It was suggested by Iwanoff (1898), and supported by Meyer and Kitai (1924), that the origin of the aberrant endometrial tissue was a serosal metaplasia. This was based on the fact that the entire epithelial apparatus of the female genital tract (endometrium, germinal epithelium of the surface of the ovary, etc.) is derived from the primitive coelomic epithelium of the urogenital fold and is a mere modification of the peritoneum. According to this theory, under the influence of ovarian hormonal stimulation the pelvic peritoneum forms tubular invaginations which sink into the subjacent tissue, and the flat endothelium of the involved peritoneum becomes columnar, thus producing an adenomatous condition.

The hypothesis of serosal metaplasia has received influential support, especially for the endometrial cysts of the ovary and endometriosis of the umbilicus, laparotomy scars, uterine ligaments, intestine, and recto-vaginal septum.

An explanation of the stimulus of metaplasia may be found, in the above case, in a study of the behaviour of epithelium in the presence of inflammation. Inflammation beneath a mucous or serous surface produces an infiltrative proliferation in the adjacent epithelium. This epithelial proliferation is at first a protective process, but it can overstep the limit of physiological hyperplasia by producing epithelial tubules which have all the appearances of glandular invasion. Peritoneal endothelium behaves in the same way, and in the present case, in which a chronic intussusception probably gave rise to such a stimulus, the glandular elements are seen to reproduce the exact structure of normal endometrial glands and stimulate a surrounding stroma which is an exact replica of endometrial stroma. Furthermore, these areas of typical endometrial tissue are seen in the sections to lie in immediate apposition to the hyperplastic tubules which are shown to be derived from the peritoneal endothelium.

It would seem that reversions of the epithelium under inflammatory, and perhaps ovarian, stimuli are possible in the adult peritoneum, and that the embryonic cells can reproduce endometrial structure.

Summary

A case of intussusception, with tissue showing all the appearances of endometrium situated at the apex of the entering ileum, is presented.

The microscopical appearances are suggestive of a metaplastic origin of the endometrial tissue and may be held to support the observations of Meyer and Kitai (1924), Novak (1926, 1947), Moench (1929), and others.

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NEONATAL BACT. COLI MENINGITIS AFTER PROLONGED LABOUR

BY

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It is well known that *Bacterium coli* meningitis is relatively commoner in the newborn than in childhood or later life (Barron, 1918; Cruickshank, 1930; Craig, 1936; Rauch and Krinsky, 1940; Barrett *et al.*, 1942). The explanation given is that the newborn infant has not developed adequate resistance against the organism, although in later life it carries the same organism about in large numbers in relative safety (Cooke and Bell, 1922).

The route taken by the infecting organism has been the subject of some speculation, and numerous suggestions have been made. It is generally agreed, however, that the meninges are more commonly infected via the blood stream than by direct spread from adjacent areas. To support this contention Fothergill and Sweet (1933) demonstrated a *Bact. coli* bacteriaemia at the same time that *Bact. coli* were found in the cerebrospinal fluid.

The original portal of entry is difficult to determine, but the large number of suggestions made, incriminating widely separated sources, indicate that infection may enter at any of many sites. The following portals of entry have been mentioned: middle ear (Scherer, 1894; Barron, 1918; Leathart, 1932; Craig, 1936), gut (Cooke and Bell, 1922; Barrett *et al.*, 1942; Ravid, 1935), lungs (Aschoff, 1897; Goldreich, 1902; Herrman, 1915), genito-urinary tract (Neal, 1926; Köplik, 1916), spina bifida (Forbes, quoted by Rauch and Krinsky, 1940), umbilicus (Hinsdale, 1899), and morbid conditions of skin, scalp, mucosae, and subcutaneous tissues (Craig, 1936). In addition transplacental bacteriaemia, possibly arising from maternal pyelitis, has been suggested by Jahkola (1935), Karplus (1927), and Athenstaedt (1933). Aspiration of liquor infected with *Bact. coli*, causing a bronchopneumonia followed by bloodstream spread to the meninges, was suggested by Goldreich in his case. Aschoff held similar views. And, it might be noted, F. J. Browne (1921) considers aspiration of infected liquor a cause of bronchopneumonia.

