The only remark to be made about these cases is that patients suffering from ear trouble must be watched, whether the condition is just occasional earache or obvious chronic middle-ear disease; also any form of paralysis, whether it be facial, ocular, or of any of the limbs, must be looked out for. Any of these signs or symptoms should make one suspicious and decide to keep the individual under close observation.

The following account of a case of this type (symptomless) of brain abscess which the author met with a short time ago may be of interest.

Mrs. X, aged 66 years, was suddenly seized with acute pain in the right ear. Within twenty-four to thirty-six hours the pain gradually subsided, on the appearance of a thick discharge of pus from the affected ear, and the patient felt much better. No doctor was called, but the patient stayed in bed the next morning. Towards the afternoon her maid went to the bedroom and found her lying on the floor unconscious and clawing the right side of her head.

When I examined the patient I found the condition described above, together with incontinence, facial paralysis on the affected side, and slight paresis of the limbs of the opposite side. There was also evidence of meningeal and cerebral irritation. The temperature was 103.5° F. On examining the right ear I found no discharge, but a thickened and inflamed drum membrane with a small attic perforation. I diagnosed temporo-sphenoidal abscess and meningitis following middle-ear disease, and my colleague Mr. Austin McManus, who also examined her, concurred.

The prognosis was obviously hopeless, but we decided to operate immediately. I performed a radical mastoid operation, and found the following condition: The mastoid region was remarkable for having no cells; it was very sclerosed. In the antrum, which was very small and situated deeply, were a few granulations covered with pus. The tegmen was soft and spongy, as in acute osteomyelitis, and a small sinus led into the middle fossa. I removed the entire bony covering and exposed the dura. There was no extradural collection of pus, but the membrane was thickened, inflamed, and bulging under great tension.

I incised the dura, and immediately very thick and cheesy pus escaped, accompanied by blood-stained cerebro-spinal fluid. The amount of pus was not great, but the type was that found in chronic abscess; some granulations were present on the inner side of the opening in the dura. I carefully put a seeker through the opening made and met with no resistance, except in an upward direction. It seemed as if the entire temporal lobe was pushed away from the dura by the acute and profuse secretion of fluid following the meningeal inflammation. I put two fine rubber tubes in the dural opening to act as a drain, and also exposed the lateral sinus, which was quite healthy. I partly closed the original mastoid incision and drained the antrum through the external auditory meatus.

The patient lived about twenty-four hours after the operation without regaining consciousness.

Unfortunately I had no opportunity of making a post-mortem examination. The conclusion I came to was as follows: This patient had chronic suppuration in a dense acellular mastoid with a minute antrum. Instead of draining, as it normally should, into the tympanic cavity and thence through a perforation in the drum (which it eventually did, but too late), it burrowed its way gradually through the tegmen and dura, forming adhesions in its course, into the temporal lobe, there forming a small chronic abscess. Ultimately, for some unknown reason, an acute otitis media arose, and the pus under pressure caused a perforation of the membrana tympani, but spent most of its energy intracranially. Rupture of the abscess occurred, with subsequent meningitis, which would account for the symptoms and signs present when the patient was first seen.

I would add that the previous medical history of the lady was carefully inquired into and nobody had ever heard her complain of ear trouble; it was said that she always seemed very healthy and enjoyed life. The appearance of the drum membrane on examination would seem to bear out this history.

The case is of interest because (1) it shows that an intracranial condition may coexist with chronic mastoid disease and cause little or no symptoms; and (2) because it is in direct contrast to a case I reported in this *Journal* (October 19th, 1929, p. 717), in which obvious symptoms of temporo-sphenoidal abscess were present, but operation proved the condition to be one of simple mastoiditis.

TREATMENT OF BONE TUBERCULOSIS BY LARGE AMOUNTS OF VITAMINS A AND D.

BY

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OUR inability to arrest the spread of a tuberculous focus in bone, until considerable destruction has occurred, makes it worth while to investigate any therapeutic agent which seems likely to control this infection.

The results of Mellanby and Green,[1] which have demonstrated the potent action of vitamin A on bacterial infection in animals and man, especially their suggestive results obtained by treating cases of puerperal sepsis[2] with preparations very rich in vitamin A, made it seem probable that the value of cod-liver oil as a therapeutic remedy in tuberculosis is really due to its vitamin content. It appeared desirable, therefore, to see whether the exhibition of much larger doses of vitamin A than those usually given in cod-liver oil would hasten the healing process in this disease; also, since bone calcification, as shown by Mellanby, is controlled by the vitamin now known as vitamin D, and since the present investigations were made on bone tuberculosis, it was decided to increase this vitamin in the diet as well as vitamin A, although not to such a great extent. It has been mentioned in a previous communication[3] that the giving of extra amounts of vitamin D alone (in the form of irradiated ergosterol—radiostol) did not appear to increase the rapidity of recalcification of bones affected with tuberculosis to any marked extent.

