SEPTICAEMIA—THE FORGOTTEN COMPLICATION OF PARAPLEGIA

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IT is well recognised that prior to the development of centres for the treatment of paraplegic patients, the majority died soon after injury from the effects of renal sepsis. In 1917 Thompson Walker found that 47·2 per cent. of the patients admitted with spinal injuries to the King George V Military Hospital died from urinary infection 8 to 10 weeks after admission. With the advent of specialised centres during the 1939-45 war, this early mortality has been abolished largely due to the better understanding of the management of the paralysed bladder, regular turning and the availability of antibiotic therapy and blood transfusions.

If the patients survive the dangerous six weeks immediately after injury they still die many years later from the long-term complications of low-grade infection of the renal tract, which are chronic pyelonephritis, renal failure, hypertension and amyloidosis. However, despite the advances in treatment, there are still an appreciable number of patients dying of the acute effects of bacterial infection.

The most recent and comprehensive figures are those of Tribe 1969 (Tribe and Silver, 1969) who found that out of a series of 220 necropsies on paraplegic patients over a 20-year period, 3 died of overwhelming toxaemia and 4 of acute septic endocarditis.

For this reason it seemed worth while to try to exclude this possibility by carrying out frequent blood cultures and this has been done on patients at the Liverpool Regional Paraplegic Centre during the last four years. Blood cultures were carried out on:

(1) all patients who on admission had pressure sores, during the early dressing and operative inference with these sores and on any patient whose sores had deteriorated;

(2) patients with presumed severe urinary flare-ups;

(3) any patient with haematuria which was suspected to be due to a traumatised urethra.

Random repeated cultures were taken into 0·1 per cent. glucose broth without waiting for the height of a fever. Many of the patients being apyrexial or with sub-normal temperatures. When a positive culture was obtained in these ill patients it was considered that they were suffering from the systemic effects of these bacteria in their blood stream, this constituting a septicaemia and not a transient bacteraemia.

There appears to be some difference in the use of these terms in the literature. Waisbren (1964) and Beeson (1968) use the expression Gram-negative bacteraemia to describe the shock-like state with peripheral circulatory collapse produced by systemic infection with these organisms, whereas Anderson (1961) refers to the identical condition as being a septicaemia. Septicaemia representing a failure on the part of the body's inflammatory reaction. It being a diffuse infection in which the infectious organisms and their toxins are present in the blood stream.

CASE HISTORIES

W. G. A 64-year-old man who in August 1966 developed an acute incomplete tetraplegia due to an external staphylococcal abscess. This was operated on at the Neurosurgical Department at the Preston Royal Infirmary and he was transferred to this Centre for rehabilitation on 10 October that year. He followed the normal pattern of rehabilitation at this Centre. An intravenous pyelogram, cystography and cystometrogram were normal and by May 1967 his catheter had been discontinued. He was passing urine into a condom appliance. On the evening of 4 May he felt most unwell, complaining of left loin and left chest pain. He kept the whole ward awake moaning due to his pain, throughout the night (fig. 1). X-ray of the chest and abdomen were thought to

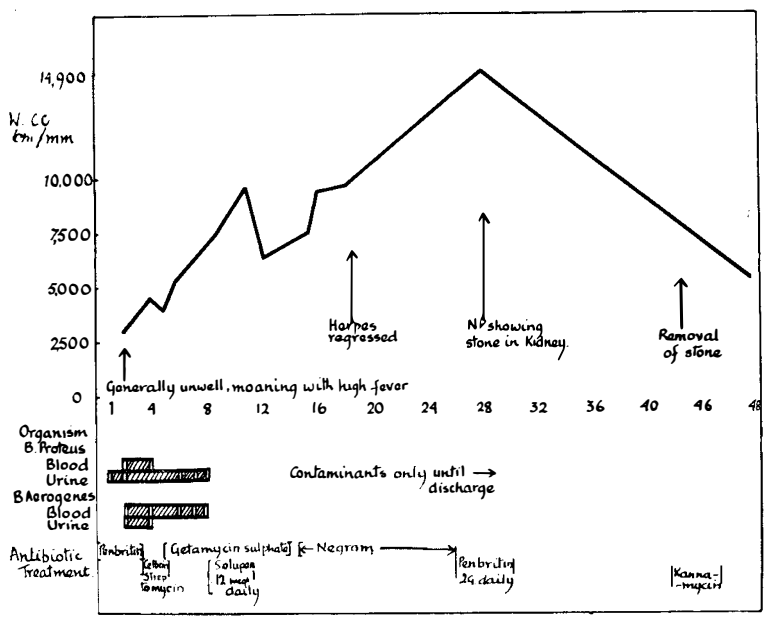

FIG. 1

W. G. Gram-negative septicaemia. Note the leucopenia. The white blood count only rose to normal levels when the blood cultures became negative.