In the following case it would seem that this was also the method and route of infection. The foetal head lay in the maternal pelvis, deeply engaged, for at least four days, and during the latter two days the cervix was between half and full dilatation. Further, both mother and child showed clinical signs of infection three days after delivery —the mother with *Bact. coli* pyelitis and endometritis and the child with fatal *Bact. coli* meningitis and bronchopneumonia.

Some previous cases have been reported in which the same organism was found in the maternal urine and lochia as was found in the child's cerebrospinal fluid (Jahkola, 1935; Trillat and Notter, 1939; Davis and Fernando, 1935).

Case History

A primipara aged 25 had labour surgically induced by a Drew Smythe catheter when 38 weeks pregnant. The indication was pre-eclamptic toxaemia with a rising blood pressure.

Labour started next day, but it was soon clear that primary uterine inertia existed. Four days later she had two eclamptic fits. She was then given 1/4 gr. (16 mg.) of morphine and sent into hospital.

On admission at 6 a.m. on Aug. 28, 1947, treatment based on Stroganoff's principles was immediately started. In the first 24 hours she received 1¼ gr. (81 mg.) of morphine and 6 gr. (0.4 g.) of sodium phenobarbitone. She had a fit when being put to bed and another two hours later, but the rest of the day she passed in a semicomatose condition. The foetus was small, with its head deeply engaged in the maternal pelvis. There were no uterine contractions at this stage and the foetal heart was easily audible.

The patient slept deeply throughout Aug. 29. Only 1/6 gr. (11 mg.) of morphine was given. A specimen of urine showed : specific gravity, 1010 ; alkaline reaction ; albumin, 0.6% ; sugar, *nil* ; scanty pus cells ; a few granular and hyaline casts ; sterile culture. At 2.30 a.m. on Aug. 30 the cervix was fully dilated, and at 4 a.m. the child, a female, was delivered by forceps. It was somewhat narcotized, but breathed and cried satisfactorily after receiving 1.7 ml. of nikethamide and 10 mg. of vitamin K intramuscularly. Both mother and child were as well as could be expected for the remainder of the day and throughout the following day.

On Sept. 1 the mother's temperature rose to 100.4° F. (38° C.) and clinical examination suggested acute pyelitis and endometritis. A catheter specimen of urine showed: specific gravity, 1024 ; strongly acid reaction ; albumin, 0.85% ; sugar, *nil* ; few pus cells, numerous organisms ; many granular and fatty casts ; crystals, *nil* ; culture, coliform bacilli. A high vaginal swab showed : direct smear—scanty pus cells, many coliform bacilli ; culture, coliform bacilli. Penicillin and sulphadiazine therapy was instituted, and after 24 hours she was apyrexial. The remainder of the puerperium was uneventful.

The Child

At birth the child was given nikethamide and vitamin K and was not disturbed except to be given feeds of glucose-water during the first 48 hours of its life. On the morning of the third day the child was fretful and restless and refused its feed of water. Some stiffness of the neck and head retraction were noticed. The temperature was 100° F. (37.8° C.). Cerebral haemorrhage was suspected, and vitamin K and 1 gr. (65 mg.) of chloral were given.

The child's condition deteriorated. It developed twitchings of the face and limbs, nystagmus, and, later, convulsions which became more frequent until they were almost continuous. Head retraction became more marked and the child vomited once, bringing up blood-stained fluid. Sodium phenobarbitone, 1/8 gr. (8 mg.), had no apparent effect.

Before death occurred moist sounds and patchy consolidation at the bases of the lungs were noted and penicillin, 100,000 units, was given. But the child became progressively dyspnoeic and cyanosed, and died at 6.20 p.m. on Sept. 1.

