floor and cervix descends after a short interval, and the condition of prolapse is not improved.

For *cystocele* the operation consists in: (1) removing an adequate portion of the anterior vaginal wall; (2) plecting the base of the bladder or the platysma layer covering it; (3) approximating the two edges of the vaginal mucous membrane by mattress and continuous sutures.

For *rectocele*, posterior colpoperineorrhaphy is performed as follows: (1) remove a large triangular area of posterior vaginal mucous membrane; (2) bring levatores ani and perineal muscles together by interrupted sutures of strong chromic catgut; (3) approximate edges of vaginal mucous membrane; (4) approximate edges of skin of perineum. The cervix uteri is sometimes amputated at the time of performing anterior colporrhaphy.

The pelvic floor having now been restored to its *status quo ante*, suspension of the uterus by laparotomy may be done if necessary. This is often unnecessary in old people, in whom it is permissible very nearly to close up the vagina by the plastic operation. In younger people, who must be left nubile, the vagina must not be closed up too much, and ventral suspension is often required as a safeguard against recurrence. The best operation is suspension by attaching the round ligaments in a special manner to the midline of the anterior rectus sheath—generally called the Gilliam type of operation. I have yet to see a recurrence after this operation when performed for retroversion alone or for retroversion with prolapse, when it is preceded by a plastic operation on the pelvic floor. Its anatomical soundness is shown by the subsequent occurrence of pregnancy and labour without risk.

These operations—anterior and posterior colporrhaphy and ventral suspension—are somewhat intricate and difficult to learn, but when proficiency is acquired they give a 99 per cent. cure. Further, the worse the prolapse the less difficult the operation.

Hysterectomy is no cure for prolapse, and should never be performed unless the uterus is diseased, for it is difficult to suspend the upper vagina or cervix adequately when the uterus has been removed.

Ventral Fixation—in which the body of the uterus is stitied to the back of the abdominal wall—is not so anatomically correct as suspension by the round ligaments, and is now but rarely performed.

For severe congenital prolapse only extensive and complete operations are successful.

The Bladder and Urine in Prolapse.—It is unusual to find residual urine in prolapse, but occasionally *B. coli* is present, causing irritability of the bladder or dysuria. In such cases it is advisable to get rid of the *B. coli* before operating.

Prolapse associated with pregnancy and labour is a troublesome complication, as it is difficult to keep the uterus up during the first four months. A modified cup-and-stem pessary will help, but a good deal of rest will be essential. In labour, great care is required against sepsis, since the cervix is low down and may even come outside the vulva during delivery.

Malignant Diseases and Prolapse.—Carcinoma of the cervix or body of the uterus is but rarely associated with prolapse. When bleeding from the cervix occurs with prolapse it is generally due to traumatic ulceration.

CHILD-BEARING AFTER OPERATIONS FOR PROLAPSE

It is not advisable for a woman to have another child after colporrhaphy for prolapse has been done, as the vagina will be lacerated and prolapse recur. It is therefore quite reasonable to consider sterilization at the time

of operation, which can easily be carried out by ligature of the Fallopian tubes. My own opinion is that it is definitely indicated when two living children have been born and prolapse of the second degree is present. The patient and her husband should hear the facts and make the decision.

SUMMARY

In conclusion, I will summarize the points I have tried to emphasize in this short paper.

1. Simple retroversion of the uterus in single women or in women who have not borne a child should be left alone, as it rarely causes symptoms.

2. Prolapse of the uterus and pelvic floor is preventable to some extent by care and treatment during pregnancy, labour, and the puerperium.

3. The treatment of prolapse is by operation and not by pessaries.

4. Sterilization at the time of operation is indicated in certain cases.

A CASE OF CHRONIC MENINGOCOCCIC SEPTICAEMIA WITH BACTERIAL ENDOCARDITIS

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Cases of chronic meningococcic septicaemia are sufficiently few in the British literature to warrant this report. G. Lovell Gulland and W. R. Logan¹ described in 1925 a case of prolonged fever in which, towards its termination, Gram-negative cocci were obtained by a blood culture. A meningitis developed, and a meningococcus was isolated from the cerebro-spinal fluid, which showed a large white cell count. At the post-mortem examination the only pathological condition of note was found in the leptomeninges. J. C. Kennedy² in 1926 described four cases in soldiers, in two of which meningitis supervened, and states that from 1919 to 1926 he could only find two cases in the British literature. The American literature^{3, 4} gives fourteen cases, with three deaths. In Dock's⁵ case the post-mortem appearances were those of meningitis, which developed after seven months of fever; Gram-negative diplococci were obtained in blood culture, and, after death, from the spleen and spinal fluid. According to Marlow³ the literature, other than American, contains approximately 110 cases.

