first intention and the patient went home on April 8, a fortnight after the operation. He has been seen and examined several times since. In February, 1937, he served as second engineer on a trip to the East, and returned in June. He was then carefully examined by us, but nothing abnormal could be found in the chest or abdomen. In fact he felt and looked perfectly well.

The Specimen.—Before hardening in formalin this was six inches long and one inch in diameter, and slightly curved. It was quite solid and hard, with a small black spot representing the original lumen. The cross-section at the root was greyish pink, with no evidence of necrosis or suppuration—in fact, the only sign of inflammation was in the peritoneal coat, which was dark red. The specimen, which has been placed in the museum of the Royal College of Surgeons, is now smaller and the colour of the peritoneum has faded. Fig. 1 (see Plate) shows the size and shape after hardening and mounting. Fig. 2 gives the microscopical appearance of a section taken from the cut surface at the root. A few giant cells can be seen in a mass of lymphoid tissue. The various layers normally found in the wall of the appendix have been completely replaced by tuberculous tissue, the peritoneum alone having survived.

Comments

It is unnecessary to state that the exact nature of the change in the organ was not realized until the microscopical slides were examined. The specimen was unlike any appendix we had seen previously. It is obvious that the disease must have started months, if not years, before it was discovered. The absence of symptoms until within three hours of operation allowed us to remove the specimen before it had been damaged by inflammatory processes. We are of the opinion that the acute symptoms were caused by interference with the blood supply, as there was no visible lumen which could have been infected. At the operation the other abdominal organs were looked at in the ordinary way, but nothing abnormal was found. It must be admitted that small tuberculous lesions were not specially sought, as the diagnosis of tuberculosis had not then been suspected. But in view of the subsequent history of the patient it is reasonable to assume that there could not have been any well-developed tuberculous changes in the intestines or lungs. He returned to work in a few weeks, and has since enjoyed perfect health.

We are indebted to Mr. W. J. Salter, technician to Llandough Hospital, for the photographs.

REFERENCE

Pagel, W., and Weichherz, E. (1936). *British Medical Journal*, 2, 1305.

La Migraine Ophtalmique, by Drs. G. Renard and A. P. Mekdjian (the publishers are Masson of Paris and the price is 22 francs) gives a convenient summary of what is known of migraine, well set out under carefully designed subheadings. No unifying conception emerges, the authors stressing the multiplicity of associated disturbances, and dealing in particular with the allergic basis of the affection. One point of interest which they bring out is the disturbance in blood pressure, which they studied both from the general aspect and by ophthalmodynamometry of the retinal vessels. In general they found a tendency to hypotension during the attacks. Their thesis is illustrated by some case reports. They argue that hypotension can be taken as a diagnostic feature distinguishing true migraine from the transient blotting out of vision seen in elderly people with hypertension. They regard migraine as a condition which, though distinct from epilepsy, has in common with it the fact that there is a widespread, though ill-defined, underlying general departure from the normal, both psychological and somatic.

PELVIMETRY AND THE TRUE CONJUGATE

BY

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Every obstetrician must from time to time have been asked to see ante-natal cases in which a practitioner or midwife has felt on vaginal examination the sacral promontory; and there must be a number of women who are, because of this genuine misconception, submitted to unnecessary obstetric operations, with a diagnosis of gross pelvic contraction.

While not decrying the academic importance of attempting to measure the true conjugate clinically, it is the purpose of this brief communication to draw attention to the extreme rarity with which the sacral promontory can be palpated per vaginam; and my excuse for burdening an already adequate literature is that in the course of the last twelve months' ante-natal work three cases have been referred to the Middlesex Hospital Ante-natal Clinic labelled with such a phrase as: "Sacral promontory felt per vaginam: ? Caesarean section for pelvic contraction." All three were subsequently delivered by the natural passages. The only explanation that answers this fallacy is that the "sacral promontory" is in reality one of the lower bodies of the sacral vertebrae.

Three Factors to be Considered

Whether or no the sacral promontory is palpable depends on three factors: first, the length of the true conjugate; secondly, the effective length of the examining finger; and, thirdly, the rigidity of the vaginal introitus, which largely depends on the co-operative relaxation of the patient and her parity.

The anatomical true conjugate on the average female dry bony pelvis measures between 4.25 and 4.5 inches: the distance of the point behind the symphysis, from which the true conjugate is measured, to the subpubic angle is 1 inch on the bone, and must be nearly 50 per cent. greater than this when measured to the anterior lip of the vaginal introitus, in order to take into consideration the triangular ligament and its associated superficial small muscles and soft structures. This latter is greatest in the primipara and least in the multipara, for obvious reasons. Applying the well-known principles of geometry, the total effective length of an examining finger must be roughly 4.75 inches to feel the promontory in a normal pelvis.

The effective length of the examining finger, assuming that the index and middle fingers are used together, varies slightly with different surgeons, but surprisingly little. The determining factor here is largely the resistance encountered by the ring finger knuckle against the perineum, and to increase this useful length the perineum must be pushed upwards and backwards. Discounting this last factor, the uncompensated effective length of one's fingers is about three and a half inches. In a normal pelvis, therefore, with a true conjugate of 4.5 inches, the finger must be lengthened by depressing the perineum at least 1.25 inches before the promontory is reached, or, conversely, the pelvis must be foreshortened down to a true conjugate of 3.5 inches before the tip of the middle finger is likely to meet the posterior bony landmark. The exact location of the promontory is, moreover, a matter of difficulty, the manoeuvre of sliding the finger up the

bodies of the sacral vertebrae until it is felt to recede at the lumbo-sacral angle being plainly impossible in all but a grossly contracted pelvis. The error of position of the fingers can be readily proved by attempting internal pelvimetry with radiological control, using a piece of lead in the glove tips of the index and middle fingers. The lead shadow will be seen to be well wide of the mark, even when the perineum is forcibly depressed.