be normal and a presumptive diagnosis of an acute urinary flare-up was made. A catheter was inserted and intramuscular Penbritin was commenced. By the next day his condition had deteriorated. He had drunk very little. He was sweating, confused and shocked with a blood pressure of 90/60 and a pulse of 160 per minute. Intravenous therapy was commenced but his white blood count was only 2,800. He was hyperpyrexial with a temperature of 105.5° F. A diagnosis of Gram-negative septicaemia was made and after initial blood cultures had been taken he was treated with intravenous saline dextrose and intramuscular Penbritin. This was changed to Streptomycin ½ c.c. and 1 gram Ceporin via the drip, four hourly. An initial dose of ½ gram Hydrocortisone was given through the drip together with intramuscular and intravenous injections of Aramine. Despite this therapy his blood pressure became unrecordable and he was anuric for some 12 hours. After this crisis his general condition improved slowly but he still required Avamine to maintain his blood pressure at 70/50. Blood cultures substantiated the diagnosis and as a result of the sensitivities obtained, Genticin 1 ml. four times per day and 20,000,000 units of penicillin was instituted. The blood grew, and the urine initially grew the same two

organisms, Proteus and *B. aerogenes*. Four further positive blood cultures were obtained and it was found that the proteus organism was cleared first from the blood but persisted in the urine. The aerogenes persisted in the blood whilst being cleared from the urine. All organisms were cleared from both urine and blood by the 8th day of the illness and the patient made a slow recovery by the 12th day, the white blood count gradually increasing. His improvement coincided with a severe widespread attack of herpes simplex over his face. However, the patient was far from well and continued to complain of pain on breathing in his left lower chest. A chest X-ray on the 16th day of his illness showed some segmental collapse in the left lower lobe. On the 29th day of his illness an intravenous pyelogram showed a calculus in the left proximal ureter. This was substantiated by an infusion I.V.P. and showed hydronephrotic changes in addition (fig. 2, 15.6.67). The stone was removed and after an uneventful post-operative course he was discharged well to another hospital (figure 3, 30.8.67) as it was not possible to discharge him home. In his case the septicaemia was due to an impacted renal calculus.

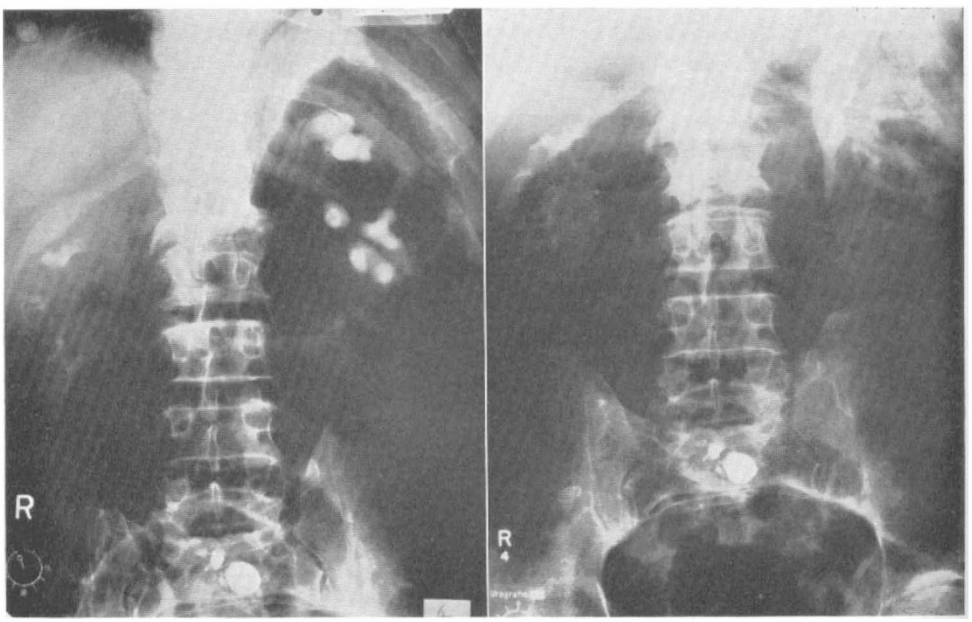

FIG. 2 FIG. 3

Fig. 2.—W. G. Infusion I.V.P. 15.6.67. Hydronephrotic changes on the left.

Fig. 3.—W. G. I.V.P. 30.8.67 after removal of the stone. Substantially normal on the left.

