Returning to the abdomen, it was found a little difficult to locate the upper end of the speculum, so as to make the safest possible aperture for the isolated loop of gut; but when well placed, the peritoneum over it was incised and the open end of the speculum cut down upon.

A pair of sponge holders was passed up from below, and a ligature on the closed end of the freed sigmoid loop seized and pulled down to the perineum. The peritoneum and such subjacent parts as could be gathered, together with the lower ends of the rudimentary uterine halves, were attached to and round the upper closed end of the new vagina. The lower end was now opened and the mucosa and muscular wall sutured to the skin margin.

At first there was some tendency of the new tube to diminish in diameter, but an obturator of stent, and later one of vulcanite, was provided, so that the lumen might be kept patent. After a few months an interesting observation was made: about a couple of inches within the orifice a band of circular muscular contraction, about one-half to three-quarters of an inch in extent, was encountered by the examining finger. The first stimulus to the mucosa excited a very strong contraction, which gripped the finger, but if gentle pressure were maintained the muscle relaxed completely, and the whole of the rest of the passage could be reached. Eventually the new vagina admitted a full-sized tubular speculum.

At first there was a considerable discharge of mucus, but with simple douching that was controlled, and generally has been very much less, though it has not entirely stopped. Moreover, on two or three occasions there has been a slight loss of blood, which appeared to come from a little ulcer in the mucosa high up in the passage. The mucosa itself is slowly losing its distinctly intestinal appearance, so that, with the speculum in place, it might pass for a normal vaginal lining.

Marriage, which was postponed for nine months, has been followed by sexual intercourse, believed to be normal. The patient experiences normal sensations, and states that she is able to relax and contract the sphincter-like muscular tract, if not quite at will, certainly with conscious effort. It is an interesting question whether this very definite sphincter-like section of the sigmoid loop represents the sphincter responsible for Hirschsprung's megacolon and allied conditions.

In this case the mesentery of the small intestine was very short, and to have carried a double loop successfully to the perineum would have been a difficult task; indeed, it appeared impossible without putting such tension on the vessels as would have made the life of the loop extremely precarious.

Upon a review of all the circumstances it seems to be wiser to use colon rather than small gut; at any rate, this case indicates that, as an alternative, the colon is available.

The prospective husband was informed of the circumstances, the findings, and the implications. The psychological and other aspects of the whole undertaking were carefully considered.

This is not the first use of sigmoid for the construction of the vagina. At least five cases are recorded—those of Albrecht (three), Boldt and Rüge¹—and probably there are others.² A short bibliography is attached to Mr. Norman Hodgson's report of a successful case, using small intestine, in the *British Medical Journal* of May 13th, 1933 (p. 822).

For the success of this case I am indebted to the skilful and patient co-operation of Miss Burton, the ward sister.

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LEAKING CEREBRAL ANEURYSM

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As cases of leaking cerebral aneurysm diagnosed *intra vitam* are unfortunately still rare, the case mentioned below seems worthy of record. It is of additional interest, moreover, in that the leak occurred during an eclamptic fit, which somewhat complicated the diagnosis.

CASE REPORT

A multipara, aged 38, was sent into hospital as a case of ante-partum haemorrhage. The history revealed nothing of note except that six months previously she had experienced a sudden intense occipital headache, which had gradually subsided. There was no history suggesting renal involvement. On admission the patient did not appear very ill, but there was marked albuminuria and a blood pressure of 215/140; there was no oedema. There were no labour pains, but the cervix was three fingers dilated; the placenta could not be

A = Ruptured aneurysm. B = Second aneurysm, unruptured.

felt. The following night the patient suddenly fainted, labour pains began, and there was a large discharge of blood from the uterus. The membranes were ruptured, and shortly afterwards a stillborn child was delivered. The mother was very collapsed, with a pulse rate of 130 and a blood pressure of 140. Two hours later she had a fit, lasting six minutes, with opisthotonos and clonic movements of the limbs. Under treatment for eclampsia with morphine and chloral she improved a little, but remained somewhat cyanosed. Till the fourth day there was little change, the patient remaining drowsy. The temperature rose to 101°, the breasts became very tender, and at the same time the patient became more lethargic. The blood pressure had now risen to 180.

On the sixth day after delivery the patient developed a right hemiplegia. The diagnosis at this stage appeared to lie between that of a leaking cerebral aneurysm or of an embolic cerebral abscess. A uraemic hemiplegia was also considered, as was a syphilitic thrombosis, but this was thought to be unlikely. After due consideration it was decided that the most probable diagnosis was leaking aneurysm, and this was confirmed by lumbar puncture, when the cerebro-spinal fluid was found to be intimately mixed with blood, with a coloured supernatant fluid after centrifugalization. The only other findings in the fluid of note were a urea content of 40 mg. per cent. and a negative Wassermann reaction. The patient gradually became more comatose, and died on the tenth day. On post-mortem examination a small ruptured

aneurysm was found at the origin of the left middle cerebral artery, and another aneurysm, about 3 mm. in diameter, was situated close by on the posterior communicating artery.

On reviewing the case it seems likely that six months previously the patient had a slight leak from a congenital aneurysm. In the latter months of pregnancy she became toxæmic, and this resulted in an accidental haemorrhage and, finally, in an eclamptic fit, which caused the aneurysm to start leaking again, with a fatal result.