Two preparations of different strengths, both containing a large quantity of vitamin A and one of them having a high vitamin D content in addition, have been used in this investigation. Both were kindly supplied by the British Drug Houses Ltd.

Preparation No. 1.—The vitamin A of this preparation gives 150 blue with the antimony trichloride test, and has about twenty times the vitamin A potency of cod-liver oil. It also contains 10,000 antirachitic units per c.cm. (Coward).

Preparation No. 2.—This has double the vitamin A content of preparation No. 1 (that is, 300 blue), but only 100 antirachitic units per c.cm.

Forty-three patients were given large doses of these preparations as described below, while 35 patients were given 10 c.cm. of cod-liver oil daily. These latter were intended to act as controls, and were selected because they had similar degrees of infection and bone invasion to certain patients in the other group of cases. The patients included in these tests were of varying ages—from 3 to 15 years—and in both groups there were cases with early disease, with acute rapidly progressing disease, with chronic slowly increasing disease, and with disease in which recalcification of bone was commencing.

Of the 43 patients, 15 were given an average dose of 7.8 c.cm. of preparation No. 1 daily for an average period of 3.5 months, and were then given an average dose of 9.4 c.cm. of preparation No. 2 for a further average period of 3.4 months. The remaining 28 patients were given an average dose of 9.6 c.cm. of preparation No. 2 for an average period of 4.1 months.

The 35 control patients were given 10 c.cm. of cod-liver oil daily during the period of investigation. It may be well to mention that the patients of all three groups had received cod-liver oil daily before the actual test started. It will be noticed that all the patients, including the controls, got a fair supply of vitamin A and D, but that those receiving the special vitamin preparations Nos. 1 and 2 had very much more vitamin A than the others.

The clinical condition of the patients in the different groups and the *x*-ray appearance of their affected bones had been carefully recorded some months before the investigation; this was done again both at the commencement and at the end of the test period. An examination of the accompanying table will show that of the 43 patients having the vitamin preparations 32 were improved clinically, and 11 were not; that in 25 of these 43 cases the *x* rays showed arrest of disease or increased calcification, while in 18 there

was no evidence of such improvement. Of the 35 patients taking cod-liver oil, 25 were clinically improved, and 10 were not. The skiagrams of 18 of these 35 indicated progress towards cure, while those of 17 did not.

	Cases receiving much Vitamin A and D.		Cases receiving moderate amounts Vitamin A and D (Cod-liver Oil).	
	Number.	Percentage.	Number.	Percentage.
Total cases	43	—	35	—
Clinically improved ...	32	74.4	25	71.4
Not clinically improved ...	11	25.6	10	28.6
Increased calcification in radi gram	25	58.1	18	51.4
No increase in calcification in radiogram	18	41.9	17	48.6

If one considers the impossibility of selecting exactly similar patients, and of estimating the exact degree of activity of disease, it will be appreciated that the patients having the concentrated vitamins and those taking cod-liver oil showed about the same amount of progress. Therefore, for the types of patients described, and with the dosages and under the other conditions mentioned, the use of preparations containing large amounts of vitamins A and D does not appear to be of more curative value than that of cod-liver oil.

REFERENCES.

[1] Mellanby, E., and Green, H. N.: *British Medical Journal*, 1928, ii, 691.
[2] Idem: Ibid., 1929, i, 984.
[3] Pattison, C. Lee: Ibid., 1929, i, 419.

THE TREATMENT OF ACUTE PUERPERAL INVERSION.

BY

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DRUMMOND MAXWELL stated in 1915 that "all cases of acute puerperal inversion warrant a clinical record for several reasons." Among the reasons he gave were: (1) that no case had been admitted to the London Hospital for the previous fifteen years; (2) the very divergent views on treatment; and (3) the tendency in modern literature to invoke surgical rather than classical treatment.[1]

The incidence of acute puerperal inversion is computed variously by different authors, and no two authors agree; T. W. Eden puts the figure at one inversion in 180,000 to 200,000 labours. All authors are agreed, however, that accuracy cannot be arrived at, since practically all cases occur in general practice, and general practitioners as a whole are not prone to put their cases on record.

In the classical type of case the uterus is found to be inverted with the placenta centrally attached; this occurs immediately or a few minutes after the child is born. In the case here reported the placenta came away fifteen minutes after delivery, but it was not till seven days later that acute inversion with prolapse occurred. Fox, in 1924, recorded a case where acute inversion with prolapse developed fourteen hours after delivery.[2] This is the only case of some delay before the onset of acute puerperal inversion that I have come across in the literature.