R. J. A 34-year-old housewife, married with two children, whose paraplegia was due to a congenital meningocele which had been closed by early operation, developed a small right ischial sore in December 1967. She was admitted to another hospital where a primary closure of this sore was attempted. This broke down within a few days. She felt very ill with nausea and vomiting but she was discharged home for Christmas with her sores unhealed. She noticed severe stiffness of the left shoulder and upper third of her arm and swelling of the left calf. Owing to her general debility she collapsed one day while still wearing her elastic stockings. When they were removed by the District Nurse some hours later, the skin of her swollen leg was adherent to the stocking, producing a circumferential sore of the lower third of the left leg (fig. 4). Following this, two weeks after the development of the painful shoulder she developed a swelling on the palmar

aspect of the right hand with an inability to flex her right ring-finger. She was seen initially at her home where a presumptive diagnosis of septicaemia was made and she was admitted to the Centre in a sick condition. Clinical examination revealed: Pulse 110 per minute. Blood pressure 120/70. Heart, chest and abdomen were normal. She had a complete spastic paraplegia below L1. She had extensive sores. The initial blood culture was positive and grew a *B. haemolytic* streptococcus. Her haemoglobin was 70 per cent. The pressure sores which were the most likely cause of her septicaemia, however, grew proteus organisms and her urine coliform bacteria in small numbers. She was treated with Penbritin 500 mg. four hourly intramuscularly and made a gradual clinical recovery from her toxicity in eight weeks, during this time she was given six pints of blood. The pain in the shoulder was localized to the tendon of supraspinatus. Initial X-rays were normal.

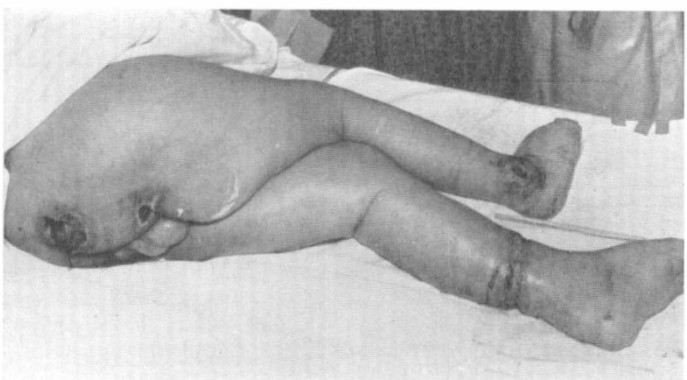

FIG. 4

R. J. Circumferential left stocking sore, caused by the patient collapsing while in her elastic stocking. The swollen left leg became adherent to the stocking. Note also the superficial right ischial sore.

X-rays some months later confirmed that she had osteomyelitis of the head of the humerus close to the tendon of supraspinatus. The swelling of her finger also came on suddenly. This developed so that she lost flexion of that finger and a diagnosis of ruptured profundus and sublimis tendons was apparent. Both episodes were considered to be embolic phenomena. She had several bouts of chest pain accompanied by a productive cough and X-ray changes of consolidation in the chest. It was concluded that the patient suffered from a Gram-positive septicaemia which produced embolic phenomena, mainly to the end arteries supplying the tendons. Eventually the lady's sores healed but some months later when she was feeling quite well nearly all her hair fell out. Examination of the roots by the dermatologist confirmed that this was a toxic phenomenon secondary to her septicaemia. The hair re-grew. She is now back at home looking after her two young children and her husband.

J. L. Aged 59. Developed a complete T9 paraplegia due to a spinal cord tumour, presumed to be an angiomatous malformation, some three years before the following events. He was re-admitted to the Centre with a recurrent right-sided orchitis. When the acute phase of orchitis had subsided an orchidectomy was performed. Post-operatively the Redivac drain collected a fair quantity of blood, however, during the second post-operative night the man had a blocked catheter, the subsequent retention was alleviated on changing it and the following morning the patient was found to be toxic, vomiting and in shock. On examination the scrotum was the size of a grapefruit and there was evidence

of blood tracking to the anterior abdominal wall. The wound sutures were removed and a large haematoma was evacuated.

During this procedure, the patient's condition became worse and a blood culture taken at the time grew *Staph. albus*. Urine culture at the time was a proteus organism and a later swab from the healing wound cultured *Ps. pyocyanea*. Intravenous fluids were given. The operation upon this man's genito-urinary tract produced a septicaemia, which accounted partly for his shock. The man made a clinical recovery within 16 days and the scrotal wound healed in 44 days. It is interesting to note that this man returned to the Unit two months after his discharge with his remaining testis hard and the size of a large hen's egg, giving a history of a recurrent urinary infection. After this orchitis had settled an epididymectomy was performed and the patient made an uneventful recovery and was last seen as an out-patient in a good state of health.

G. M. Aged 46. Sustained a fracture T7 and severe chest injury following a road traffic accident, resulting in a total paraplegia at T7. Throughout his admission he

