cases were there any reflex changes in the upper limbs, and, although x-ray films of the cervical spine were taken, evidence of cervical spondylosis was found in only one case.

RESULTS

At the end of one month's treatment 29 out of the 35 patients were either symptom-free or much improved, but six showed no improvement; thus the treatment was initially successful in nearly 90% of cases. In one case treatment was combined with splinting of the wrist. Of the six cases in which the treatment proved unsuccessful four were cured by splinting and two eventually underwent operation. At the end of three months 19 of the 29 patients were still symptom-free, but 10 had relapsed on the treatment.

At a subsequent follow-up, varying from 3 to 13 months, there were three further relapses, so that 16 remained symptom-free without drug therapy. In one of the late relapses chlorothiazide was recommenced and the patient again became symptom-free.

The patients were not weighed as a routine, but 14 were weighed during the course of treatment and striking discrepancies were found; eight lost an average of 5 lb. (2.3 kg.), four gained an average of 10 lb. (4.5 kg.), and in two the weight remained stationary. Thus there was no significant change in the average weights during treatment.

It is evident that many cases of the carpal-tunnel syndrome can be successfully treated with chlorothiazide. There was no ultimate difference between cases with physical signs and those without, though the latter seemed to respond better initially; nor was duration of symptoms of importance in determining response, since two cases with symptoms for 15 years responded.

Patients with purely nocturnal symptoms and those with nocturnal and diurnal symptoms seemed to respond equally well to treatment, but those with continuous symptoms suggesting permanent nerve damage rather than recurrent ischaemia did not respond.

Although improvement appeared to be correlated with diuresis it was surprising to us that so few patients lost weight. This might be explained by the fact that some admitted to drinking more. Removal of oedema beneath the transverse carpal ligament need only be very small in amount to be sufficient to break a recurrent cycle of nerve ischaemia. However, it is possible that improvement may have been due more to redistribution of body fluid than to generalized dehydration. Such an explanation has been put forward to explain improvement with systemic steroid therapy, which may even cause oedema.

Side-effects were few; only two patients complained of slight nausea at the beginning of treatment. We encountered no clinical instances of electrolyte imbalance, nor was postural hypotension observed. Given with a weekly rest day and with a potassium supplement the drug appears to be safe, even when administered over a period of months.

Splinting and chlorothiazide can be combined: this was done successfully in three cases. Combined treatment might well have succeeded in more cases, but was purposely avoided.

Obesity appeared to be of importance in more than a quarter of this series, and many patients commented that symptoms began after a sudden increase in weight.

Adipose tissue in the carpal tunnel might be a precipitating cause in these patients or, alternatively, subcutaneous fat may compress the transverse carpal ligament from outside. It seems possible that treatment might have been successful in more cases had the patients not been simultaneously gaining weight, as these comprised a number of our failures.

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The Scribner Arteriovenous Fistula for Haemodialysis

Experience has shown that some patients with acute renal failure reach artificial kidney units with veins inadequate for repeated cannulation for haemodialysis; and when such patients require haemodialysis every second or third day the deficiency may significantly affect their practical management. Alwall *et al.* (1948-9) attempted to resolve the problem by the employment of arterial and venous glass cannulae connected by a fistula of capillary glass tubing. Despite continuous heparinization, however, the technique proved unsuccessful. The indwelling cannulae and fistula recently introduced by Scribner (Quinton *et al.*, 1960; Scribner *et al.*, 1960) for the treatment of patients with chronic renal failure by repeated haemodialysis are made of polytetra fluoroethylene (P.T.F.E.), which is inert in body tissues and does not provoke clotting: patency is maintained without anticoagulants.

THE APPARATUS*

The cannulae and fistula are prepared from thin-walled P.T.F.E. tubing† of 2.84 mm. external and 2.76 mm. internal diameter. The cannulae are heat-drawn to sizes suitable for cannulation of a radial artery and a superficial radial vein: they are bent to a J-shape with the short tapered arm 5 cm. and the long untapered arm 10 cm. in length. "Swagelok" fittings are used to connect the ends of the long arms of the cannulae separately to two short stainless-steel conduits which are part of a stainless-steel plate: the opposite ends of the stainless-steel conduits are similarly connected to a single short (6 cm.) U-shaped P.T.F.E. tube previously heat-drawn so that its middle third is constricted down to approximately 1.25 mm. internal diameter (Fig. 1). With the cannulae in a radial artery and superficial radial vein, blood can pass freely from artery to vein through the fistula. When haemodialysis is carried out it is necessary only to disconnect the fistula temporarily and to connect the long arms of the arterial and venous cannulae to standard polyvinyl chloride (P.V.C.) tubing

*All items of equipment, including apparatus for moulding P.T.F.E., may be obtained from the Medical Division, Tally Register Corporation, 1310 Mercer Street, Seattle 9, Washington.
†"CLIX" TWT/10, supplied by Siemens Edison Swan Ltd., 132 Long Acre, London W.C.2.

leading respectively into and out from an artificial kidney machine (Fig. 2). The fistula is re-established when haemodialysis has been completed.

