atmospheres pressure was also given, the first treatment being started shortly after his admission in Aberdeen and each session lasted two hours.

On the morning after his transfer to the unit, he was pyrexial, pale, and slightly jaundiced. Inspection of the wound showed a small area of necrosis in the vastus lateralis and pale oedematous areas in the vastus medialis and the anterior part of the tensor fascia lata. These parts of the muscles did not respond to mechanical stimulation, and they were therefore excised under general anaesthesia. The haemoglobin now was 66% (9.6 g/100 ml) despite the transfusion of 2,000 ml of blood. The serum bilirubin level was 2.5 mg/100 ml. The serum aspartate level was 51 IU and the aspartate transferase level 102 R.F. units. The excised muscle was examined bacteriologically and showed profuse numbers of Gram-positive rods confirmed by culture to be *C. welchii* with a high concentration of alpha toxin production.

After a further five periods of hyperbaric oxygen treatment and three days later the patient was much improved clinically with a steady haemoglobin level, absence of jaundice, and a near normal temperature. The wound showed no further evidence of extension of the infection and there was only scanty growth from one of three swabs taken from the wound. This was now dressed daily with Furacin (0.2% nitrofurazone in water-miscible vehicle). He improved rapidly and the wound showed healthy granulation tissue appearing throughout. His further course was uncomplicated.

Comment

Gas gangrene, although well known, is a fairly rare complication in surgical practice. It has been estimated by Altmeier and Furste (1948) that the incidence after major open wounds is less than 2%, although the same writers found clostridial contamination to be present in up to 39% of bacteriologically cultured accidental wounds. There is also evidence from the Korean war that clostridial contamination of war wounds was present in 88% of cases. Infection usually starts at the site of injury or operation. Nevertheless, there are a few cases reported where the infection occurred in a tissue distant from the site of injury. Brummelkamp (1966) in a series of 55 patients reported one patient who developed gas gangrene in a leg after cholecystectomy. MacLennan (1962) on examining the world literature found a number of reports of non-traumatic infections in which *C. welchii* was identified. In this connexion it should be remembered that clostridial spores can remain dormant in scar tissue for years after the original injury.

In the present case it is difficult to explain the course of events. Close questioning of the patient revealed that he had had pain in his right thigh since a horse-riding accident one week before the car accident. He also admitted to have

injected himself with drugs, but never in the thigh and not during the preceding 12 months. He maintained he had knocked the right thigh in the car accident and he did complain of pain, but nothing was found on the initial clinical examination.

If we accept that the patient never gave himself hypodermic injections of drugs in the thigh, there seems only two possible explanations. The incubation period of gas gangrene is usually less than three days but infection may start from one hour to six weeks after the injury (MacLennan, 1962). It is possible that the infection started when the patient fell off a horse a week before the car accident, the site of entry of organisms being either a minor abrasion or from the gastrointestinal tract (MacLennan, 1962). The more likely explanation, however, is that the patient had a bacteraemia of *C. welchii* as a consequence of the open fracture of the left leg and that there existed anoxaemic tissues in the form of injured muscle or a recent or an old haematoma in the right thigh which afforded the anaerobic conditions in which *C. welchii* could grow producing clostridial myositis.

The place of hyperbaric oxygen treatment in patients with gas gangrene infection has been frequently debated and it is not the purpose of this communication to argue for or against its beneficial effect. Suffice it to say that we believe the mainstay of gas gangrene treatment is surgery combined with a high dosage of an antibiotic, preferably penicillin. Hyperbaric oxygen should be regarded as an ancillary measure in the treatment of gas gangrene (Irvin et al., 1968), although Roding *et al.*, (1972) claim that hyperbaric oxygen is the treatment of choice in gas gangrene infection.

At this time when road traffic accidents are becoming a more frequent cause for admission of patients to hospital, gas gangrene is likely to increase in frequency. We feel that this case history is a reminder to the profession that it may occur at sites distant from the sites of injury.

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Syndrome Resembling Kwashiorkor after Partial Gastrectomy

RICHARD WALDRAM

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A patient who developed protein-calorie malnutrition 20 years after a partial gastrectomy complicated by depression and a diet grossly deficient in protein is reported. This syndrome, which closely resembles kwashiorkor, has rarely

been reported in the Western world and usually follows gastric surgery complicated by other factors.

Case History

The patient, a man of 54, was admitted to another hospital where his wife gave the history of abdominal swelling with dyspnoea increasing for several months. Before admission he had become increasingly slow and confused. Over several years there had been progressive loss of appetite and he became apathetic and thin. In 1948 he had a Polya partial gastrectomy for duodenal ulcer, followed by a vagotomy a year later for recurrence of symptoms. He had not suffered from indigestion since. He drank four pints (2 l.) of beer daily and some spirits. He had broken his false teeth six months before admission and failed to obtain a new set.

At the referring hospital he was confused, irritable and slow. There was leg oedema and some ascites, but the jugular venous pressure was not raised. There were no stigmata of cirrhosis and the spleen was not felt, but the liver was enlarged. The cranial nerves were normal, but the limbs were wasted and weak with brisk

St. Thomas's Hospital, London SE1 7EH

RICHARD WALDRAM, M.B., M.R.C.P., Medical Registrar (Present address: Liver Unit, King's College Hospital, London S.E.5)

reflexes and increased tone. Plantar responses were flexor. A clinical diagnosis of cirrhosis with portal-systemic encephalopathy was made, and an E.E.G. showing slow waves (4-7 Hz) appeared to support the diagnosis.