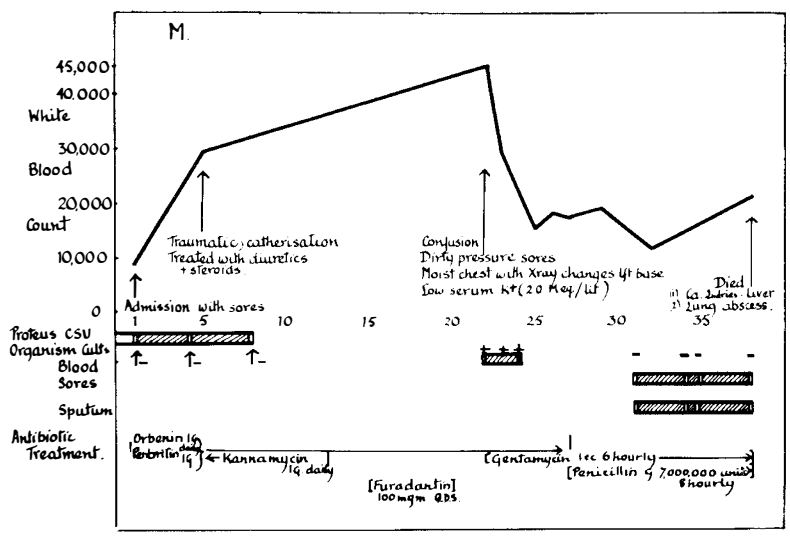

FIG. 5

G. M. Gram-negative septicaemia with a leukaemoid white blood count. This only fell when the blood culture became negative.

always appeared thin and emaciated. He was physically rehabilitated within six months but he was not passing urine spontaneously and, therefore, still had an indwelling balloon catheter. He was allowed leave to visit his mother, who had attempted suicide in Northern Ireland, from whence he did not return but was re-admitted from another hospital some two months later with gross pressure sores and a traumatised urethra with several false passages. Five days after his re-admission to the Centre his urethra was re-traumatised by a catheter and he developed suddenly the clinical picture of bacteraemic shock (fig. 5). The urine grew proteus but the blood culture was negative. Treatment was by forced diuresis, Hydrocortisone and Kanamycin 250 mg. Intravenous fluids were given slowly. The man made a slow recovery from this episode, but his sores failed to improve. On the 15th day after re-admission his condition deteriorated, he became drowsy, confused, would not drink, eat or keep his position in bed. His white blood count was 45,000. Haemoglobin 86 per cent. Blood urea 16 mg./100 ml. Electrolytes: Chlorides 65 mEq./litre, Sodium 96 mEq./litre, Potassium 2·5 mEq./litre. A lumbar puncture was performed but was a completely dry tap. Electrophoresis confirmed the low serum potassium.

Chest X-ray showed a local inflammatory process in the right lower lobe. Cultures: Blood—Proteus. Urine—no growth. Sores—*E. coli*. Sputum—Proteus.

In view of the fact that his sores had deteriorated, initially it was considered that these were the cause of his septicaemia. His low serum potassium was restored to normal by intravenous therapy, in addition to treating his 'chest infection' by postural drainage. From this time, after a transient improvement, there was a steady deterioration in his condition. The sores became worse. His cough did not improve. He eventually died in a breathless condition some 39 days from his re-admission. At the post-mortem two interesting points emerged:

1. That there was a large lung abscess in the right lower lobe.
2. There was a necrotic secondary carcinoma in his liver (primary not found), surrounded by several smaller deposits.

In retrospect it would seem that the bacteraemia from his traumatised urethra produced a metastatic lung abscess which later discharged organisms to produce his final septicaemia.

K. S. Aged 56. This man developed an incomplete paraplegia due to spinal deposits from a carcinoma of the prostate some two years before admission to the Centre for rehabilitation. His course of treatment included the healing of a pressure sore. During his period of decubitus an indwelling balloon catheter (14 Ch) was introduced to ensure free drainage of the bladder. This catheter was changed three times a week, as are all such catheters on the Unit. During one such change undue difficulty was found and several attempts were made; finally, however, a catheter was introduced and its balloon inflated. All seemed well except that the patient has an uncomfortable feeling in his urethra. The following morning the catheter did not seem to be draining and the patient still had pain so it was decided to change it. Removal of the catheter produced a gush of blood per urethram and a generalised shock reaction in the patient, which included a continuous rigor. Treatment consisted of intravenous fluids, intravenous Tetracycline, Kanamycin and Hydrocortisone. Blood culture showed *E. Coli* whilst urine showed *B. proteus*. Both these organisms were sensitive to Kanamycin. The proteus was also sensitive to Negram but the *E. coli* was not set up against this and was sensitive to Kanamycin only. With intensive treatment the man made a satisfactory clinical recovery within 48 hours. Eventually his catheter was discontinued and he was returned to his original hospital fit. The findings are summarised in Tables I and II.

Discussion

Bacteriology. At the time that four of the patients were studied, owing to the undermanning of the Pathology Department, it was not possible to carry out detailed blood cultures and typing of the organisms. The necessity for this is appreciated. The procedure now is that the urine specimens are separately studied by J. H. M. and a combined round is carried out once a week by all three authors, when the cases are discussed, therapy is settled and future problems delineated.

Four patients had a single organism and one patient had two organisms in their blood stream. Two patients had a proteus organism. One patient had a *Staph. albus*. One patient had a haemolytic streptococcus (see Table II).

The patient with two organisms had a proteus and a *Klebsiella aerogenes*.