FIG. 1.—Indwelling arterial and venous P.T.F.E. cannulae (*in situ* for 21 days) with fittings and U-shaped P.T.F.E. connexion forming the arteriovenous fistula.

FIG. 2.—Indwelling cannulae connected to inflow and outflow tubing during haemodialysis.

THE CANNULATION

The cannulation is best carried out in an operating-theatre under local infiltration anaesthesia. When possible, the cannulation is undertaken several hours before haemodialysis; this avoids untoward subcutaneous bleeding after subsequent heparinization for the haemodialysis. Through an S-shaped incision at the wrist (Fig. 1) the distal 2 cm. of the radial artery is exposed and ligated 4 cm. proximal to the radial styloid: a superficial radial vein, usually a large tributary of the cephalic vein, is isolated and ligated on the lateral aspect of the wrist through the same incision. A subcutaneous tunnel 5–8 cm. long is then made by passing the closed blades of a large curved artery forceps up from the exposed artery and vein on the ventral aspect of the forearm: two small adjacent skin punctures are made at the proximal end of the subcutaneous tunnel so that the long arms of the two J-shaped P.T.F.E. cannulae can be passed up subcutaneously from the incision at the wrist to emerge through the skin in the mid-zone of the ventral aspect of the forearm. Because the cannulae kink easily it is expedient to site them comfortably, to avoid overlapping, and to ensure that they are correctly aligned before they are connected to the conduits of the stainless-steel plate. It is also important to cut the ends of the cannulae cleanly and at right angles.

The venous cannula is introduced into the radial vein just proximal to the ligature and is secured in position by a ligature of fine thread: its proximal end is connected to the lateral conduit of the stainless-steel plate. A sphygmomanometer, previously applied to the upper arm, is then inflated and used as an arterial tourniquet before the radial artery is incised and cannulated; the

cannula is secured in position by a ligature of fine thread and its proximal end is connected to the medial conduit of the stainless-steel plate. The sphygmomanometer is then deflated and the fistula completed. The incision at the wrist is sprayed with polybactrin, sutured, and covered with a dry dressing and bandage. The exit wounds of the cannulae are also sprayed with polybactrin and covered with a dry dressing and bandage. The stainless-steel plate is anchored to the forearm by two narrow strips of adhesive tape, and, if the patient is restless, the forearm is splinted on a light plaster-of-Paris cast.

EXPERIENCE

The Scribner arteriovenous fistula has been used in 10 patients and has proved to be eminently satisfactory: in only a single case did a minor inconsequential degree of sepsis eventually develop. The following two case histories exemplify its use.

Case 1.—A 21-year-old man was involved in a road accident and sustained an open fracture of the right tibia and fibula, a comminuted fracture of the right acetabulum, and multiple lacerations of the scalp and thighs. Acute oliguric renal failure developed, and when he was transferred to Hammersmith Hospital on the sixth day after injury the blood urea was 465 mg./100 ml., the serum potassium 6.7 mEq/l., and the CO_2 combining power 18 mEq/l. The rapid biochemical deterioration, with an average daily rise of blood urea of 72 mg./100 ml., indicated the need for urgent haemodialysis, and as only six days had elapsed since the accident it was thought that repeated haemodialyses would be required. A Scribner arteriovenous fistula was applied to the left forearm and haemodialysis was carried out two hours later. Two further haemodialyses, at four-day intervals, were required before the onset of a spontaneous diuresis occurred 16 days after the original accident. The fistula was removed two days later. Diuresis continued and blood urea fell to 39 mg./100 ml. by the 29th day after injury. Although the illness was complicated by a phase of hypertension, melaena, and infection of the leg wound and the urine with *Pseudomonas pyocyanea* and *Streptococcus faecalis*, the incisions required for the application of the fistula remained sterile and healed per primam.

