrhage from the pharynx.

Physiologically, the intrinsic value of intratracheal insufflation is exemplified not only in the original work of Fell, but also in the laboratory experiments of Shaklee and Githens on the treatment of strychnine poisoning, in which, although the very centers of respiration were paralyzed, intratracheal insufflation reached the climax of its usefulness as a measure of resuscitation and the conservation of life.

Meltzer has also found that anesthesia by intratracheal insufflation is far superior in many respects to the usual methods of administering ether. The anesthesia is much safer, far more readily controlled; less of the anesthetic agent is used, patients go under and come out more rapidly, and an efficient method of artificial respiration is immediately at hand to take care of untoward complications.

In recent studies on the influence of intratracheal insufflation on blood pressure and respiration, Meltzer and Githens found that a manometric pressure of from 30 to 40 mm. and a percentage of ether just sufficient to complete anesthesia, from 50 to 75 per cent. were innocuous, but that an increase of pressure and the percentage of ether for any appreciable length of time would result in an undulating fall of blood pressure, a slowly and diminished excursion of respiratory movements, with final cessation of breathing, although the heart continued to beat.

Resuscitation was possible in animals within 20 minutes by the insufflation of pure air. From the experiments it appeared that an overdose of ether first paralyzed the functions of the medulla and then much later the functions of the heart. Hence the caution in administering intratracheal insufflation to reduce the ethery percentage in ratio to the shallowness and diminished rate of respiration.

Untoward respiratory and associated cardiac complications may also result from operative manipulation of the vagi and their branches, and the resulting false apnea may be controlled by the hypodermic use of atropin.

An increase of pressure, accomplished either by means of the air current or indirectly by momentary pressure on the larynx, is valuable not only in gauging the proper distention of the lung, but also

in obviating cyanosis and in overcoming the resistance of neurotic individuals to the anesthetic effects of the ether. All investigators have found it expedient to add a tank of oxygen to their armamentarium for intratracheal insufflation. Under certain circumstances a persistent cyanosis will develop, which nothing short of oxygenation will control.

While Brauer uses the Roth-Drager, and Davies the Alcock regulating chloroform apparatus in conjunction with mechanical respiration for purposes of pulmonary anesthesia, ether seems to be a far safer agent for routine narcosis by this method.

As early as 1827 Portal produced an artificial pneumothorax without thoracotomy by the injection of sulphuretted hydrogen gas into the thoracic cavity to place the lung at rest when affected with tuberculosis. Holmgren pursued the same principle of treatment for unilateral pulmonary tuberculosis even in cases in which adhesions between the visceral and parietal pleuræ contraindicated its use. Hamman of Johns Hopkins is now utilizing a similar technique with nitrogen gas. In the presence of adhesions, the preliminary injection of saline solution to collapse the lung is advisable before injecting the gas to produce the therapeutic pneumothorax.

Perforation of the thoracic wall does not always result in pneumothorax, unless the opening is larger in diameter than that of the glottis, or is valve-like in character as in oblique puncture of wounds. As a rule, pneumothorax will disappear when the opening in the chest wall has been closed by the adjacent integumentary structures. These two facts have enabled surgeons, by means of tubes under water and the use of rubber tissue dressings, to allow for post-operative drainage of the thorax without artificial control of pneumothorax.

Technique of Intratracheal Insufflation Anesthesia.—Apparatus for intratracheal insufflation anesthesia has multiplied rapidly since the popularization of the method by Elsberg. However, all apparatus is similar in certain essentials. The source of air current may be provided by foot bellows, hand-driven or electrically-driven pumps, and tanks of compressed air. The air-current may pass directly into the ether container, or as is more advisable, is stored in a low-pressure tank or gasometer from which it passes into a Wolf bottle, to be heated and moistened, and thence by regulating

valve, either directly into, or only partially through the ether container, thereby providing for aeration pure and simple or insufflation with varying percentages of ether.

Also a source of oxygenation is an expedient adjunct. The tube from the apparatus connects with a mercury manometer and thence to the intubation tube.

Davies, adopting Kuhn's idea of a double intubation tube, uses a manometer to gauge the inlet and outlet air pressures.

All experimenters have concluded that for the Meltzer-Auer technique a silk-woven catheter 30 cm. long and of a diameter one-half that of the glottis, usually from 22 to 26 of the French scale, serves as the best intubation tube. It should have an opening similar to the rectal tube at the tracheal end, should be absolutely smooth and semi-rigid to prevent it from being expelled by coughing or from being compressed while in position.

Its introduction is best accomplished after the preliminary introduction of narcosis by ethyl chloride-ether or nitrous oxide-oxygen-ether anesthesia.

Intubation is greatly facilitated by means of either the Jackson direct laryngoscope, Fischer's modification of Hayes's instrument, or the introducer devised by Cotton. After the patient has been deeply narcotized the mouth is opened wide and so held by a gag. The head is well brought forward and the tongue pulled forward by an assistant until the opening of the larynx is brought into view. The metal guide is introduced into the opening and the intubation tube is gently pushed onward until it is seen to pass over the epiglottis into the larynx, after which the metal guide is withdrawn and the tube is pushed further into the trachea until it meets an obstruction, which is either the wall of the right bronchus or the bifurcation of the trachea. It is then withdrawn an inch and is anchored in position to special mouth gags provided for the purpose.