Repeated cultures were carried out. In two patients the culture was persistently positive, despite anti-bacterial therapy which judged from the results of

TABLE I
Summary of the Findings

Name	Age	Sex	Lesion	Precipitating factor	Aggravating factor	Organism
G	64	Male	Incomplete tetraplegia due to extradural abscess at C6	Stone in ureter	Failure to remove the stone	Proteus *B. aerogenes*
L	60	Male	Complete paraplegia at T9 due to angioma of the cord	Orchidectomy followed by a blocked catheter. He developed a large haematoma in the scrotum	——	*Staph. albus*
S	56	Male	Incomplete paraplegia at L3 due to 2° deposits from the prostate	Traumatised urethra	Malignant disease on oestrogen therapy	*E. coli*
M	46	Male	Complete paraplegia T6 due to trauma	Pressure sores traumatized urethra	Malignant disease, steroids, diuretics leading to hypokalaemia	Proteus
J	34	Female	Spina bifida complete below L1	1° suture of a pressure sore	Discharge from hospital to sit on her sores at home	Haemolytic streptococcus

TABLE II
Summary of the Bacteriology

Name	Blood	Urine	Sores	Sputum	Presumed source	Antibiotic	Other agent
G	Proteus *B. aerogenes*	Proteus	—	—	Urine	Penbritin Genticin Penicillin Streptomycin Ceporin	Hydrocortisone Aramine
L	*Staph albus*	Proteus	*Ps. Pyocyanea* (wound)	—	Wound	Tetracycline	—
S	*E. coli*	Proteus	—	—	Urine	Kanamycin	—
M	Proteus	Proteus	Proteus	Proteus	Sore	Orbenin Penbritin Genticin	Diuretics Hydrocortisone
J	Haemolytic streptococcus	—	Proteus	—	—	Penbritin	—

sensitivities carried out on the blood cultures should have been effective in sterilising the blood. These were the most severely ill patients as reflected by abnormal white blood counts, shocked condition, persistently low blood pressure and the death of one of the two patients.

In three patients the culture was only positive on one occasion, the first time that it was taken. The culture became negative on the initial treatment with a broad spectrum antibiotic, instituted before the sensitivities obtained from the blood cultures were available. In an endeavour to find the source of the septicaemia, repeated cultures were also made upon the throat, urine, pressure sores when present, sputum and in the only female patient from the vagina. In two of the patients (G and M) the same organism, a proteus, was recovered from elsewhere in the patients body—the urinary tract. M., the most severely affected who died, had a lung abscess. Proteus organisms were recovered from this abscess and from his sputum. In three of the patients it was not possible to isolate the same organisms from elsewhere in the patient's body. In one of the patients *E. coli* was obtained from the blood culture and a proteus from the urine and we presumed from the history of the onset of septicaemia immediately following traumatisation of the urethra that the urinary tract was also the source. Recovery of *Staph. albus* from J. L. was at first thought to be a contaminant, but this organism was also obtained immediately following trauma to the urinary tract by operation and this organism has been reported as a common pathogen in urinary tract infections (Gallagher *et al.*, 1965) and particularly following urethra trauma (Mitchell, 1965). It was not possible to prove the source of the *B. haemolytic* streptococcus but it was presumed to be from the pressure sores.

Pathogenesis. A transient release of bacteria into the blood stream from, for example, the roots of the teeth is of daily occurrence and does not result in septicaemia, since these organisms are rapidly eliminated. Septicaemia demands the constant egress of infected material into the blood stream and a failure of their elimination. For this to take place some of the following criteria must be fulfilled: (1) infected material must be present; (2) the infected material cannot be eliminated or drained naturally; (3) there is a breakdown in the natural barriers of the body between the infected material and the lymphatics or blood stream.

Precipitating Factors. All five patients had infected urine. If the infection remains restricted to the bladder a low-grade cystitis results and many patients can live in symbiosis with the organisms from such an infection. Should these organisms enter the blood stream a bacteraemia will result, and depending upon the other factors mentioned above this can develop into a septicaemia. There are two routes of infection, (*a*) back pressure through the kidneys due to distal obstruction. One patient (G) had a stone impacted in his ureter. The infected urine dammed up behind it and he had a persistent septicaemia from a proteus organism recovered from the blood stream and urine. He remained unwell with a persistently positive blood culture despite anti-bacterial therapy that should have been effective judging from the sensitivities obtained from the blood cultures, until the stone was removed and free drainage established he then became quite well. This mode of infection is the common one in urinary flare-up secondary to a blocked catheter, and with the use of modern drainage equipment and monitoring of the catheter this has been almost completely eliminated at Southport. The other method by which

infected material can get from the bladder into the blood stream is through trauma to the mucosa of the urethra by a catheter (fig. 6). Even minimal degrees of trauma produce small tears permitting infected material to enter the highly vascular corporal tissue which surrounds the urethra. This was the mechanism in three of the patients. In two of whom the same organism was recovered from the urine and the blood stream. This can occur in several ways: (1) Inflation of the balloon of the catheter in the urethra instead of in the bladder—one patient (S) with partial sensation described how he experienced immediate pain on inflation of the balloon of the catheter accompanied by haematuria and general malaise. A further patient, not included in the series, experienced an identical picture although his blood culture did not grow any organisms. (2) Traumatic removal of a fully inflated balloon catheter either by the confused patient, the ignorant staff or by oversight while the patient is being turned. Two patients who did not have positive blood