He was given neomycin, frusemide, Slow-K, chlorpromazine, and a low protein diet, but he deteriorated further. Ascites increased despite diuretics, and he became more confused and aggressive when disturbed. He was given an infusion of salt free albumin (50 g) and diuretics were stopped but his weight remained constant and he was transferred to this hospital.

On examination he was listless, irritable, and uncooperative. The signs elicited previously were unchanged but it was noted that he had leuconychia and dry, atrophic skin. There was "crazy pavement" cracking of the keratin with erythema in the fissures (eczema craquelé) seen best on the forearms, legs, thighs, buttocks, and lower back. Hair was lost from these areas and from the scalp.

His wife said that he had been depressed and had taken a diet consisting largely of canned soups and fruit for about two years. A detailed assessment by our dietitian subsequently corroborated by the patient, showed that his daily intake of protein was 23 g with some 1,900 calories. The diet appeared adequate in other respects. The clinical diagnosis was therefore protein-calorie malnutrition associated with a poor diet and a gastrectomy.

Investigations were: Hb 13.1 g/100 ml, normal film; E.S.R. 5 mm in 1 hour; electrolytes, normal; urea 17 mg/100 ml; serum albumin 2.5 g/100 ml, globulin 1.3 g/100 ml, SGOT 78 U/ml (normal 20-110); alkaline phosphatase 7 K.A. U/ml; bromsulphalein retention 69% at five minutes, 24% at 45 minutes; liver scan normal; faecal fats 0.9 g/day. CrCl₂ test excluded significant enteric protein loss.

TREATMENT AND PROGRESS

The only initial change in therapy was a high protein diet (100 g), and during the next month his recovery was remarkable. He gained 17 kg and lost all oedema, his mental state and appetite improved dramatically, and the skin lesions cleared. Serum albumin rose and the E.E.G. became normal. A jejunal biopsy performed at this time showed normal villous architecture.

Subsequent progress has been disappointing although protein deficiency has not recurred. He was readmitted recently with a cardiomyopathy and sensory peripheral neuropathy. Because these

were likely to be due to alcohol a liver biopsy was performed, and showed an active cirrhosis without Mallory's hyaline.

Comment

The dietary history, the clinical signs, and his response to treatment leave little doubt that at the first admission he was suffering from protein-calorie malnutrition. The precipitating factors were his previous partial gastrectomy coupled with depression and loss of his teeth. He has now been shown to have cirrhosis and indeed the two conditions may have co-existed, although there is no direct relation between them (McLaren *et al.*, 1968). The E.E.G. appearances are common to both, but it is highly unlikely that a patient with portal-systemic encephalopathy would make a dramatic recovery on a high protein diet. Although subsequent events have shown the toxic effects of alcohol in this man, his mental state did not resemble delirium tremens and he was never hallucinated.

The cardinal features of kwashiorkor are oedema, irritable apathy, atrophic skin lesions, and the development of red hair. It occurs in children after weaning, and adult cases are unusual even in developing countries. Only 20 cases have been reported in the West. Neale (1968) reviewed the literature and added seven cases. All followed gastric surgery complicated by the contaminated bowel syndrome, pancreatic insufficiency, or anorexia. The exceptions are isolated cases associated with coeliac syndrome, pancreatic insufficiency, and ulcerative colitis, all with anorexia (Jeejeebhoy, 1964), and one which was the result of a food fad alone (Jegenburg, 1968).

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Addiction to Prednisone

H. GETHIN MORGAN, JULIAN BOULNOIS,
CHRISTOPHER BURNS-COX

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It is well known that cortisone and its analogues when taken in high dosage may cause acute mental disturbance such as delirium (Engel and Romans, 1959), in about 50% of cases a euphoric rise of mood being the main clinical feature (Michael and Gibbons, 1963). It follows that when steroids are used therapeutically subjective improvement may not only be related to actual suppression of the underlying organic disease

process but also to euphoria. Further, it is well known that reduction of steroid medication, especially when carried out rapidly after prolonged therapy, may lead to a temporary insufficiency of adrenal output of steroids. Unpleasant psychological effects may therefore be very real during steroid withdrawal, and may be misinterpreted as a recrudescence of the patient's disease process. Kimball (1971) reported two such patients, one with bronchial asthma and the other with rheumatoid arthritis. We report a case of bronchial asthma in which the psychological dependence on prednisone reached such a degree that it acquired all the characteristics of an addictive state.

Case Report

A 22-year-old single woman was referred urgently to the out-patient clinic by her general practitioner. She showed symptoms of intense anxiety and expressed fears of recurrence of bronchial asthma although there was no objective evidence of bronchospasm. During interview she was restless and tremulous, smoked heavily, and she consumed tablets of prednisone and Valium in twos and threes. Three days previously she had been prescribed 100 tablets each of diazepam (5 mg), prednisone (5 mg), and salbutamol (2 mg) and by the time she came to the clinic she had consumed nearly all of these. She was also seen to use a salbutamol inhaler frequently. Prednisone medication had started some seven years previously, but this had been intermittent until six months before her referral, when she began to take increasing doses daily.

Department of Mental Health, University of Bristol, Bristol BS8 1TH
H. GETHIN MORGAN, M.R.C.P., M.R.C.PSYCH., Consultant Senior Lecturer

Glenside Hospital, Bristol BS16 1DD
JULIAN BOULNOIS, M.B., CH.B., Senior House Officer

Frenchay Hospital, Bristol
CHRISTOPHER BURNS-COX, M.D., M.R.C.P., Consultant Physician