For the clinical notes in this case I am indebted to Dr. J. C. Middleton, consulting physician to the Western District Hospital, Glasgow. During his absence on vacation the case was under the care of Dr. Andrew Allison, who sent in the blood for culture and the other material for examination.

HISTORY OF CASE

A married woman, aged 41 years, a rubber worker, was admitted to the hospital on March 8th, 1929, complaining of headache and sickness of two days' duration. The condition came on suddenly; she felt shivery; pain developed in the back; she was sick, and severe headache followed. On admission nothing abnormal was found in the heart or lungs or nervous system; the urine was seen to contain a little albumin; nothing abnormal was found in the abdomen. She became very drowsy, headache continued to be severe, and she was persistently fevered. Pus appeared in the urine, and in the absence of definite head signs the case was regarded as one of pyelitis. She was put on alkalis, and the temperature gradually came

down, but she remained drowsy and apathetic, and complained of headache. She refused to be transferred to the parish, and left hospital on May 7th. On June 20th she was readmitted.

During the time she was out of hospital she suffered from weakness and loss of energy; on June 13th she developed a troublesome cough, and applied for readmission on this account.

On readmission she was dull and listless; the temperature was 102° F. She was flushed and looked ill. There was some bronchitis present, and a systolic murmur was audible over the mitral area. The spleen was palpable. The urine contained no albumin. The pupils reacted to light, the right being larger than the left. She did not complain of headache, and there was no head retraction or rigidity of the neck. Kernig's sign was not present. Leucocyte count, 16,000 per c.mm. In a few days the cough had practically disappeared and the bronchitis had cleared up, but fever persisted. The patient complained only of lack of energy, and took no interest in her surroundings. She was perfectly clear mentally. Vomiting commenced on June 26th, and persisted. She became drowsy during the day, and this drowsiness was marked by June 28th. On July 11th she was incontinent, gradually became emaciated, and got weaker, but remained mentally clear until a few days before death, though the drowsiness increased. Fever was present throughout the illness, but there was no hyperpyrexia before death. From June 20th to 30th the temperature was irregular and remittent in type; on June 22nd and 23rd it was almost normal. From July 1st to 10th it was intermittent in type, the temperature falling below normal on every day except one, and rising to 101° up to 104° in the course of the day. From that date, while being irregularly intermittent in type, it never rose above 100.5°. During the last few days of life there was practically no pyrexia. She died on July 27th.

PATHOLOGICAL INVESTIGATIONS

The Wassermann reaction was negative. From the blood received on July 6th a Gram-negative coccus was obtained in pure culture after seventy-two hours' incubation. This coccus varied in size and intensity of staining, did not grow in subcultures between 18° and 23° C., and, while growing on agar media in first transplant, did not grow on that medium in subsequent subcultures. Growth was continued on blood-agar. It produced acid readily on maltose media—solid and fluid—and also, but later and more faintly, in glucose media. It did not acidify saccharose. Through the kindness of the public health laboratory of Glasgow, typing serums for the meningococci, obtained by them from inoculations with type cultures from the national collection, were received. On three occasions, shortly after the isolation of the organism, agglutination tests were carried out by Dryer's method. The result, I think, places the organism in the closely allied classes of Types I and III, but I was unable to classify it further.

The third test was as follows. A growth of the organism after forty-eight hours' incubation on tryptic agar was emulsified and diluted down by the aid of Brown's opacity tubes to 150 millions per c.cm. This was heated for one hour between 56° and 58° C. With Dryer's method, and incubation at 52° C. for twenty-four hours, the readings gave a good agglutination as follows:

Type I.—1 in 250 dilution (the titre of the serum said to be 1 in 800).*

Type II.—1 in 83 dilution (the titre of the serum said to be 1 in 800).

Type III.—1 in 250 dilution (the titre of the serum said to be 1 in 400).

Type IV.—Only a partial agglutination, 1 in 25 dilution.

This organism was agglutinated by the patient's own serum in 1 in 125 dilution, a control serum showing no trace of agglutination. The cultural and serological tests identify the organism as the meningococcus, and as such it was reported. The fermentation of maltose and the

high agglutination titre with anti-meningococcal serum distinguish it from the gonococcus, which is a commoner cause of endocarditis.

The Widal reactions against *B. typhosus*, *B. paratyphosus* A and B, and the Salmonella group, Mutton and Newport, and *B. enteritidis* (Gaertner) were negative, as also against *B. abortus* and *M. melitensis*. The patient's serum agglutinated *B. paratyphosus* C in 1 in 125 dilution, being 25 agglutination units (M.R.C.). A repeated examination of the serum taken several days later showed no change in titre. I have had on a previous occasion a high agglutination titre of the paratyphoid C emulsion in a case of a man who died from cancer, and I concluded that *B. paratyphosus* C was not the active agent in this infection. No pathogenic germs were isolated from the stool or urine. The urine contained pus, a coli group organism, and a Gram-positive coccus. The cerebro-spinal fluid received on July 16th showed a normal cell count—3 leucocytes per c.mm. (an average of three counts); it should be observed that the fluid had stood overnight. There was no increase of globulin. Fehling's solution was normally reduced. Cultures were made. I regret I have no note as to what developed, but that fact and my memory point to them as having been sterile.