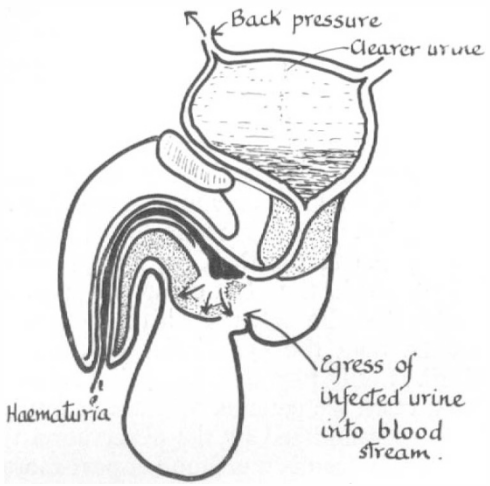

FIG. 6

Mechanics of septicaemia associated with trauma to the urethra and a faulty catheter. Either the failure to drain the bladder or the trauma to the urethra can produce a septicaemia. The combination of the two are particularly dangerous.

cultures and were not included in the series but had an identical clinical picture developed this from the mechanism. (3) Slow deflation of the balloon of the catheter. This mechanism has been described by Hardy (1968) and experienced by all doctors in Spinal Centres whereby although the catheter is correctly inserted into the bladder but due to failure of the valve mechanism it gradually deflates and extrudes several hours later, while still partially inflated. As a consequence, the urethra may be traumatised by the balloon of the catheter on its way out. This happened in one of the patients due to a fault in the valve mechanism on a new type of catheter (M). This is a highly dangerous cause of septicaemia, since it combines two dangerous factors. On the one hand it allows infected material to enter the blood stream through the highly vascular corporal tissue and on the other hand it does not allow the free drainage of urine which accumulates in the bladder and can thus enter the blood stream through the mechanism described earlier (see Fig. 6). Unfortunately, several hours may elapse before the diagnosis is made and treatment established and the morbidity is greatly increased. The lack of free drainage also pertains in the case of L, who had an orchidectomy. His catheter blocked during the first post-operative night and a large haematoma developed within his scrotum. A haematoma is a recognised complication of operations upon

the testicle since the tissue is lax in this region. There was thus again blockage to free flow of the urine with a highly vascular area in contact with infected material. The patient who developed septicaemia secondary to pressure sores (J) is another example of this failure to drain infected material. She devleoped a superficial sore over her right ischium at home. This was treated by excision and primary suture at another hospital. In her case the suturing had prevented the free drainage of infected material thus giving rise to the septicaemia. At a later date the wound broke down and free drainage was established. A further almost identical case (W) was seen where a woman admitted to a teaching hospital had a sore excised and sutured. Some days later she became extremely ill with a high swinging fever and eventually two pints of pus was drained from the opposite hip, the joint becoming completely destroyed. The initial sore never broke down. This woman was only admitted to the Centre some months after this episode of septicaemia but there are no positive blood cultures to substantiate the diagnosis.

Depression of the Defence Mechanism

Medical Causes. The cause of the paraplegia in four of the patients was non-progressive neurological disease. In only one patient was the paraplegia traumatic in origin. Since the Centre, like all spinal centres in the United Kingdom, gives priority of admission and re-admission to traumatic injuries, the incidence of septicaemia is out of proportion to the relative frequency of medical cases within the Centre. A check of the Centre on 19.8.69 showed that there were 19 traumatic admissions within the Unit compared with 10 non-traumatic. This would seem to indicate that the patients with non-traumatic paraplegia are at greater risk of septicaemia than the paraplegics of traumatic origin, presumably because the paraplegia is only part of a generalised disease which is still present whereas the traumatic cases are patients who are fit until they sustained their injury. This is strikingly substantiated by the observation that the one traumatic case (M) who developed septicaemia was found at post-mortem to have a large necrotic secondary carcinomatous deposit in his liver. Until post mortem we had no knowledge of this although his appearance had always been emaciated and his sores had failed to make the normal recovery one would have expected with treatment. Two other patients who are referred to in this paper where we had no proof by positive blood culture (R and W) were also non-traumatic in origin. The only other figures relating to the relative incidence of this condition in paraplegia are those obtained from the post-mortem series of Tribe (Tribe and Silver, 1969). There were seven patients with septicaemia; only one of those occurred in a non-traumatic case and six in traumatic cases. This would correspond to the relative incidence of the respective post mortems performed and the preponderance of traumatic admissions at the Stoke Mandeville Centre, but we could not substantiate from our findings the view advanced that the cause of death is the same in chronic paraplegic patients irrespective of the cause.

Malignancy. One of the patients (S) paraplegia was due to secondary deposits in his spine from the prostate, and (M) was found to have secondary deposits in his liver. Septicaemia is a recognised complication of malignancy due to a reduction in the synthesis of globulin, and neutropenia which may be due to the infiltration of the bone marrow with malignant deposits.