The distance from the incisors to the bifurcation of the trachea is from 9 to 10 inches in the infant, 12 in a child, and about 17 inches in the adult. The glottis in the adult is one-half the distance between the incisors and the bifurcation of the trachea, and Elsberg suggests making the intubation catheter accordingly to insure greater accuracy in adjusting its location.

One-eighth to ¼ gr. of morphine hypodermically ten minutes before the administration of ether to reduce the irritability of the larynx; or from twenty minutes to half an hour previously when preliminary anesthesia by ether is not resorted to. The induction of narcosis by intratracheal insufflation produces spasmodic coughing while the patient remains conscious.

With the intubation tube introduced to the correct position, air may be heard rushing through the catheter. Spasm of the larynx may now occur for a few moments, but is of no consequence. At this juncture the tube from the apparatus is connected to the catheter with the pressure gauge of the manometer controlling the air supply at 20 mm. and the ether percentage at 50. If the lungs are not kept properly distended by a pressure of from 10 to 20 mm. Hg, the intratracheal tube is either out of position in the right bronchus or is too small and is allowing too much air to escape by way of the trachea. In the first instance the tube must be retracted and in the second either a larger sized tube must be introduced or else slight compression of the trachea around the tube at the jugulum must be intermittently utilized. Too large a tube causes CO_2 accumulation and cyanosis.

Complete muscular relaxation is usually obtained with from 50 to 75 per cent. of ether, and during the course of narcosis the breathing is quiet, respirations are reduced by one-third, the face remains pink, while the veins of the forehead become prominent; the pulse usually remains full, bounding, and regular, the pupils do not dilate, and frequently the corneal reflex is active, so much so that the condition of the patient is rather one of analgesia than anesthesia. Reaction from the anesthesia is so rapid that care must be exercised to keep up etherization throughout the entire operation. The depth of narcosis is controlled by increasing the air pressure and the percentage of ether while at the same time avoiding a condition of apnea, which supervenens at pressures of from 30 to 40 mm. Cyanosis and the accumulation of CO_2 during the operative procedure are controlled by periodic deflation and occasional oxygenation with air. Pure oxygen is dangerous on account of its toxicity when its tension becomes too great, and it is too freely absorbed by the circulation.

At the close of anesthesia, ether is turned off, and pure air, or a combination of air and oxygen, is in-

sufflated under slightly increased pressure to blow out the ether from the trachea and alveoli. Patients come out from under the influence of pulmonary anesthesia by this method almost as soon as the insufflation is discontinued and the tracheal tube removed. Apnea is present for a few moments after the removal of the intubation tube, but regular breathing is then rapidly re-established. Post-anesthetic vomiting, headache, dyspnea, and cardiac complications are of rare occurence. Postoperative pneumonia, interstitial emphysema and pleural effusions have followed intrathoracic operations done under both cabinet and intratracheal insufflation methods and the determining etiological factor in the complication has not been readily determined. However, caution must be directed to a technical mishap which may occur, and that is an accidental injection of fluid ether into the lungs. Fischer quotes a case in his own experience in which death resulted from this contretemps and the mishap has occurred in laboratory experiments. Only apparatus should be used which mechanically precludes the possibility of such an occurrence.

Again, post-operatively, it may become necessary to resume insufflation without anesthesia to control shock, to aid flagging respiration and circulation, and to prevent serious effusions. Under such circumstances the success of Fell in saving life under the most disheartening conditions must be remembered, and the method pursued to its limits.

Aside from its value purely as a resuscitative measure in asphyxia, drowning, poisoning from drugs and anesthetics, the convulsive stage of rabies and tetanus, impaired respiratory function in certain diseases, it must also be remembered that intratracheal insufflation as a method of anesthesia is a very desirable adjunct to the surgery of the head and neck, and especially for operations on the spine when the patient must be placed flat on the stomach during the course of operative procedure.

While etherization in association with intracheal insufflation appears to be the safest form of anesthesia for intrathoracic operations, Boothby, after using the nitrous oxide-oxygen-ether technique, has been favorably impressed with the latter method, and Willy Meyer also suggests that the innocuousness of nitrous oxide may play an important rôle in conserving patients under thoracotomy the additional shock of a poisonous anesthetic agent.

FOUR CLINICAL LECTURES.

(Brief Abstracts)

HELD AT THE NEW YORK POLYCLINIC MEDICAL SCHOOL AND HOSPITAL DURING THE WEEK OF THE MEETING OF THE INTERNATIONAL SURGICAL CONGRESS.

I—FRACTURES AND BURNS.

By JOHN A. WYETH, M.D.

