drome and pyramidal tract damage in the folate-deficient patients. When we compared alcoholic with non-alcoholic folate-deficient patients the incidence of organic brain syndrome and positive Babinski response was similar in the two subgroups, but cerebellar syndrome and neuropathy were more common in alcoholics, suggesting that the trend towards an increase of neuropathy with folate deficiency (table II) may have been more a function of alcoholism than folate deficiency alone.

Both folate-deficient and control groups also contained patients with other medical conditions known to cause neurological complications (malignant disease, diabetes, partial gastrectomy, chronic renal disease, malabsorption, and malnutrition). These diagnoses, however, were fairly evenly distributed between the two groups. Furthermore, after these patients and the alcoholics were excluded there was again a remarkable increase of neurological complications in the remaining folate-deficient patients even though the numbers in each group were small (table V).

It is difficult to draw conclusions about the relation between folate deficiency and neurological disease in patients whose neurological disorders, even in individual cases, are probably multifactorial in origin. Although it is probable (1) that organic neurological disease can lead to folate deficiency as a result of dietary deficiency (Batata *et al.*, 1967) and (2) that nutritional factors were an important cause of deficiency in many of our patients, our findings are also consistent with the hypothesis that folate deficiency can in certain circumstances result in neurological complications, in particular organic brain syndrome and pyramidal tract damage. Our data provides encouragement for more detailed studies in larger and more homogeneous populations. Little is known about the biochemical basis of the neurological complications of alcoholism apart from the role of thiamine (Victor *et al.*, 1971), and the possible contribution of folate deficiency deserves further evaluation.

Among additional factors which might possibly have contributed to the neurological problems in the present study we only assessed the role of vitamin B₁₂. Only one patient was vitamin B₁₂ deficient and her neurological examination showed nothing abnormal. In our understanding of the effects of either folic acid or vitamin B₁₂ deficiency on the nervous system an explanation is required for the many instances with either vitamin deficiency in which no neuropsychiatric disturbance is

found. This may reflect, at least in part, variations in the severity and duration of the deficiency. Although the serum folate level may not always be a good guide to the folate status of the patient, in this study we deliberately chose patients with very low serum values. It is clear that these patients were indeed folate deficient as all except one had a macrocytic anaemia. However, a further problem with this type of study is the uncertain relation of serum folate to brain folate. Although a good correlation between serum and C.S.F. folate concentrations has been found (Reynolds *et al.*, 1972), it has yet to be shown that this is necessarily reflected in changes in brain folate. There is experimental evidence, however, that folate deficiency can impair cerebral nucleic acid metabolism (Haltia, 1970).

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MEDICAL MEMORANDA

Contamination of E.C.G. Electrode Pads with *Klebsiella* and *Pseudomonas* Species

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Any moist site in the hospital environment provides a potential site for the survival and multiplication of Gram-negative bacilli, particularly *Pseudomonas* species (Parker *et al.*, 1971).

The important reservoirs and routes of infection for *Klebsiella* species have not been so clearly defined. Until recently, epidemiological studies have been hindered by there being no centre for *klebsiella* typing in this country (*Lancet*, 1967). Important hospital epidemics with *Klebsiella* species have shown some sources, including a hand-cream dispenser (Morse, *et al.*, 1967), aerosol solutions used in respiratory apparatus (Mertz *et al.*, 1967), and intravenous fluids (Sack, 1970; House of Commons, 1972).

Any new reservoir or unfamiliar route for these organisms presents a particular threat to the patient. We report here a patient with *klebsiella* septicaemia, associated with contamination of saline used to moisten his electrocardiograph (E.C.G.) electrode pads, as well as the contamination of commercially prepared electrode pads with *Pseudomonas aeruginosa*.

Case Report

On 22 September 1972 a 43-year-old man with extensive coronary artery disease underwent a triple saphenous vein, aorta to coronary artery, bypass graft operation using cardiopulmonary bypass. Vein grafts were taken from the lower leg. Antibiotic cover started with the premedication and consisted of gentamicin 120 mg intramuscularly

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12-hourly for 48 hours and then 80 mg intramuscularly 12-hourly for a further three days. Flucloxacillin, 250 mg six-hourly, was given intravenously for the first 48 hours and then by mouth for a further three days.

An intravenous catheter inserted at operation into the right atrium via the left innominate vein was maintained postoperatively. Limb electrodes, with saline-soaked pads beneath the metal contacts, were left in place for several days. On the second postoperative day the patient had a rigor and became hypotensive, with a pulse rate of 140/min and a temperature of 101°F (38.3°C). Clinically there was a reduction of cardiac output with a cold poorly-perfused periphery. There was no change in the E.C.G. Without alteration of antibiotic treatment the patient became haemodynamically stable within six hours. A blood culture taken at this time yielded a Gram-negative bacillus after three weeks' incubation. On the fourth postoperative day, when the gentamicin had been reduced to 80 mg 12-hourly, a second blood culture yielded a similar Gram-negative bacillus after 24 hours' incubation. A primary source of infection was not apparent. Urine culture on three occasions gave no significant growth. The patient was discharged for convalescence 14 days after his operation.