Post-mortem Report.—"External: post-mortem lividity is very marked. Head : Skull-cap and dura mater are normal ; there is no evidence of injury to the brain substance or venous sinuses, but there is a well-marked acute purulent meningitis over the base of the brain. Thorax : The pericardial sac contains a large excess of clear yellow fluid ; myocardium shows marked toxic changes ; pleural sacs are dry ; a copious fibrinopurulent exudate is present over the lower lobes of both lungs, and these show bronchopneumonic consolidation. Abdomen : The peritoneal sac is normal ; liver, spleen, and kidneys show toxic changes ; other organs show nothing of note. Cause of death: Acute purulent meningitis ; bronchopneumonia. Culture of pus from the base of the brain grew coliform bacilli, and biochemical investigations gave typical reaction of coliform bacilli of intestinal origin."

Discussion

When the membranes are ruptured the way is open for the liquor amnii and the uterine cavity to become infected. This we know to be rare in normal labour or where the induction–delivery interval is short. But when there is delay in the onset of labour or a prolongation from any cause infection of the liquor becomes an important consideration. And the longer labour continues, with or without vaginal examinations, the greater the degree of infection.

Eventually the liquor becomes frankly purulent and may lead to pyrexia and other signs of toxic absorption in the mother. The risks of caesarean section are greatly enhanced by the possibility of peritoneal and wound infection. At the same time the foetus is surrounded by a septic fluid which enters the mouth, the nose, the nasopharynx, and remains there to be inhaled into the lungs at the child's first breath, or to track down the Eustachian tubes to the middle ear.

Davis and Potter (1946) injected thorotrast into the amniotic fluid before hysterotomy or caesarean section and found it widely distributed throughout the lung fields of the foetuses. Even if this does not prove that intrauterine respiration occurs, it does indicate that amniotic fluid enters the lungs of the foetus, and if the fluid is infected serious disease of the foetus may be set up *in utero*—e.g., bronchopneumonia.

Bact. coli is one of the commonest of the bacteria likely to be present in such a situation, and it is not surprising that this organism is responsible for the majority of meningeal infections in the newborn.

Instrumental delivery, with more or less vaginal manipulation and trauma, is relatively often required to terminate a prolonged labour, and this will nearly always produce minor abrasions and bruises on the child's scalp, which will be immediately subject to infection. Further, the child in such cases is likely to be born with some degree of cerebral oedema and congestion associated with atelectasis, which in turn favours lung infections.

The possibility of serious neonatal infection must therefore be considered in the management of prolonged labour. And where there is evidence of intrauterine infection, either in the shape of a maternal pyrexia without other cause or in the discharge of purulent liquor from the vagina, chemotherapy should be started, and the case assessed with a view to the earliest possible termination of labour. Woltz and Zintel (1945) have demonstrated that penicillin reaches the foetal blood and the liquor in adequate therapeutic quantities, and recommend its use prophylactically in cases of uterine inertia or unduly protracted labour.

Diagnosis and Treatment.—The diagnosis and treatment of *Bact. coli* meningitis is the province of the paediatrician, but it might be stressed that after a prolonged labour the attendant should be on the watch for early signs of infection of the child and chemotherapy should be started. Sulphapyridine, sulphadiazine, and sulphathiazole alone or in combination with penicillin have been recommended, and Alexander (1946) has had success with streptomycin in one case. Diagnosis is difficult, and it is usually necessary to examine the cerebrospinal fluid in order to differentiate between cerebral haemorrhage and meningitis.

Summary

A short review of the literature is made with particular reference to the portals of entry of *Bact. coli* infection which leads to *Bact. coli* meningitis in the newborn.

The history of a fatal case of *Bact. coli* meningitis in an infant 3 days old is given. The mother had intra-partum eclampsia and prolonged labour, and on the third day of the puerperium developed *Bact. coli* pyelitis and endometritis.

The dangers of prolonged labour, from the point of view of the child, are stressed.

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CERVICAL SYMPATHETIC PARALYSIS

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The effects of unilateral paralysis of the cervical sympathetic chain, almost invariably referred to in standard textbooks as Horner's syndrome, are generally given as ptosis, enophthalmos, pupillary constriction, and anidrosis of the face on the affected side. This description of the syndrome not only is inaccurate and incomplete but differs from that published in 1869 by Horner himself. Further, accounts of sympathetic paralysis were published by many earlier workers, and it therefore seems justifiable to review the whole status of the eponym.

