TABLE II.—*Effect of Propranolol 10 mg./l. on Contour of Transmembrane Action Potential of Ventricular Fibres of Hypertrophic Cardiomyopathic and Control Human Cardiac Tissue*

	Resting Potential (mV)	Amplitude (mV)	Duration (msec.) Measured to Repolarization Within 5 mV of Resting Potential	Upstroke Velocity (V/sec.)
Control (5)	96	122	310	235
After propranolol 10 mg./l.	96	121	300	195
Hypertrophic cardiomyopathy (6) ..	95	120	500	186
After propranolol 10 mg./l.	95	118	440	148

Values are mean of the number of preparations studied in the control and number of different cells in the cardiomyopathic tissue (number in parentheses).

action potential cannot be explained on the basis of present speculative concepts of transmembrane electrolyte concentrations or ionic fluxes. A block in the normal potassium transmembrane permeability would delay repolarization and

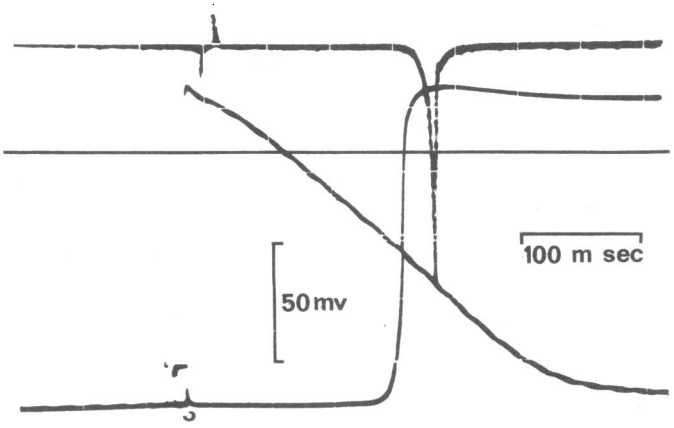

FIG. 2.—Typical human (control) ventricular transmembrane action potential. The horizontal line indicates zero potential; the superimposed middle traces depict intracellular potentials at slow and fast sweep speeds. The upper trace first shows the stimulus artefact followed by the output from the differentiator; the depth of the spike is proportional to dv/dt of the intracellular record.

prolong the refractory period; an antiarrhythmic property. A restraint on potassium permeability, however, would be expected to reduce the resting potential but this was unchanged in the present study.

Hypocalcaemia increases the duration of repolarization of the action potential mainly by prolonging the rapid early phase of repolarization and only to a lesser extent the slower and later phase as seen in the cardiomyopathic tissue. The effect of hypocalcaemia would be exaggerated by hypomagnesaemia. It therefore seems unlikely that an abnormal transmembrane permeability of any one of these electrolytes could explain the observed action potential, but a combination of a deficient flux of all three could be postulated.

Freedberg *et al.* (1970) have shown in the atria of thyroidectomized rabbits that the duration of the repolarization phase was greatly prolonged, yet it was shortened in the atria of another group rendered thyrotoxic. The patient in this study was euthyroid. The prolonged repolarization in hypertrophic cardiomyopathy might be expected to give a prolonged Q-T interval in the electrocardiogram. No such change was observed in the present case, and prolonged Q-T intervals have not been reported in this condition. We have, however, shown that there is a poor correlation between the Q-T interval as measured from the electrocardiogram and the duration of the action potential (Coltart *et al.*, 1970).

Normal subjects can generally respond with 1:1 A-V conduction to atrial rates up to 200/min. Linhart *et al.* (1965) found an average basal functional refractory period of 350 msec. in patients without abnormalities of A-V conduction, which would thus allow maximum rates of follow of 170/min. This agrees well with the maximum rate of follow of 160/min. in the control human tissue in this study, the difference being possibly due to the diseased state of the papillary muscle excised from these patients undergoing mitral valve replacement.

The gross reduction in the maximum rate of follow to 72/min. in the hypertrophic cardiomyopathic muscle shows an inability of this tissue to shorten its effective refractory period and would explain the late and irregular sequence of propagation of the activation impulse through the ventricular cavity previously supposed to be due to discrete myocardial fibrosis or stretching of the conduction tissue.