On May 14th I was called to a Poor Law institution to attend a primipara, aged 41, who was in labour. The head had been on the perineum for over an hour, and in spite of good labour pains no progress was being made. Instrumental delivery under an anaesthetic was satisfactorily accomplished, and on expression of the placenta a large retro-placental clot came away. A sharp post-partum haemorrhage ensued, and this was speedily controlled by the injection of 1 c.cm. of pitoxylin. The placenta on examination was found to be intact, with a dark area where the retro-placental clot had formed. A perineal tear was sutured with three stitches, and half-drachm doses of liq. ext. ergot were prescribed for the next few days.

The height of the uterus was charted daily by the midwives of the institution. On the first day after delivery it was 7 in. above the symphysis; the second day 6 in., the third day 5 in., the fourth day 4½ in., the fifth day 4 in., and on the sixth day it was missing. On the seventh day, while the patient was using a bed-pan, the uterus inverted completely, and prolapsed outside the vulva. I arrived to find a flaccid, inverted uterus, about the size of a large coco-nut, occupying the perineal area. There had been much haemorrhage (about 2 pints), but the matron and midwife had applied towels rung out in hot water at a temperature of 120° F., and the bleeding was now being kept well in check by such applications. There was a moderate degree of shock, and the pulse was 130.

Under chloroform anaesthesia the uterus, which had been well in contact with faecal matter in the bed-pan, was swabbed and douched with 2 per cent. iodine solution at 120° F., and reduction was attempted. I squeezed the mass with both hands till it was reduced to the size of a foetal head, blood spurting on pressure from various points, but found it very difficult to force the mass through the vaginal entrance; it will be recalled that the perineum had been well sutured. Adopting the same methods, however, my colleague Dr. Fraser succeeded in forcing the mass through the vaginal entrance. Once this obstacle was overcome the uterus returned quite easily to its proper position. So much force was employed to overcome the constriction at the introitus vaginae that the perineum was ruptured again at the original site.

An intrauterine douche of 2½ pints of 2 per cent. iodine at 120° F. was instantly given, and the haemorrhage ceased. With the idea of preventing recurrence of the inversion and haemorrhage the vaginal vault was next plugged with sterile cyanide gauze, well around the fornices, counter-pressure being applied with an abdominal pad and binder. The usual treatment for moderate shock and loss of blood followed, but saline infusions were not given.

On recovering from the anaesthetic the patient was definitely comfortable, in contrast with her distress before the anaesthesia and reduction. The temperature was 103° F. and the pulse 126, but the temperature fell next day to 100° F., and the cyanide gauze was removed after twenty-four hours. There was no haemorrhage. Neither ergot nor pituitrin was ordered, for fear of inducing return of the inversion. Since septic endometritis was inevitable, 50 c.cm. of a polyvalent antistreptococcal serum was injected subcutaneously. There was a moderate sapraemia for twelve days, but otherwise recovery was uneventful. A subsequent vaginal examination disclosed the uterus to be situated in the normal position. The patient is now very well, and the perineum will be repaired in the near future.

In practically all cases of acute puerperal inversion of the classical type the placenta has been found to be centrally placed and somewhat adherent. The tension on the cord caused by a rapidly expelled foetus or the accoucheur has started the process of inversion, and the uterus proceeds to expel itself, using the placenta as a foreign body. In some cases, however, the cord has been too long to render tension by the projected foetus a likely cause, and the midwife has emphatically denied pulling on the cord. It must here be assumed that the uterus after evacuating the foetus treats the centrally placed placenta as another foreign body, and promptly proceeds to expel it. Credé's method of expression of the placenta, massage of the uterus, denting of the fundus when measuring the height of the uterus, and straining at evacuation, have all been blamed as causes of acute inversion.

In treatment various points have to be considered. Study of the cases reveals that shock and haemorrhage usually occur in inverse ratio; the greater the shock the less the haemorrhage. This is easily comprehensible as the blood pressure falls in proportion to the degree of shock. Miles Phillips in 1911 conclusively showed that in profound shock treatment should be directed to restoring the patient before attempting to replace the uterus.[3] Immediate replacement of the uterus in a patient moribund from shock has only hastened the onset of death.[4] In three cases of profound shock, Miles Phillips waited two hours, five hours, and twenty hours respectively before attempting replacement, the patients in the meanwhile being treated with saline infusions, pituitary extract, and morphine. Hellier in 1912 returned an inverted uterus after four days.[5] In a few cases where there was little or no shock immediate replacement was followed by recovery.[6 7 8]

The importance of reduction under an anaesthetic has not been duly emphasized in the textbooks;[9 10 11] in my opinion it should always be conducted under anaesthesia. The process of inversion is accompanied by acute pain and sometimes shock.[12] So also is the process of reduction