Case 2.—A 58-year-old man who had a six-year history of episodic ureteric colic with a passage of calcium oxalate and phosphate stones, and who showed radiographic evidence of bilateral renal calcification, was admitted to hospital during an attack of ureteric colic and again passed a small stone. Three weeks later there was a rapid deterioration in his general condition with the development of oliguria and azotaemia. He was transferred to Hammersmith Hospital, where the blood urea was found to be 570 mg./100 ml., the serum potassium 6.4 mEq/l., and the CO_2 combining power 10 mEq/l. Bilateral groin scars were present, and it transpired that high ligation and stripping of the long saphenous veins had been carried out some years previously; there was also a severe acute thrombophlebitis in the right upper arm. Although it was thought that he had bilateral calculous pyelonephritis, the cause of the relatively rapid and recent biochemical deterioration was not clear, and it was decided to carry out haemodialysis in the hope that some degree of long-term restitution might ensue. A Scribner arteriovenous fistula was therefore applied to the left forearm and haemodialysis was carried out 12 hours later with excellent clinical and biochemical improvement. Subsequent attempts to provoke a diuresis, however, failed, and the patient died 49 days after admission to Hammersmith Hospital. Post-mortem examination revealed the diagnosis of calcium oxalate nephro-calcinosis and oxalosis. Throughout his illness in Hammersmith, the Scribner arteriovenous fistula remained patent although bacteriological cultures from the incisions made for the application of the fistula grew *Staphylococcus pyogenes* during the last few

days of his life; the same organism was also grown from blood cultures and the sputum.

DISCUSSION

A total of 24 haemodialyses have been performed in 10 patients with the Scribner arteriovenous fistula. Satisfactory flow-rates were achieved during haemodialysis with both the rotating-coil and twin-coil machines, and there were no technical difficulties. It was easy to connect the fistula to the artificial kidney; the time saved by omission of the repeated cut-downs, otherwise necessary when frequent haemodialyses were required, was significant, and there was correspondingly less discomfort for the patients.

Although it is sometimes possible, for the purpose of repeated vein-to-vein haemodialysis, to recannulate a vein on five or six occasions, the use of indwelling arterial and venous cannulation is an attractive alternative. Scribner *et al.* (1960) and Scribner (1961, personal communication) introduced the P.T.F.E. arteriovenous fistula for the treatment of patients with chronic renal failure and have been able to maintain patency of the fistula for many months. The fistula may, however, be even more valuable for repeated haemodialysis in some cases of acute oliguric renal failure, particularly when there is rapid biochemical deterioration and haemodialysis is required every two or three days—for example, in cases of acute oliguric renal failure which complicates surgical or accidental trauma (Shackman

et al., 1960). The fistula is particularly valuable when extensive soft-tissue injury, burns, thrombophlebitis after repeated venepuncture or intravenous infusions, or skeletal injuries requiring extensive plaster-of-Paris casts limit access to suitable veins.

Tissue reaction to P.T.F.E. is minimal or negligible (J. Calnan, 1961, personal communication). Histological examination of an artery and a vein in which P.T.F.E. cannulae had remained for 25 days showed partial necrosis of the wall but no inflammatory reaction (Fig. 3). An acute vasculitis, with local destruction of the internal and external elastic laminae, may, however, follow local infection (Fig. 4). Uraemic patients are particularly prone to infection, and it must be recognized, therefore, that an indwelling arteriovenous fistula, even of P.T.F.E., may sometimes provoke sepsis: this potential risk, however, is small. Moreover, the risk is not excluded when the alternative method of repeated cannulation is used.

SUMMARY

The Scribner arteriovenous fistula of P.T.F.E. is described.

Haemodialysis has been successfully carried out with the fistula on 24 occasions in 10 patients.

It is concluded that the technique represents a significant advance in the treatment of some patients with acute oliguric renal failure, particularly of traumatic or surgical origin.

FIG. 3.—Artery and vein in which P.T.F.E. cannulae had remained for 25 days, showing partial necrosis of wall but no inflammatory reaction. ($\times 74$.)

FIG. 4.—Acute vasculitis in artery and vein in which P.T.F.E. cannulae had remained for 49 days, showing polymorphonuclear leucocyte infiltration and destruction of elastic laminae and muscle. Sections include part of cannula within the vessel wall. ($\times 74$.)