TREATMENT

Specific serum treatment was commenced on July 13th, 25 c.cm. of anti-meningococcal serum being given daily (intramuscularly) till the 19th, when 50 c.cm. were given; this produced ecchymosis. The serum treatment was discontinued on July 22nd. The total quantity injected was 275 c.cm. No benefit accrued.

POST-MORTEM FINDINGS

The necropsy was performed thirty-three hours after death. The findings, naked-eye and microscopical, will be given in some detail, as the lesions were numerous and interesting. Over the surface of the neck and chest there was a profuse rash consisting of minute petechiae, and these were also present in the buccal mucosa.

Heart.—There was some clear fluid in the pericardium. Under the visceral layer overlying the right ventricle petechiae were present. Petechiae were also plentiful under the endocardium of the right ventricle. A soft white friable vegetation of considerable size was attached to the left cusp of the mitral valve. This consisted, on microscopical examination, of a pale thrombus, with leucocytes and masses of Gram-negative cocci attached to its free surface. The diplococcal arrangement was plainly visible in parts, and many leucocytes were packed with cocci. No other organisms were seen. At the necropsy no other peculiarities were noted in the heart, but microscopical examination of the musculature showed, especially in the wall of the right ventricle, minute abscesses in which intracellular Gram-negative diplococci could be demonstrated in the pus cells—multiple infections of each cell. Commencing necrosis of muscle fibres was present, and the condition would have been regarded as toxic had not the cocci been seen.

Lungs.—Approximately 3 oz. of clear fluid were present in both pleural sacs. There were no adhesions of pleural surfaces. The bases of both lungs showed congestion and marked oedema. Under the visceral pleura of the left lung there was some haemorrhagic mottling, with two small areas of consolidation. Microscopically the picture was one of a patchy embolic pneumonia, some alveoli being packed with pus cells, and others with red blood corpuscles, while surrounding alveoli showed an exudation of lymph. Solid clumps of Gram-negative cocci were present in the centre of some patches, but in addition clumps of Gram-positive cocci, some streptococci, and Gram-positive bacilli were also present.

Liver.—To the naked eye there was a little yellowish mottling visible. Microscopically the lobular arrangement was largely lost; liver cells were separated from each other and were of various sizes, some cells showing fatty infiltration—though this was not at all pronounced—some cells being multinucleated, and some having lost their nuclei. Many liver cells contained a yellowish granular pigment,

* The strength of the emulsions used to determine titre is not known by me.

which did not respond to tests for iron, either ferrous or ferric. Capillaries were not compressed. There was nothing of note in the portal tracts. I consider the condition to be an early toxic change, but it may possibly be a post-mortem condition.

Spleen.—This was enlarged, weighing 18 oz. No perisplenitis was present. Smears showed typical intracellular diplococci, with multiple invasion of the large endothelial cells. Sections showed some congestion of the splenic pulp, and much blood pigment throughout it. Some of the vessels were filled with aggregations of polymorphonuclear leucocytes. A few endothelial cells contained ingested red blood corpuscles.

Kidneys.—These were both pale, especially the cortex. Microscopical examination showed: (1) a somewhat thickened and adherent capsule, and (2) acute infection of several, but not all, glomeruli, accumulation of pus cells filling the tufts and cavities, and extending occasionally into surrounding tubules. In a few places the minute abscesses had started near but not in the glomeruli—probably from the afferent arterioles. Intracellular Gram-negative diplococci were seen in many pus cells; no other organisms. Other glomeruli were intensely congested. There was, however, a chronic condition present also, many glomeruli showing a multiplication of nuclei of the capillary tufts with no alterations of glomerular capsule. In others, attachment of the tuft had occurred to the outer capsule layer, and in a few fibrosis was commencing. Aggregation of lymphocytes surrounded a few glomeruli. Secretory tubules showed desquamative and degenerative changes in cells. Endarteritis was present in some small arteries. The appearance suggested there was a subacute glomerular nephritis on which an acute embolic condition had supervened.

Suprarenals.—The left showed massive necrosis with no inflammatory reaction; this may have been a post-mortem condition. The right showed minute abscesses in the cortex in which multiple Gram-negative diplococci could be observed in the pus cells; no other organisms.