Meerschwam (1969) reported delayed and polyphasic action potentials from the skeletal muscle of patients with hypertrophic cardiomyopathy. He suggested that there might be a generalized myopathy even though these people rarely manifest any skeletal muscle disability. Thus there is further evidence for an electrophysiological abnormality in this disease.

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D. J. COLTART, M.R.C.P.,
Cardiologist.
S. J. MELDRUM, B.SC.,
Physicist.
St. Bartholomew's Hospital, London E.C.1.

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Medical Memoranda

Recurrent Meningitis

British Medical Journal, 1970, **4**, 218–219

Meningitis appears to have been previously unreported as a complication of decubitus ulcers; in the case described it occurred in association with sacral or perineural cysts. It is suggested that the association of sacral cysts with osteomyelitis of the adjacent sacrum as the result of decubitus ulceration may have facilitated entry of infection to the subarachnoid space.

CASE REPORT

A 43-year-old spinster with an 18-year history of seropositive rheumatoid arthritis causing little disability developed active hepatitis in August 1968. Three months later she had staphylococcal septicaemia, which apparently arose from a primary focus of infection in a toe, and this illness was complicated by acute renal failure and jaundice. The infection responded well to penicillin, and renal function returned to normal on a conservative regimen. She was started on prednisone 20 mg. daily to control the hepatitis. During the initial period of her illness she was semicomatose and immobile, and by the fifth day a decubitus

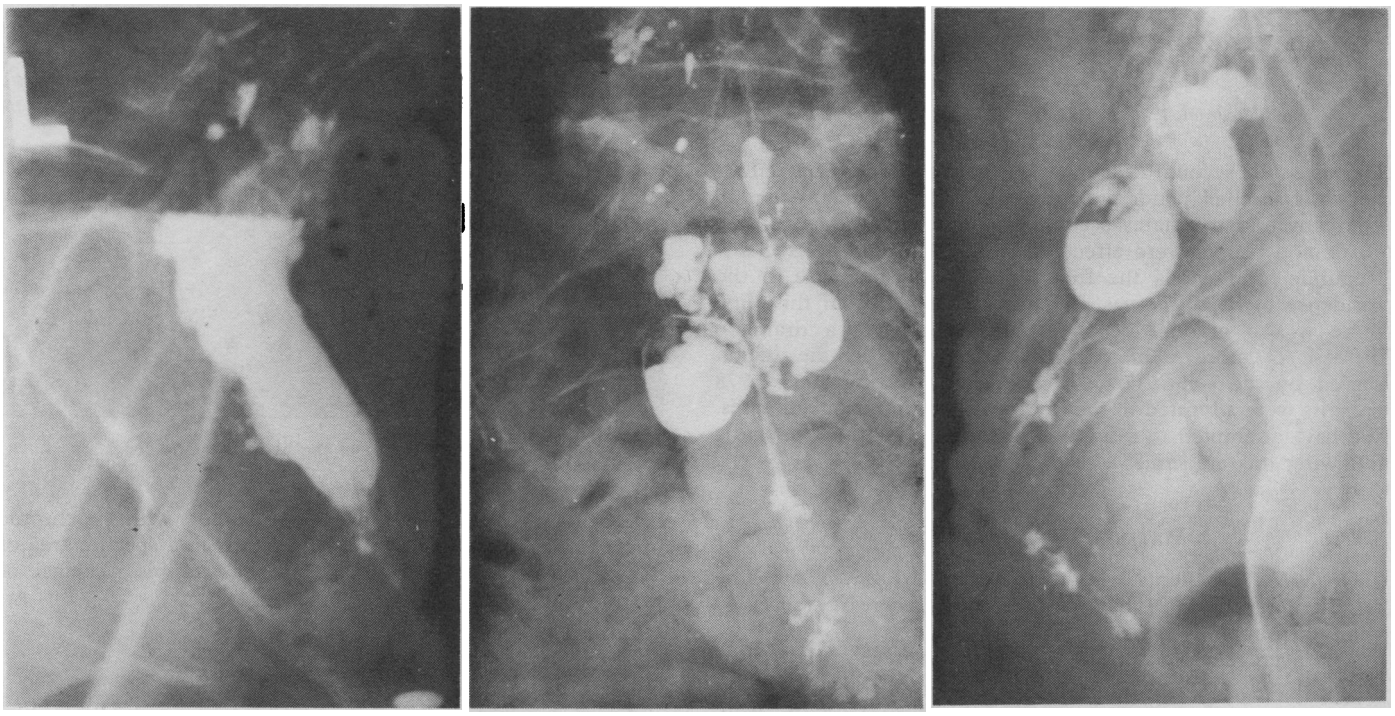

FIG. 1.—Erect film at 2 hours showing small collection of iophendylate at level of 4th segment. FIG. 2.—Anteroposterior view at 24 hours showing several contrast-filled cyst-like spaces on each side of midline, overlying lower half of sacrum. FIG. 3.—Lateral view showing cysts; in addition contrast outlines a narrow track running forward through destroyed part of sacrum and ending in region of natal cleft.