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Medical Memoranda

Sensory Loss in Poliomyelitis

The clinical picture of poliomyelitis is one of meningeal irritation followed by flaccid paresis due to injury of the lower motor neurone. Pains in the back and leg muscles are the rule, and loss or even alteration of sensation is extremely rare. It is only recently that confirmation of infection with the poliovirus has been easily obtainable. Formerly the presence of sensory change would throw doubt on the diagnosis. Plum (1956) reports the only case with full virological confirmation which could be found in the literature. He refers to 10 previous case reports, the earliest in 1909. He throws doubt on the diagnosis of many of these cases, although the diagnosis of the case described by Sandifer (1950) is well substantiated on clinical grounds.

In the past 10 years 800 cases of poliomyelitis have been admitted to this hospital. In only one of them, the case described, was there sensory change.

CASE REPORT

On September 8, 1959, a man aged 21 developed pain in the back and had little sleep that night. Next day he complained of headache and pins-and-needles in the legs, and found he could not walk. He was admitted to another hospital on that day and transferred to the neurorespiratory unit on September 11 because respiration was failing.

On admission his temperature was 103° F. (39.4° C.). He had marked stiffness of the neck and back. There was slight weakness in both arms, the tendon reflexes being reduced; his vital capacity was 1,000 c.cm.; spinal and abdominal muscles were very weak, and the lower limbs were completely paralysed. There was some retention of urine. He had complete loss of all forms of sensation up

Complement-fixation Test for Poliovirus

	Type I	Type II	Type III
September 14	<5	<5	<5
October 12	20	<5	<5
.. 17	20		

to the level of L2 dermatome, both sides. The cerebrospinal fluid contained 21 cells and 262 mg. of protein per 100 ml. No spinal block was found. Blood was taken for complement-fixation test for poliovirus on September 14 and on October 12 and 17 (see Table). A specimen of faeces was obtained on September 29, and from this type I poliovirus was grown.

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The patient was placed in a tank respirator soon after admission. In the next few days he developed abdominal distension and vomiting. He lost the power of swallowing, and his vital capacity fell to 450 c.cm. A tracheostomy was performed under local anaesthesia, and positive-pressure respiration was started through a cuffed tracheostomy tube. By September 15 his acute illness had come to an end and he began to improve. The power of swallowing returned on September 21. By September 24 his vital capacity had increased to 800 c.cm. and artificial respiration was no longer required. By this date sensation was beginning to improve, but retention of urine persisted until November 23. Sensation continued to improve and the upper limit slowly descended. By January 2, 1960, the only sensory abnormality was sensation of cold in the legs. By then the vital capacity was 2,500 c.cm. The power in the upper limbs had much improved, although the recovery in the lower limbs was slight. He was soon afterwards transferred to another hospital for long-term orthopaedic treatment.

COMMENT

The complete loss of sensation within a well-defined upper border and a prolonged retention of urine were striking features in this case, which otherwise presented the picture of poliomyelitis, confirmed by the finding of virus in the stool and a rising complement-fixation titre in the blood. The cases described by Plum and Sandifer, three in all, present a very similar picture with complete loss of sensation up to the same level, retention of urine, complete flaccid paralysis in the legs, as well as scattered weakness in the upper limbs. Their patients, however, failed to recover any power in the legs and their sensation only partly returned. Plum (1956) believed that the changes in the legs in his cases were due to the destruction of the sensory nerve cells in the posterior horn of the spinal cord, which has been described in many cases coming to necropsy, although he recognized that clinically the picture was one of transverse myelitis.

Destruction of the nerve cells of the posterior horn is common in cases which come to necropsy and has been seen regularly in patients dying at this hospital, but in none of these cases has there been any sensory loss of life. As Bodian (1949) makes clear, in these severe cases the destruction of the posterior horn cells is patchy. Sharrard (1955) has shown that 40% or more of the motor cells have been destroyed in every case with the slightest degree of paralysis. It is probable that sensory-cell destruction never reaches this extent. If loss of sensation was due to destruction of the posterior horn cells one would expect it to occur often in severe cases, to be slight in degree, and to be scattered. In contrast the loss of sensation in this case and in the three mentioned was complete and had a definite upper limit. It is probable, therefore, that some process different from that commonly occurring in poliomyelitis is taking place. The diagnosis of transverse myelitis, possibly due to a vascular lesion, as suggested by Sandifer (1950), provides a more satisfactory explanation for the clinical picture during the acute stage and the subsequent progress of this case and the others under discussion.

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