Dr. Wyeth, as President of the Faculty and Senior Surgeon of the New York Polyclinic Medical School and Hospital, after welcoming the visiting physicians and surgeons, spoke briefly on "Fractures and Burns," presenting illustrative cases of each. Dr. Wyeth said in part:

I shall speak this morning of factures of a single bone, the patella. As you know, fractures of the patella are caused, as a rule, by violent contractions of the quadriceps extensor muscle while the leg is in extreme flexion. The bone may be broken by a direct blow or by a fall on the knee. A blow and muscular action may combine to break it.

The line of fracture is usually transverse, or nearly so, just below the middle of the bone. The break may occur, however, above or below this plane. Occasionally the bone is split longitudinally by direct violence, or it may be comminuted. Fracture of the patella is rarely incomplete, the separation of the fragments varying from the smallest fraction of an inch to as much as two or more inches, and being wider at the inner than the outer border. This lesion occurs, in the majority of instances, between the ages of twenty and forty, and is more common in males than in females.

Because of the superficial location of the lesion the diagnosis is easily made, the depression between the separated fragments serving as a guide. Should the separation be very slight, lateral motion of one fragment upon the other will elicit crepitus.

In the treatment of this condition I have lately used a very simple, satisfactory, and painless method of holding the fragments in continuous apposition. After the transverse incision, which exposes both broken surfaces, the clot is washed out with hot sterile salt solution, and the frazzled

periosteal edges are sutured together with fine linen, thus approximating the fragments. The skin incision is then sutured with chromicized catgut.

On a two and a half inch, half-curved Hagedorn needle, a very strong linen thread, twelve inches long (No. 4) is carried from side to side, deep into the substance of the ligamentum patellæ, just at its insertion into the lower rim of this bone. A like thread is inserted well into the substance of the quadriceps extensor tendon at the upper margin of the upper fragment. A light gauze dressing is placed over the line of incision, and over this the apposing ends of the two linen loops on each side, above and below, are tied tight enough to hold the fragments in close and continuous apposition, and without the possibility of overriding.

A dry gauze dressing covers the field of operation, and a plaster-of-Paris cast is applied, holding the knee immobilized for eight weeks. At the expiration of this time the holding sutures are removed and the fragments are held securely in apposition, while the knee is bent to not more than 20 degrees. The cast is reapplied and worn for four weeks longer.

The limb is now put to use, with the necessary precaution to prevent severe strain in overflexion for at least six months.

I have employed this method in the cases presented, with satisfactory results.

I wish also to present a case of an extensive burn of the face, neck, chest, and right arm. This patient was brought to the hospital in the ambulance last night, in a state of shock, after having been rescued from a burning building.

In connection with this case it may not be amiss to recall to your minds a few points concerning burns and scalds, which, as you know, may vary in degree from the mildest form which produces a simple inflammation of the epidermis, to the most severe form, which destroys all the tissues or organs or a part, and which may result in the death of the individual. The gravity of the prognosis is usually proportionate to the extent of the surface of the integument destroyed, rather than to the depth of the destructive process.

Burns of the head and face, such as the patient presented, are the most dangerous; those of the extremities the least grave. Recovery rarely follows destruction of one-third of the cutaneous surface. Death may result from shock, from ulcer of

the duodenum, or from exhaustion following prolonged suppuration and septic absorption.

When a severe burn or scald is encountered the immediate indication is relief of pain by the hypodermatic administration of morphia, or by some form of opium given by rectum or stomach. The most convenient local remedy is a saturated solution of baking soda in water, with submersion of the burned surface, if possible, or a mixture of bicarbonate of soda and cornstarch, one teaspoonful of each to a quart of water. The dressing should be kept wet with the solution, which is applied freely to the burned area. After five or six hours the free application of the following mixture, made into an emulsion, will be found beneficial:

Ichthyol ..... ℥ss
Cotton-seed or olive oil..... O.ss
Limewater ..... O.ss

This should be continuously applied for the first three to five days during the stage of acute inflammation.

In order to bring about rapid repair of the skin the following ingredients, mixed thoroughly, may be used:

Ichthyol ..... ℥j
Diachylon ointment,
White vaseline ..... āā ℥iij

If these remedies are not convenient, the following may be substituted with equal benefit:

Lead plaster,
Liquid albolene,
Lanolin,
Vaseline ..... āā ℥j

These are melted together, and, when cooling, 40 minims of ichthyol added.

Either of these ointments should be applied thickly on the soft, linty side of canton flannel, on surgeon's lint, or on several layers of sterile gauze. The application should be repeated daily at first, after opening all blebs. In opening the blebs care should be taken not to remove the epidermis of the bleb, as this may become revitalized, thus greatly accelerating the healing process. In changing the dressing it is important not to disturb new granulations, but simply to wipe over them. When healing is well under way the dressing need be changed only every second or third day.

In the treatment of the depression or shock which often follows severe burns, stimulation with whiskey

or brandy, by enema or by mouth, is indicated, as well as the hypodermic injection of morphine. Physiological salt solution, introduced by the colon, or injected into the areolar tissue, is of great value when the burn is extensive and the shock profound. It should always be remembered that opium and alcohol should be given sufficiently cautiously to avoid too profound narcosis with the former, and with the latter increase in the fever reaction which follows when the patient rallies from the shock.