Loss of Protein in the Urine. All the patients had urinary infections and the presence of infected material in the bladder has been mentioned earlier as a precipitating factor, due to the egress of infected material into the blood stream, but the presence of a urinary infection may facilitate the development of septicaemia by virtue of the loss of protein in the urine, leading to a depression in the serum protein. The globulin fraction contains the circulating antibodies. One of the patients (R) whose history is not given in detail earlier on had amyloid disease with a loss of 20 gram of protein in his urine per day, and a serum globulin level of 1·8. He was found, at post mortem, to have an abscess in his spleen and acute ulcerative endocarditis. The relevance of amyloid can be judged by the observation that four out of seven patients with septicaemia in the series of Tribe and Silver suffered from amyloid disease.

Drugs. The use of steroids which depress the defence mechanisms is well recognised as causing septicaemia in patients with rheumatoid arthritis and in transplant operations. The prolonged use of diuretics with hypokalaemia and confusion leading to immobility appears to be a significant factor in one of the patients.

Reaction to the Severe Infection. The two most severely affected patients (M and G) developed a severe herpes simplex infection of the mouth and face after the initial episode. The other three patients who were less severely affected did not. Many patients have shown herpes as a reaction to severe infections in the centre. It is recognised that the virus of herpes simplex lies dormant and may be reactivated by systemic illness. Another example of response to severe infection is in the patient J, who lost her hair some four months after the initial episode. This has been seen in one other female patient who lost her hair some three months after an acute infective episode. The hair dies at the time of the infection but takes some three months to grow out. The diagnosis was substantiated by examining the roots of the hair.

Shock. Four patients were shocked. In two patients (M and G) it was prolonged, lasting for several days and required intravenous fluids, hypertensive agents and steroids. In these patients it was associated with a Gram-negative septicaemia. In two patients (S and L) it was present for only 24 hours, requiring intravenous fluids. One of these patients (S) had a Gram-negative septicaemia. The fifth patient (J) with a *B. haemolytic* septicaemia was not shocked but was given blood transfusions for her general condition. In this limited series the hypotension was of diagnostic value pointing to a Gram-negative septicaemia.

Abnormal White Blood Count. One patient (M) had a leukaemoid blood reaction, that is, the occurrence in a patient not suffering from leukaemia of a peripheral blood picture resembling leukaemia because of the marked elevation of the total white blood count. In his case it was above 45,000. The reaction was myeloid and although the blood picture did not suggest leukaemia because there were no primitive or abnormal cells and his obviously ill state from his septicaemia suggested the true diagnosis. For this reason it was not necessary to carry out bone marrow aspiration, chromosome cultures or leucocyte alkaline phosphatase score to substantiate the diagnosis. The picture was quite typical and it is a

recognised manifestation of severe infections and malignancy. The other patient (G) showed a severe leucopenia. Leucopenia is a recognised manifestation of overwhelming infections, such as tuberculosis and septicaemia. It also can occur in specific infections, such as typhoid, viral and protozone infections as a complication of debilitating states and in certain haemopoietic disorders. It is naturally also a complication of certain toxins and drugs as the patient was obviously desperately ill and had only been on Penbritin for 24 hours. Little difficulty was found in arriving at the correct diagnosis.

Treatment. The treatment of septicaemia is a complex controversial subject and it was based in these patients on giving initially a broad spectrum antibiotic, relieving the precipitating factor, maintaining the blood pressure by intravenous fluids and hypertensive agents and steroids and then giving the appropriate antibiotic as soon as the sensitivities from the blood cultures were obtained. Steroids were not employed as a routine procedure. Peripheral vasodilator agents were not given and the use of steroids and hypertensive agents is controversial.

Mortality. The mortality in septicaemia surprisingly has not changed strikingly between 1935 and 1960, despite the advent of effective chemotherapy. Finland (1959) pointed out that because of more adventurous surgery, more drastic radiotherapy procedures and the use of immunosuppressive drugs there are many new cases of debility on the hospital wards so that many more severely debilitated patients are surviving to die of septicaemia, resulting in a higher mortality. He found that there is a higher mortality in 1959 than in 1935 from septicaemia, while Weil, Shubin and Biddle (1964) reported that when shock accompanied 169 out of 692 cases of Gram-negative bacteraemia there was a mortality of 82 per cent. McCabe and Jackson (1963) reported a mortality in Gram-negative bacteraemia of between 30 per cent. and 80 per cent. Beeson (1967) reported a mortality of 75 per cent. in Gram-negative bacteraemia when accompanied by shock. The latest figures are those of Chalmers and Tiller (1969), who found that of 173 patients with septicaemia between 1962 and 1968 that the mortality was 43 per cent. between 1962 and 1965 and only 28 per cent. between 1966 and 1968. The improvement in the mortality was largely in those patients with Gram-negative septicaemia accompanied by shock. They attributed this improvement to the greater awareness of the condition so that it was diagnosed earlier, the monitoring of the central venous pressure to regulate the administration of fluids. They did not find steroids of any particular advantage. It is apparent that with such a small series of cases only limited conclusions can be drawn from the figures. Nevertheless, the fact that of all the five patients in whom the diagnosis was made by blood culture only one died and that patient had a secondary carcinomatous deposit in the liver is encouraging.