ulcer appeared over the sacrum. At the time of discharge she was no longer jaundiced and renal function had returned to normal, but the sacral ulcer, 5 cm. in diameter, persisted, though she was ambulant. Prednisone dosage was 7 mg./day. *X*-ray examination of the sacrum showed no abnormality.

Four months later she presented with symptoms and signs of meningitis of 12 hours' duration. Cerebrospinal fluid contained 1,000 white cells/cu. mm. and scanty Gram-positive cocci were seen, but organisms were not grown for culture. The sacral pressure sore was now larger and grossly infected. A swab taken on admission grew *Proteus* species, *Escherichia coli*, and *Staphylococcus aureus*. Blood cultures were sterile. Treatment was started with cephaloridine and kanamycin. Next day benzylpenicillin was added and streptomycin was substituted for kanamycin. Initial improvement was followed after four days by relapse, and on this occasion a *Bacteroides* organism sensitive to chloramphenicol was cultured from the cerebrospinal fluid. Antibiotic treatment was accordingly changed, with good response, but eight days later recurrence of symptoms necessitated further lumbar puncture, which showed infection with *Pseudomonas pyocyanea*. Symptoms again resolved on intensive chemotherapy with carbenicillin 24 g. daily and gentamicin 120 mg. daily, both given by intravenous infusion, together with oral probenecid. *X*-ray examination of the sacrum showed osteomyelitis of the middle portion. After two weeks of intravenous antibiotic therapy a skin graft was applied to the raw area over the sacrum; but this failed to take, and when after a further week antibiotics were stopped there was a prompt recurrence of meningitis. Culture of the cerebrospinal fluid failed to grow any organism, but there was a satisfactory response to reintroduction of carbenicillin and gentamicin.

Because of the likelihood of a communication from the area of sacral osteomyelitis to the subarachnoid space a myelogram was performed. The lowest portion of the subarachnoid space appeared normal, but an erect film obtained two hours later showed a small collection of iophendylate (Myodil) at the level of the fourth segment (Fig. 1). When a series of films were taken at 24 hours several contrast-filled cyst-like spaces were seen on each side of the midline overlying the lower half of the sacrum (Fig. 2). In addition the contrast outlined a narrow track which ran forwards through the destroyed part of the sacrum, ending in the region of the natal cleft (Fig. 3). The patient was transferred to Stoke Mandeville Hospital for closure of the sore. This was performed in two stages under cover of continuous therapy with intravenous carbenicillin and gentamicin. The wound healed satisfactorily and she was discharged after seven weeks. *X*-ray appearances of the sacrum were consistent with healing of the osteomyelitis. She remained well on 7 mg. of prednisone daily.

At no time were any abnormal neurological signs detected in the legs, nor was there any bladder dysfunction. There was no history of low back pain or trauma to the spine previous to her 1968 admission.

COMMENT

In the present case recurrent episodes of meningitis together with the isolation of several different organisms from the cerebrospinal fluid were strongly suggestive of a communication between the subarachnoid space and the infected area of bone underlying the decubitus ulcer. The myelogram findings appeared to confirm the presence of such a communication and in addition showed an unexpected anomaly—namely, multiple cystic spaces lying within the sacral spinal canal and filling from the subarachnoid space. These were likely to be perineural or Tarlov (1938) cysts. The presence of a communication between the cerebrospinal fluid and the sacral ulcer posed a problem regarding the most effective form of treatment, but debridement of the infected area together with closure of the skin defect gave a satisfactory result.

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O. G. BROOKE, M.B., B.S.

P. M. FORD, M.B., M.R.C.P.

A. MACKINTOSH, M.B., CH.B., D.M.R.D.

Department of Medicine and Radiology, Hammersmith Hospital London W.12.

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