In an emergency, when the remedies mentioned may not be obtained, a coating of ordinary white lead, as mixed for use in painting dwellings, is an efficient protection when poured over the burn. Flour sprinkled over until all the excoriated surface is well hidden is a method of treatment which is applicable in almost any emergency. Rubber tissue, or oil-silk, sterilized and laid over the raw surface, with cotton batting applied over it, but never directly on the burned surface, is equally efficient. Lint, or a soft cloth, dipped in a 2 per cent. carbolized oil, may be employed directly on the burn.

No pressure should be exercised in holding the dressings in place. When the back and posterior aspects of the extremities are chiefly involved, the prone position is of necessity maintained.

FOUR CLINICAL LECTURES.
II—OPERATIONS FOR INGUINAL HERNIA UNDER LOCAL ANESTHESIA.

By JOHN A. BODINE, M.D.

Dr. BODINE presented a case of ordinary right-sided inguinal hernia, upon which he operated under local anesthesia, employing a solution of novocain, 1-500. Dr. Bodine said in part:

In operating under local anesthesia the nervous apprehension of the patient may be quieted to a great degree by a calmative demeanor upon the part of the surgeon and his assistants. This is also influenced by the patient's position upon the table. A position of comfort and relaxation should be maintained. If the arms are crossed above the head the patient will be "fidgety" throughout the operation.

In the effort to avoid bleeding points it is of great importance that the lower end of the incision should not extend beyond the lateral end of the suprapubic skin fold. It may be started as high as one desires. This incision may be deepened to the operative field without encountering a vessel large enough to demand a ligature, whereas, if the incision be extended an inch lower, a large number of ligatures will be required. Not only does the catgut thus employed, when softened by the tissues, invite sepsis, but, in cocaine work, it means a number of acute stabbing pains whenever a blood vessel is cut or tied. The incision so placed gives ample room by downward traction of the mobile skin, and nearly always permits the completion of the operation without the use of a ligature.

The line of incision is infiltrated with the novocain or other anesthetic solution throughout its extent. This is accomplished by introducing the needle just under the superficial epithelium. The anesthesia thus induced will permit the incision to be painlessly deepened to the aponeurosis of the external oblique. Upon splitting and reflecting the aponeurosis, the iliohypogastric nerve is anesthetized by injecting a little of the solution into the sheath of the nerve as high up as possible.

The ilio-inguinal nerve is also sought, but if it is not found the hernial coverings should be infiltrated in a straight line over the neck of the sac, and the

incision deepened until this structure is reached. If omentum is excised it is generally gently withdrawn, ligated and amputated without additonal injection, and, in my experience, without pain. If, however, only the iliohypogastric nerve has been found some infiltration will be needed into the conjoined tendon. The cord is lifted from its position and the operation concluded according to the Bassini method.

Two suggestions are offered which have proved of worth to me: First, if a strip of gauze is used to hold up the cord it may, during the necessary manipulation, roll the cord on its long axis, and exposure to air may cause agglutination in this position of torsion. A wetted strand of catgut as a retractor will obviate this danger. Second, the deep suture should include but one-half the thickness of the conjoined tendon, thus avoiding strangulation.

FOUR CLINICAL LECTURES.

III—FRACTURES.

By ALEXANDER LYLE, M.D.

Dr. LYLE presented three patients whose histories were briefly detailed, in part, as follows:

CASE I.—This patient, a man forty-nine years of age, while working in the well of an elevator was crushed by the descending car, sustaining injuries to the pelvis. Upon being released from his confined position he complained of scarcely any pain, and it was not until he made an attempt to stand that the pain became noticeable.

Careful examination revealed a fracture of the pelvis, the exact extent of which could not be determined by palpation. Examination by rectum elicited no evidence of injury to this organ, and catheterization of the bladder showed no blood in the urine. Shock was not marked. The patient was placed in bed and a very tight wide muslin binder put about the pelvis. The following day an *x*-ray examination was made, which revealed a very marked bilateral fracture of the pelvis. In the skiagraph an irregular line of the fracture could be seen, starting from the crest of the ilium and extending down, terminating in the greater sciatic notch. There were no wounds in the skin.

The patient was observed very closely after being put to bed, in order to determine the presence of any internal injuries, but the abdominal viscera proved to be uninjured.

The treatment of this case, like that of all fractures of the pelvis, has been very simple. Straps of adhesive plaster were brought around the pelvis, in order to immobilize it, and this was reinforced by a firm muslin bandage. The patient's convalescence has been uneventful. He will be kept quiet in bed for eight weeks before he is allowed to stand.

In all cases of this kind it is well to bear in mind the great danger of rupture of the urethra, of the bladder, and of the pelvic vessels. As soon as internal injury can be determined operation should be resorted to.

CASE II.—The next patient, a woman of thirty-five years of age, sustained a transverse fracture of the surgical neck of the humerus, as shown in Fig. 1. The head of the humerus may be seen in the glenoid fossa, while the broken end of the shaft has pierced the pectoral muscles and lies immediately beneath the clavicle.