Historical

Pourfour du Petit (1727) recognized that injury to the sympathetic chain in the neck could cause pupillary changes. In many domestic animals he noted myosis and partial closure of the third eyelid at the inner canthus. His results were confirmed by Mollinelli in 1755 and by many others. Similar effects after section of the trigeminal nerve near the Gasserian ganglion were observed by Fodera (1823), Magendie (1824), and Sir Charles Bell.

Budge and Waller in 1841 demonstrated that the sympathetic supply to the iris has its origin in the anterior and middle columns of the spinal cord from the sixth cervical to the fourth thoracic segment, passing via the spinal nerves to the cervical sympathetic chain and thence to the first division of the trigeminal nerve. Brown-Séquard and Bernard in 1854 noted that section of the sympathetic chain caused pupillary contraction, closure of the eyelids, and increased vascularity and temperature on the affected side, all these changes being reversed by galvanic stimulation of the upper divided end of the nerve.

Despite all these observations, clinical descriptions of such conditions occurring in the course of natural diseases do not appear in the earlier literature. In 1809 Sir Astley Cooper, in a case of aneurysm of the internal carotid artery, noted ptosis on the affected side together with "a feeling of hot and cold in the ear." He did not record whether or not these signs disappeared after he had cured the aneurysm by ligation of the common carotid artery, nor did he remark on the state of the pupils, an omission which is understandable when it is realized that the pupillary constriction of opium-poisoning was not recognized until Kinnis described it in 1818. Coates, in 1822, recorded a case in which successful ligation of the common carotid artery for an aneurysm was followed by restoration to normal of a pupil which had been markedly constricted. Sir Benjamin Brodie (1828) noted bilateral pupillary constriction in a man with fracture-dislocation of the cervico-dorsal spine. Willebrandt (1854) recorded similar pupillary constriction in a case of cervical tumour, and Gairdner (1855) described an instance of aortic aneurysm accompanied by eye changes which he rightly regarded as similar to those produced experimentally by section of the cervical sympathetic chain.

Notwithstanding the value of these earlier contributions it is probable that much of the credit for the clinical evaluation of the syndrome produced by cervical sympathetic paralysis should be accorded to J. W. Ogle, physician to St. George's Hospital, London. In his carefully reasoned publication (Ogle, 1858) he pointed out some of the fallacies in the application to man of the results of animal experiments, summarized the previous literature, and collected and reviewed a series of 27 cases. In these, unilateral myosis and ptosis were sometimes associated with vascular and temperature changes, and the interference with the cervical sympathetic chain was the result of intrathoracic aneurysms, cervical lymphadenopathy, or spinal injury. He recorded one case in which an acute abscess of the neck was accompanied by dilatation of the pupil, which returned to normal after incision of the abscess. The role of the cervical sympathetic seems to have been well recognized by this time. Weir Mitchell *et al.* (1864) described a patient in whom a gunshot wound of the neck was followed by unilateral pupillary constriction, narrowing of the palpebral fissure, and very pronounced vasodilatation of the corresponding side of the face and neck, ending abruptly at the midline. These changes were confidently ascribed to injury of the cervical sympathetic chain.

The Work of Horner

Horner (1869) based his observations on one case, that of a woman aged 40, who, six weeks after parturition, developed right-sided myosis, ptosis, and diminished ocular tension without evidence of eye disease or of trigeminal paralysis. Although remarking that some slight enophthalmos was present, Horner regarded this as of little importance, concentrating more on the study of the vascular effects. He noted that the affected side of the face was redder and distinctly warmer than the other and that it did not perspire. He measured the temperature on both sides of the face, finding a difference of 5 ° C. under normal conditions. Bandaging the face resulted in an increase in temperature on the normal side so that the difference was reduced to about 1 ° C. The vasodilatation of the face when warmer was, he noted, accompanied by a corresponding dilatation of the retinal veins. Atropine enlarged the pupil but slowly, irregularly, and to a lesser extent than on the normal side. Although recognizing that cervical sympathetic paralysis was the cause of the signs he expressed no views as to the fundamental pathology of the condition.