Differential Diagnosis. The patient (R) who died of renal failure and amyloid disease was found at post mortem to have a large abscess in his spleen and bacterial endocarditis. He presumably had septicaemia. Seven cases were reported in the series of Tribe to have died of this complication. The diagnosis in a staphylococcal septicaemia may be overlooked since shock does not develop until late and in a Gram-negative septicaemia the diagnosis may be missed because it may mimic a coronary thrombosis or a pulmonary embolus. Again the diagnosis

of septicaemia may not be established if it is assumed that all temperatures are due to urinary infections and blunderbuss therapy of a broad spectrum antibiotic is instituted. In order to avoid these pitfalls, the following practice is adopted at this Centre. Any patient who develops a temperature has a full clinical examination with particular reference to the leg veins, scrotum, chest and where there is a catheter, whether this is draining freely. Straight X-rays of the abdomen and chest are taken, haemoglobin, white blood count, blood urea carried out with urine cultures, electrocardiographs, blood cultures and, if necessary, an infusion I.V.P. It is to be emphasised that if the diagnosis of a septicaemia is to be established and effective treatment administered, blood cultures should be taken before any antibiotics are instituted since the indiscriminate use of antibiotics in the case of septicaemia may render the isolation of an organism from the blood impossible while failing to treat adequately the initial focus. The large number of aspiration pneumonias as a cause of fever in the early stages after spinal injury in patients with paralysed expiratory muscles has been emphasised by Silver (1968), where 18 out of 50 patients admitted within a few days of injury had pneumonia diagnosed by chest X-ray. Seventeen of these 18 patients had paralysed expiratory muscles. Another potent source of fever is the presence of sub-clinical pulmonary emboli within the first few months after injury. For this reason routine anticoagulants are given to all acute traumatic admissions, a total of 33 patients, during the last 18 months. Since that time there has been a striking reduction in the mortality, not a single acute paraplegic patient being lost and the morbidity from chest infections has decreased, presumably many of the 'pneumonias' that we had previously treated were pulmonary infarcts.

Summary and Conclusions

The occurrence of septicaemia in five patients is described. In four of the patients it arose from the genito-urinary tract whilst the patients were in the Spinal Centre. The pathogenesis of the development of this complication is described. Accepting that the management of the genito-urinary tract is not the only responsibility of the doctor looking after these patients, it is certainly one of his most important tasks and even closer surveillance could have prevented this complication.

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Discussion

Chairman (Dr. T. Gregg). We are used to expecting something special from Southport, and Dr. Silver has certainly lived up to it. We have gone over time in this particular period, and perhaps if anyone has any detailed points they could speak to Dr. Silver afterwards. If there are any points of general interest or any questions anyone would like to ask now, if they could be very brief, Dr. Silver has to leave in five minutes. The paper will be published and it was very comprehensive, and thanks are due to you, Dr. Silver.

THE SPINAL UNIT AT MURNAU (GERMANY)

By Dr. E. Stophasius

If you look at the first slide, don't be afraid that I am going to hold a paper on agriculture or that I wish to make advertisement for Bavarian milk—I would just like to give you a general view at the new Spinal Centre at Murnau.

This year, on 24 January, the Centre of the Accident Hospital at Murnau was opened in the presence of Sir Ludwig. It has to be mentioned that it was Sir Ludwig who stimulated the German authorities to attach a Spinal Centre to the Murnau Accident Hospital (Berufsgenossenschaftliches Unfallkrankenhaus), which has been directed until 31 January 1969 for almost 15 years by Professor Dr.med. A. Lob, who planned the new Unit and supervised the construction of it.

The Hospital as well as the Centre itself is run by a union of several so-called Berufsgenossenschaften—that are Employers' Liability Insurance Associations, which are compulsory. The insurance premium is paid exclusively by the employer.

The capacity of the centre is 60 beds in three wards of 20 beds. Each ward consists of 2 four-bed rooms and 6 two-bed rooms located in the south of this five-floor building. They are connected by three balconies. To each room belongs a sanitary unit, 4 patients sharing one toilet. Each room is connected to the central oxygen and air pressure supply system as well as to an optical and acoustic call system.

In the north of each ward are located the functional rooms divided from the bedrooms by a wide floor.

In the east of the wards are the eating and sitting rooms and the elevators.

The floor below the wards is occupied by the occupational therapy department, including a kitchen for training of paraplegic women; dressing-rooms for the personnel and an office.

The operating theatre, the X-ray department, the laboratories, the central sterilisation, the urological theatre and the rooms for the consulting specialists are at the next lower floor.

At the same floor southwards in another complex there is the physiotherapy