After several attempts to reduce this fracture and to hold it in position by mechanical means had failed, it was determined to open through the deltoid and replace the fragments. With complete ether anesthesia, the bone was exposed. Upon bringing the fragments to-

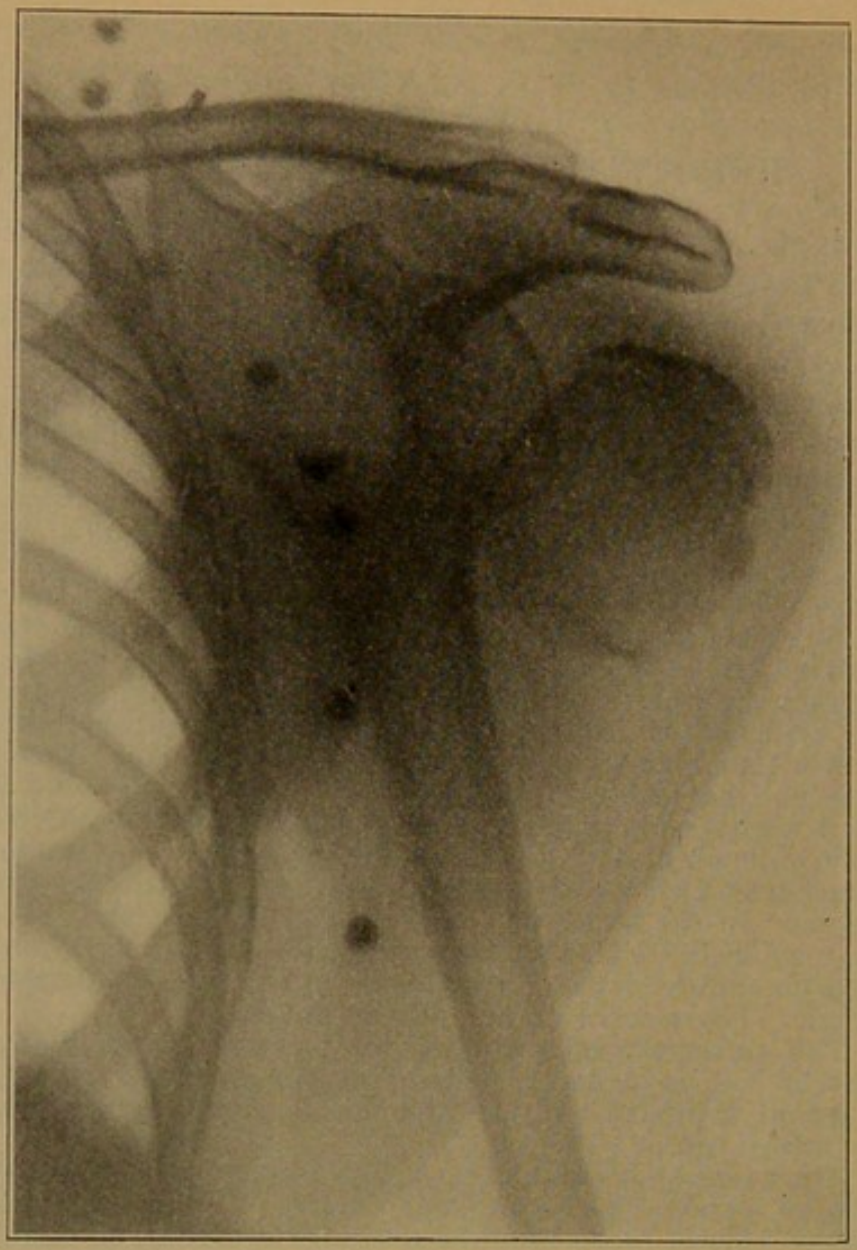


FIG. 1.—Fracture of the neck of the humerus.

gether it was found that they could be dove-tailed into each other so firmly that plating or wiring was unnecessary. The tissues were then sutured, a small pad of cotton placed in the axilla, and a firm plaster-of-Paris spica applied.

The patient's condition being excellent, the dressing was not removed for four weeks. It was then taken down and a light one applied, which remained for two weeks longer. Following removal of this, massage and passive motion were used, the patient now having almost complete use of her arm. Fig. 2 shows the condition after operation.

CASE III.—The third patient is a young man twenty-four years of age, who sustained a fracture of the lower third of the right femur, as shown in Fig. 3. He is a truck driver by occupation, and is consequently very muscular. It will be noticed in the plate that the muscles have contracted to such an extent that the fragments have overlapped fully an inch.