The patient was nursed in a postoperative recovery ward which is visited regularly by the clinical pathologist. On the fifth postoperative day it was noted that the patient's E.C.G. electrode pads were routinely moistened with saline kept near the bed in an open 200-ml glass vessel. This saline, originally from a sterile bottle of saline, was sampled and yielded a Gram-negative bacillus similar to that isolated from the patient's blood.

This finding prompted the bacteriological investigation of commercial electrode pads. These premoistened lint pads (4 by 6 cm) are supplied in sealed foil envelopes with five pads in each envelope.

Blood was cultured by inoculating 5 ml of the patient's blood into two bottles, one containing 50 ml Brewer's medium and the other containing 50 ml of 0.1% glucose digest broth plus penicillinase and *p*-aminobenzoic acid. Each bottle was subcultured on 5% horse blood agar (Oxoid) after 24 hours and then twice weekly.

Electrode pad saline from the ward was inoculated directly on blood agar. Commercial pads were removed from their foil packets and a drop of fluid was inoculated directly on blood agar plates and cultured for 18 hours at 37°C. Bacterial counts were made using the Miles and Misra (1938) technique on blood agar.

Identification methods were based on those of Cowan and Steel (1966). Christensen's urea medium was used for detection of urease activity. Frazier's (1926) method for detecting gelatin liquefaction was used. Sensitivity testing was done by routine disc methods on 5% lysed horse blood agar. Minimum inhibitory concentrations were performed using surface inoculation of solid media (Oxoid D.S.T. CM 261) containing antibiotic.

Klebsiella typing was performed using commercial antisera (Difco) to the 72 known capsular (K) strains of *Klebsiella* species in accordance with the method described elsewhere (Casewell, 1972). Pyocine typing of *Ps. aeruginosa* followed the technique described by Gillies and Govan (1966).

Blood Cultures.—The Gram-negative bacillus isolated from both blood cultures grew as a mucoid colony, was non-motile, catalase positive, oxidase negative, and attacked glucose fermentatively. Acid and gas were produced from lactose and glucose, while fermentation of inositol, mannitol, dulcitol, and sucrose produced acid only. The methyl-red reaction was variable and the Voges-Proskauer reaction was positive. Urease activity was found and the KCN test was positive. Citrate and malonate were utilized and gluconate was oxidized. Indole was produced, but gelatin was not liquefied. Lysine, but no ornithine or arginine decarboxylation occurred. H₂S was not produced. The organism was thus identified as *Klebsiella aerogenes*. Specific capsular typing sera showed a strong positive quellung reaction to titre with specific serum 68 only. The organism was thus typed as capsular (K) type 68. On routine disc sensitivity testing the organism appeared sensitive to ampicillin, carbenicillin, tetracycline, co-trimoxazole, streptomycin, gentamicin, chloramphenicol, kanamycin, colistin, and cephaloridine. The minimum inhibitory concentrations for ampicillin and gentamicin were 32 µg/ml and 2 µg/ml respectively.

Saline Used for Moistening E.C.G. Electrode Pads.—This yielded a Gram-negative bacillus indistinguishable in all respects from the *Kl. aerogenes* K 68 isolated from both blood cultures.

Commercial Electrode Pads.—Twenty-six of the 41 electrode pad envelopes tested gave a growth of a motile Gram-negative bacillus, catalase and oxidase positive that metabolized glucose oxidatively. Pyocyanin and fluorescein pigments were produced. The organisms were thus identified as *Ps. aeruginosa* and belonged to pyocine types 6, 3, and 33. Bacterial counts on fluid expressed from the pads in three envelopes taken at random showed 1.8×10^7 , 2.4×10^7 , and 1.1×10^7 organisms/ml.

Comment

There seems little doubt that the patient acquired his *Kl. aerogenes* K 68 blood infection from the E.C.G. electrode pads moistened with saline contaminated with the same serotype. The organisms were isolated within 48 hours from the two sites and there was no clinical or bacteriological evidence for any other primary source of infection.

Colonization of nurses' hands with *Klebsiella* species has been previously described (Salzman *et al.*, 1967), and it seems likely that chest wall skin adjacent to a moist warm contaminated electrode would become similarly colonized. It is then not difficult to envisage organisms gaining access to the blood via an adjacent venous catheter. Indeed, this patient had a right atrial catheter passed into the left innominate vein. *Klebsiella* capsular type 68 has not been reported as a particularly prevalent strain in the few reports of *klebsiella* serotypes isolated in hospitals. It occurred twice in the same renal transplant patient in 172 intensive care unit strains recently isolated and typed at St. Thomas's Hospital (unpublished). This patient was not having E.C.G. recordings.

Although we do not have evidence of patients developing bacteraemia from commercially prepared electrode pads, the finding that 26 out of 41 were contaminated with *Ps. aeruginosa* seems to present a dangerous avoidable hazard to intensive care patients. Gram-negative infections are steadily increasing (Lockey *et al.*, 1973) and bacteraemia after heart surgery is by no means uncommon. At the National Heart Hospital, 40 out of 2,180 open-heart surgery patients developed proved Gram-negative bacillaemia between 1967 and 1972. Nearly all these were receiving intensive therapy. The elimination of contaminated electrode pads may well reduce the risk of infection in these patients.

Intensive care units should be alerted to this hitherto undescribed, but potentially dangerous, source of infection.

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