The third factor is the rigidity of the perineum, and, as the type of case in which we most earnestly desire to measure the true conjugate is usually a primipara, this assumes major importance and demands an anaesthetic of reasonable depth, at least, for its removal.

Lastly, the measurement is likely to be of most use in those cases of minor contraction, and these are the very cases where, for the reasons above enumerated, it is most difficult or impossible to obtain.

Conclusion

The conclusion is that the clinical measurement of the true conjugate is either impossible to measure, or inaccurate when measured, in all but cases of severe pelvic contraction; that a genuine attempt to measure it demands in a primipara a full anaesthetic; that the location of the sacral promontory requires practice and skill and is valueless unless performed by one well versed in pelvic examination; and that the measurement, when truthfully obtained, is open to considerable experimental error, in addition to errors due to the particular anatomy of a given patient, such as the depth and inclination of the symphysis.

WHY "POST-ANAESTHETIC" PULMONARY COMPLICATIONS?

BY

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That the great majority of "post-anaesthetic" pneumonias are "post-anaesthetic" in time relation only is no longer open to doubt. Inhalation anaesthesia does not predispose to pneumonia unless there is pre-existing lung disease. The typical post-anaesthetic pneumonia is the result of many minute pulmonary emboli from the operation site, and so is slightly commoner after local than after general anaesthesia. Its most important causes are trauma, mobility of the affected part, and sepsis (Cutler and Hunt, 1922). The control of "post-anaesthetic" pneumonia is thus mainly in the hands of the surgeon, not the anaesthetist. The term is therefore deplorable, because it diverts attention from the real causes and possibly suggests negligence on the part of the anaesthetist.

Aspiration of Foreign Matter in the Trachea

Very interesting observations on the ease with which matter such as blood and mucus enter the trachea have been made by M. C. Myerson (1922); he demonstrated it in 76 per cent. of patients who had had a tonsillectomy under light general anaesthesia, a cough reflex being present. Ochsner and Nesbit (1927) made an even more striking demonstration by placing lipiodol in the mouth of patients who had local anaesthesia of the tonsillar region. Radiographs taken later showed that lipiodol had

passed into the lungs in every case. The same thing happens readily in patients who have pharyngeal anaesthesia as a result of disease.

AN ILLUSTRATIVE CASE

Recently I made a necropsy on the body of a woman of 65 who drowned as a result of taking a cup of tonic (sanatogen) directly into the trachea. A small amount of fluid had entered her trachea on many previous occasions; she was known to have a syphilitic paralysis of the palate. Her body was slightly wasted and cyanosed; the blood was dark and fluid; she had a syphilitic aortitis and aortic regurgitation. There was no obvious pre-existing lung disease. The bronchi were filled with a whitish frothy emulsion, which was not readily distinguishable from pus by the naked eye. Microscopically its nature was clear, but a considerable amount of the sanatogen had been taken up by phagocytic cells. Routine sections showed no bronchopneumonia. There was no post-mortem evidence of the many small amounts of foreign matter which had entered her trachea on previous occasions.

It therefore seems certain that the cough reflex fails as a mechanism of expulsion under light anaesthesia; also that blood and mucus do not appear able to provoke pneumonia by themselves. It is not suggested that foreign matter can enter the lungs with impunity, for that is obviously not so. A typical example of this was a woman of 46 who was rescued from a suicidal attempt in a canal recently. She was resuscitated fairly easily, and was hysterical when admitted to the Derbyshire Royal Infirmary. For some hours she made good progress, but pulmonary oedema developed and she died about thirty hours after jumping into the canal. At necropsy I found a simple acute inflammation of the larynx and trachea, generalized pulmonary oedema, patches of red hepatization indicating early bronchopneumonia, no evidence of pre-existing lung disease, and a normal heart. In this case the cause is clear: it was neither "post-anaesthetic" nor due to embolism. Such cases are not rare, and they serve to emphasize the danger of regarding complacently the blood and mucus which enter the trachea far more readily than most of us realize.

General Anaesthesia as a Cause of Lung Abscess

The role of general anaesthesia in the aetiology of lung abscesses after tonsillectomy has been variously estimated. The usual belief is that the condition is commonest after open general anaesthesia and rare after intratracheal ether or other types of general anaesthetic in which special care is taken to prevent foreign matter entering the trachea. The average incidence is probably 1 in 2,500 tonsillectomies (Moore, 1922), but figures vary from 1 in 358 (Glowacki, 1923) to 0 in 20,000 (Lyman, 1923). A. L. Lockwood (1923) found that out of 208 lung abscesses 201 followed general anaesthesia. M. Manges (1916) noted that lung abscesses were commoner after operations with the patient upright. J. Maxwell (1934), in an exhaustive study, however, finds that the main association is sepsis in the operation field, and favours an embolic origin: such a view frees the anaesthetic from both direct and indirect blame. He also notes that lung abscesses are commoner after tonsillectomy than after dental operations. I have recently made two necropsies which throw some light on the problem.

TWO ILLUSTRATIVE CASES

Case 1.—An emaciated boy of 4 years had a lower left molar tooth removed on October 2. An abscess about five days old was present. The anaesthetic used was an ethyl chloride spray, and the boy did not lose consciousness. He