Formerly our treatment in such cases consisted

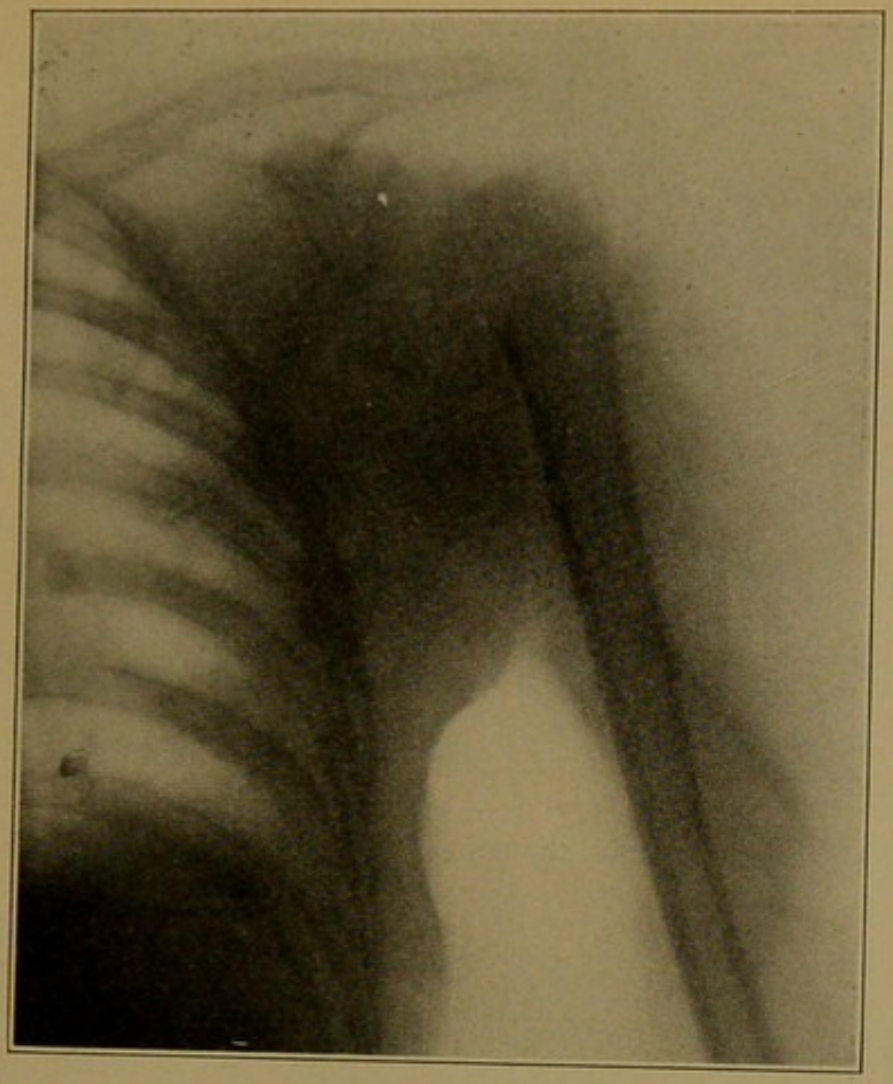
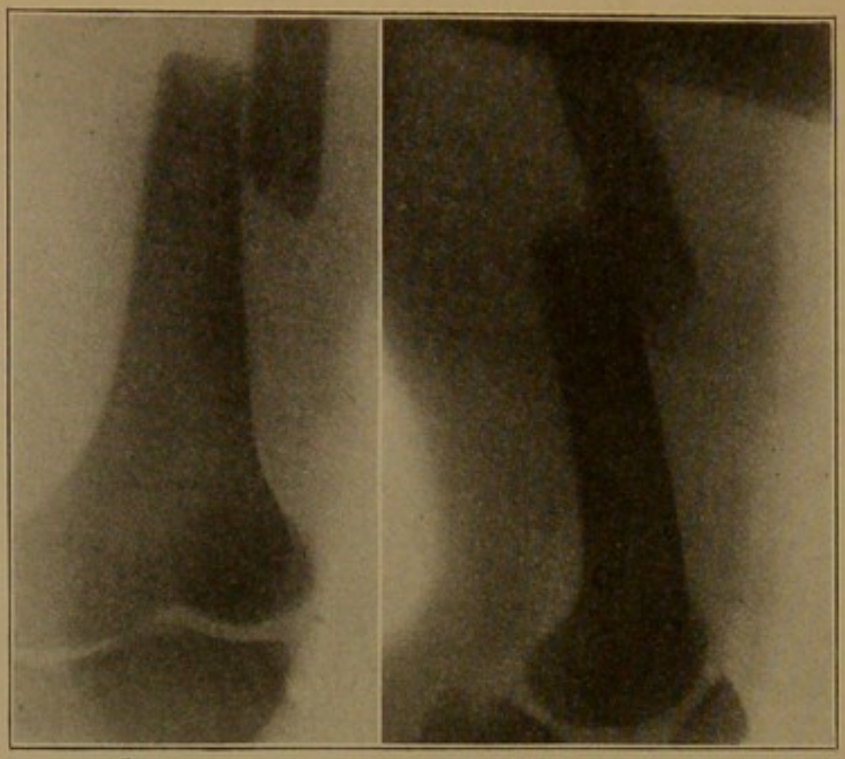


FIG. 2.—Fracture of the neck of the humerus after reduction.

in the application of Buck's extension, with sufficient weights attached to counteract the muscular force. Much of this traction was exerted upon or below the knee, and consequently knee-joint complications often became more severe than the fracture. We now have a very decided improvement in the line of traction in the Steinmann nail. This consists of a long spike of steel which is put through the lower end of the femur, just above the condyles, and by the use of tongs traction is exerted upon this, the knee-joint being left perfectly free.