Brain and Meninges.—On reflection of the dura mater a thin, diffuse, yellow, fibrinous-like deposit was seen on its under surface. The pia-arachnoid was congested, and at two sites considerable thin haemorrhagic effusion had occurred, both in the vertex—one anterior to the left parieto-occipital fissure and the other over a considerable area of the right frontal lobe. The membrane was thickened over the vertex at the inner end of the right Rolandic fissure. There was no flattening of the convolutions, no collections of pus in the sulci, the cerebro-spinal fluid was clear, and the choroid plexuses were not congested. On the under surface there was some gelatinous thickening round the stalk of the pituitary body. To the naked eye no other abnormality was revealed. Microscopical examination of the cortex (including a haemorrhagic area) and of the choroid plexus gave no evidence of an acute leptomeningitis. For the most part the pia-arachnoid was oedematous, in parts showing haemorrhagic effusions. Cells were endothelial-like, and the fibrous tissue was thin.

There were, however, some noticeable features. A few veins in the pia-arachnoid were filled with a soft clot composed mainly of polymorphonuclear leucocytes with red blood corpuscles and a little fibrin. The leucocytes had separated into a dense clump. This might have been an agonal condition. A few penetrating vessels at the surface of the cortex were found to be completely blocked with the same cells. No organisms were seen. The thickened gelatinous membrane round the stalk of the hypophysis was found to consist of a cellular connective tissue, with accumulations here and there of small mononuclears and a few plasma cells. A definite minute abscess was found here, surrounded by connective tissue; it was considered to be part of the general pyaemia. Numerous diplococci were found in the pus cells; no other organisms were seen. The pia-arachnoid over the right Rolandic area and over the central lobe on the upper surface of the cerebellum was found to be definitely thickened, the newly formed tissue being a hyaline connective tissue. The subdural exudate was seen to be

organized and loosely attached to the normal dura overlying. It consisted of a fine vascular connective tissue rich in large endothelial cells, some fibroblasts, and small mononuclear cells. No haemorrhages and little pigment were seen, and no polymorphonuclear leucocytes lying outside vessel walls. There was thus no evidence of an acute leptomeningitis, but there was evidence of a patchy chronic meningitis at vertex and base and over cerebellum. Nevertheless, a culture made at the necropsy from the surface of the pia-arachnoid over the vertex gave a pure growth of a Gram-negative coccus, and a smear made at the necropsy from the overlying subdural exudation showed a few polymorphonuclear leucocytes containing many typical bean-shaped diplococci and no other organisms. It may be, and I am inclined to this opinion, that the pus cells had exuded during the necessary manipulations from thin-walled vessels in which an agonal clot had occurred. It is possible, of course, to take the view that a terminal infection of the meninges had occurred so near to death that no cellular reaction had taken place.

COMMENTARY

1. The case was characterized by prolonged fever, mainly of a daily intermittent type with occasional rigors, with pronounced weakness and loss of energy, and with symptoms which may be grouped as a meningismus. The malaria-like temperature is emphasized in the literature as somewhat characteristic of subacute and chronic cerebro-spinal fever, but the clinical picture was not typical of any recognized condition, and in its terminal phase did not suggest a meningitis. A leucocytosis was present.

2. No benefit accrued from specific serum treatment; in fact, the impression of the clinician was that it exacerbated the symptoms, perhaps by liberation of endotoxins. For example, it was noted that the ecchymosis developed after the specific treatment was commenced. According to Marlow³ certain French writers have reported adversely on the use of therapeutic serum, although he himself believes it remains the logical procedure.

3. The infection was due to the meningococcus, as shown by the cultural and serological tests. It was isolated by blood culture, and the organisms were found as a rule in smears or sections of minute abscesses in various organs. The septicaemia was associated with a recent endocarditis, producing toxic changes in the liver and kidney (in the latter probably of some standing), petechiae in the skin, buccal mucosa, pericardium, endocardium, and leptomeninges, with minute abscesses in kidneys, right suprarenal gland, lung, heart muscle, and in the thickened pia-arachnoid at the base of the brain. The pyaemic condition must have developed shortly before death, as evidenced by the size of the abscesses.

4. An acute leptomeningitis was not present, but some of the veins in the pia-arachnoid and of some of the penetrating vessels in the surface of the cortex were packed with polymorphonuclear leucocytes. It is believed that the positive culture from the pia-arachnoid over the vertex and the positive smear from the subdural exudate were due to infected cells escaped from ruptured vessels.

5. A patchy chronic meningitis was present, which may have been the residue of an early attack of acute meningitis occurring at the commencement of the illness but recovered from, and the same may be surmised of the subdural exudate.

I wish to acknowledge my indebtedness to Miss G. Kerr for the numerous microscopical preparations which this study involved.

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