COMMENTARY

Cerebral aneurysms, both symptomless and as a cause of cerebral haemorrhage, certainly occur more frequently than has in the past been recognized. They are found in about 1 per cent. of all necropsies where the arteries of the brain are examined, but Busse records the presence of small aneurysmal dilatations on the anterior communicating artery in as many as 10 per cent. of all post-mortem specimens. Their origin, in the majority of cases, is due to a congenital weakness of the pars media, usually at the junction of two vessels, and they have been found as early as the second year of life. Embolic phenomena are, however, responsible for a certain number of aneurysms, as also, occasionally, are arteriosclerosis and syphilis.

The infrequency of *intra vitam* diagnosis of cerebral aneurysm is not surprising when it is realized that until haemorrhage occurs, as eventually it nearly always does, there are no symptoms unless calcification or clotting has taken place, for then, and only then, do pressure symptoms arise. These, of course, vary according to the site of the aneurysm, and can only be diagnosed from those due to neoplasm by the observation of calcification in the wall of the sac on x-ray examination, or, better, by means of an arterio-radiogram. When bleeding occurs it may be sudden, with a rapidly fatal result, or there may be a series of leaks, with subsequent clotting at intervals of months or even years, and death may not occur until as long as twenty years after the first haemorrhage. It is in this type of case, where there are repeated small haemorrhages, that a diagnosis is frequently possible. The leak may be subdural, with the formation of a haematoma, sometimes causing symptoms similar to those of encephalitis lethargica; it may be into the subarachnoid cavity, causing meningism, or into the brain substance, with transient hemiplegia, aphasia, or an attack simulating severe migraine. The attack may be accompanied by albuminuria or glycosuria.

Of great value in the diagnosis of aneurysm leaking into the subarachnoid cavity, as of subarachnoid haemorrhage from any cause, is lumbar puncture, when the fluid will be found to be evenly mixed with blood, which will have caused staining of the fluid after the cells have been removed by centrifuging. Red cells may be found in the cerebro-spinal fluid for about three days after a haemorrhage, but the fluid will be coloured for a period of three or four weeks.

The ultimate results of treatment are not promising, as haemorrhage, even if it ceases for a time, nearly always recurs. Collier advises repeated lumbar puncture so long as the cerebro-spinal fluid pressure is above normal, and Schmidt suggests pressure on the common carotid artery followed later by ligature of the internal carotid. This seems rather a heroic procedure, but it is certainly justified by the poor results of more palliative treatment.

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Clinical Memoranda

EXTREME HYPERGLYCAEMIA IN DIABETIC COMA

From time to time various figures of extreme hyperglycaemia in diabetic coma have been published from different countries, almost vying with each other, as sportsmen do, for the record. None has to date exceeded the blood sugar concentration of 2.06 per cent. observed in 1930 in one of my cases. This sounds more like a urinary than a blood sugar analysis, but the figure was confirmed by multiple analyses of the blood filtrate by McLean's method.

The case was one of profound and prolonged diabetic coma in an elderly woman, who was almost pulseless and so weak that air-hunger was replaced by rapid shallow breathing. Two hours before this high blood sugar figure 40 units of insulin had been given; another 40 units afterwards, with abundant saline, reduced the blood sugar to 1.568 per cent., so that the initial figure may have been even higher before any treatment. The patient died in spite of all efforts.

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A CASE OF HEART-BLOCK

The following notes on a somewhat unusual case would appear to be worth recording.

The patient was a medical man, seen in consultation with Dr. R. O. Eades and Dr. Bessie Armour. At the time he was taken ill he was carrying on a large general practice. He was very active, and was a golfer. In his time he had been an athlete and also a climber and a shot. Some years ago, when in the sixties, I had advised him to give up swimming, because of transient attacks of precordial pain. During this period he had been subject to pains of the nature of angina, and on one or two occasions had had fainting attacks. He had, however, never been laid up, and had led a regular, normal, hard-working life.

On the night of May 27th, 1932, he was awakened with violent pain in his chest and intense dyspnoea. He was greatly distressed, and became very restless. His pulse was very weak at the wrist, and the rate was below 40. His doctor was called in, and he was given morphine, with a certain measure of relief. The next day, when I saw him, the pulse rate was 36 and the rate at the apex was the same. The heart sounds were distant, and an apical systolic murmur was present. His blood pressure was 120/80 mm. Hg, and the respirations were rather rapid—36. He was cyanosed and suffering a great deal of respiratory distress. He was constantly clutching at his chest, which appeared to relieve him.

It seemed clear that he was suffering from heart-block. The suddenness of the onset and the urgency of the symptoms suggested a coronary occlusion of some kind, but the extreme slowing of the pulse indicated that a complete heart-block was present. Morphine was given at intervals; strychnine at his request, and oxygen and CO₂ from a Yandell-Henderson apparatus, were administered. Two electrocardiograms were taken the same afternoon and are here reproduced (Figs. 1 and 2). They show a four-to-one heart-block with idio-ventricular beats. Fig. 1 shows a normal QRS in Lead 1, but suggests a left bundle-branch block in Lead 3. Fig. 2 shows the left bundle-branch block well marked in Lead 1, with a very evident negative T-wave. There is complete dissociation of the auricles and the ventricles.

On the third day the patient had severe haemoptysis, accompanied by pain on the left side of the chest. At the same time his temperature, which had been normal, ran up to 101.6°. Friction subsequently developed over the area