Fig. 4 shows the femur after the Steinmann apparatus has been in service and twelve pounds of weight applied over the pulley. It will be observed that not only has the muscular force been overcome, but that the fragments have been pulled over an inch apart. At this point the weights were reduced to six pounds, and a plaster-of-Paris roll bandage applied over the lower two-thirds of the femur, in order thoroughly to immobilize the fragments that are now in perfect alignment.



A B

FIG. 3.—Fracture of the femur; A, front view; B, side view.

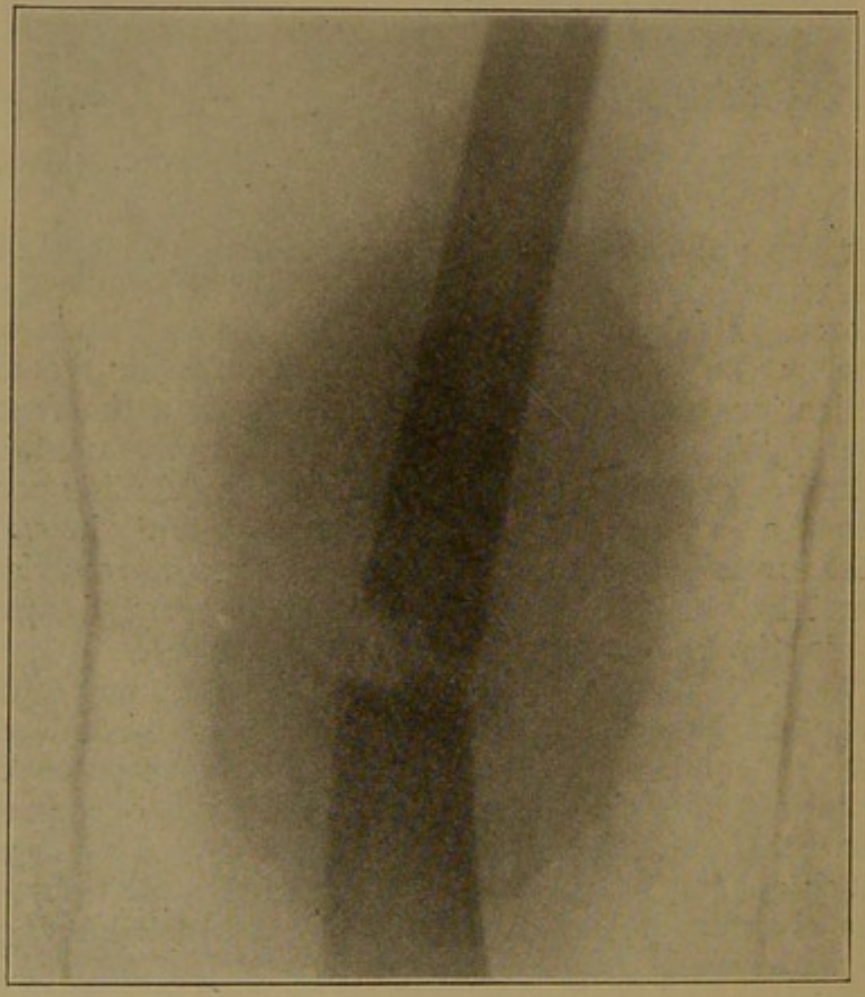


FIG. 4.—Fracture of the femur, shown in Fig. 3, after treatment with the Steinmann apparatus.

The patient's convalesence was normal in every respect and he was able to walk out of the hospital, without the assistance of even a cane, at the end of eight weeks.

FOUR CLINICAL LECTURES.

IV—CANCER.

By WILLIAM SEAMAN BAINBRIDGE, M.D.

Dr. BAINBRIDGE presented a number of patients. In some instances the patients had been operated upon for cancer at a previous time, in others operations were performed on this occasion. One particular case belonging to the first category, suggested certain practical points with reference to the diagnosis and treatment of malignant disease, a partial report of which is here given:

The patient, a woman, seventy-one years of age, presented herself at the hospital in March, 1914, complaining of "throat trouble." In January, according to the history, another physician opened the left tonsil for "quinsy," the tonsil at that time being enlarged and painful. The diagnosis of syphilis had been made by one physician consulted, but the Wassermann reaction was negative, as was likewise the history. Before I saw her for the first time, in March, a small piece of the tonsil had been removed for pathological examination, and the pathologist had reported "inflammatory tissue."

When the patient came under my observation the tonsil was very much enlarged, indurated, and the surface ulcerated. The surrounding tissue was edematous and reddened. The patient complained of great pain, and of difficulty in swallowing. Three sections were removed from the ulcerated portion, and these were given to three independent pathologists for examination. One sent in the report, "inflammatory tissue"; the other two reported "sarcoma." The latter coincided with the clinical diagnosis, and, inasmuch as the mass was growing very rapidly, with danger of death from hemorrhage, immediate operation was advised.

On April 3, at the Polyclinic Hospital, the patient was operated upon under general ether anesthesia. The external carotid artery, including the ascending pharyngeal branch, was ligated, and the mass removed, with as thorough dissection as circumstances allowed.

The patient recovered from the operation, and experienced great relief from the dysphagia and general suffering caused by this enormous mass, which had filled almost the entire mouth. To have left this mass, involving the soft palate, and interfering with respiration and deglutition, would have meant early death, either from hemorrhage or from pressure upon the trachea and consequent interference with breathing. By tying off the large vessels it was possible to remove the tumor, to relieve suffering and to prolong life.

After the operation pieces of the tonsil were again sent to different pathologists for a verification, if possible, of the clinical diagnosis of malignancy. Of the four pathologists who examined these independently,

three reported round-celled sarcoma, and one, inflammatory tissue.

A case of this kind is fraught with a number of valuable lessons.

In the first place, it emphasizes the vital importance of making a careful and thorough diagnosis, even in apparently simple affections such as "sore throat" and "quinsy." Many times, in the daily routine of the busy practitioner or of the hospital or dispensary physician, it is difficult to accord to each case the careful diagnostic consideration which it should receive. For this reason the conscientious and capable physician or surgeon may make errors in diagnosis which a little more time, care, and thought would obviate.

The same thing, it seems, applies to the pathologist. If, armed with an adequate history of the case and with the clinician's presumptive diagnosis of malignancy, the pathologist is content to examine one or two slides with negative results, and to render a negative report, he is very apt to fall into many pitfalls in diagnosis. Ordinarily, a few slides will reveal the true diagnosis, but it must not be forgotten that this may not be the case. Sometimes malignancy is established only after the examination of a large number of sections. Numbers of instances of this kind have been reported by myself and others. For this reason a negative pathological diagnosis should not be accepted in the light of positive clinical evidence of malignancy. The case under discussion illustrates this point.

In this connection it may be reiterated that adequate clinical data in each case should be given to the pathologist. It is fair neither to the pathologist, to the patient, nor to the surgeon himself, that the laboratory worker be expected, from the sections alone, to give reliable findings in all cases.

In the second place, the case emphasizes the importance of bearing in mind the question of the auto-infectivity of cancer. It cannot be positively stated, but, from the subsequent history, it is quite probable that the first operation, that of incising the tonsil for what was supposed to be "quinsy," or acute suppurative tonsillitis, stimulated malignant growth and accounted for the rapid extension of the sarcoma. It is likewise quite fair to assume that this operation very materially affected the ultimate outcome of the case, for, while the patient, at the present time, is fairly com-

fortable, can breathe and swallow with ease, and is free from the danger of immediate death from hemorrhage, suspicious induration in the neck suggests early and rapid recurrence.

This patient's history recalls another case which came under my observation in this institution in 1908. Each illustrates, in a telling manner, the dangers of breaking down nature's barriers either for the purpose of taking specimens for microscopic study or through an error in diagnosis. In the case which I have just presented to you the growth was first cut into through a mistake in diagnosis, and afterward for the purpose of taking a specimen. In the other case to which I refer the second error was committed. In this instance a little boy, three years of age, developed a small tumor in front of the ear after a fall which caused a bruise in this region. The interne in the dispensary of the hospital to which the mother carried the child cut into this small lump and took out a section for pathological examination, telling the mother to return with the child in two weeks. The result was that the tumor increased enormously in size and with great rapidity, with involvement of the lymphatics of the neck and with metastases in the liver, spleen, and testes. Within about nine months from the time the child received the fall the condition had become inoperable, and the neoplasms, which proved to be sarcoma, irremovable. This was the condition when I first saw the patient, consequently no operation was performed. The child died early in December, the fall having occurred in February.

Such cases, one in a woman of seventy-one and the other in a child of three, emphasize the importance of keeping nature's barriers intact, in all instances where there is a doubtful question of malignancy. Instead of incising the unbroken skin or mucous membrane, the entire tumor should be removed and the section taken afterward for purposes of verification of diagnosis, and for determining the advisability of more extensive removal of tissue. If, perchance, nature's barriers have already been broken down, as in the case of the woman, a section may generally be removed without increasing the danger of extension. Under all circumstances, when the clinical diagnosis of maligancy is doubtful and a section is removed for microscopic verification or reinforcement, a prompt report should be insisted upon, in order that immediate operation

may be resorted to if necessary. Inasmuch as negative pathological reports are not to be accepted in the face of positive clinical diagnosis, it is often advantageous to resort to frozen sections at the time of operation for the purpose of determining, in the light of the pathological findings, the extent of operative interference.

In closing I wish to reiterate once more the imperative duty of every surgeon who operates for cancer to utilize all the diagnostic aids now at our command before pronouncing a condition malignant or non-malignant. Sins of commission may be just as disastrous to the patient as sins of omission in the matter of operative interference, and it is only by refinements of diagnosis, plus the careful weighing of all the evidence that we may avoid both pitfalls.