which depending to some degree on the plasma lipid level. Hypertension therefore accelerates and intensifies the atherosclerotic process. It is of significance that personality may also affect cholesterol and lipid metabolism, as it has been shown that emotional stress and activation can cause a rise in serum lipids.^{2,6}

A number of measures have been adopted to counteract this increasing incidence of coronary thrombosis. Reduction in weight and smoking are obvious, but the other measures such as anticoagulant or fibrinolytic therapy and the reduction of blood lipids by dietary or drug therapy have so far given inconclusive though often beneficial results. Early reduction of a raised blood pressure, labile or sustained, should therefore lower the incidence of coronary thrombosis. Propranolol may be used, as it diminishes cardiac output and pulse rate. The other hypotensive agents available today have already proved effective in preventing the abnormal rise in blood pressure in people exposed to stressful situations. Sedation in adequate doses to achieve the same result would have to be too powerful.

It is possible that certain predisposed persons have to pay a price in the form of medication for keeping up with their environment, which causes coronary thrombosis to assume almost epidemic proportions in the western world.—I am, etc.,

ERIC FRANKEL.

Wanstead Hospital,
London E.11.

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Unusual Case of Emphysema

SIR,—A Royal Naval diver, at the outset of a recent trials series, sustained accidental damage to his left circumorbital region, on the night before doing an air dive to 300 feet in a compression chamber. Before diving he was sent to me, and as the injury was only minimal I passed him fit to dive. Four days previously he had had mild coryza, which had since cleared up. The chamber was compressed to the equivalent of 300 feet of sea water and the diver had no trouble with the descent. After 15 minutes at 300 feet the ascent to the first stop at 100 feet was begun. At about 260 feet the diver reported a sharp pain in his left cheek which was then observed by all present to be gradually swelling as the pressure was reduced. I entered the chamber at 70 feet to assess his condition.

On examination he had marked swelling of his left cheek and eyelids, and palpable crepitations. There being nothing to gain by recompressing him, I aspirated what air I could as he continued the decompression breathing oxygen. Using a 10 ml. syringe with a 21 gauge, 1½ in. (3.75 cm.) needle, I aspirated from a point about ¼ in. beyond the lateral margin of the orbit, the needle entering obliquely under the skin to about ¾ in. In spite of the withdrawal of 5 ml. of air at 50 feet and a further 5 ml. at 20 feet,

the swelling increased until it covered the entire left side of his face from neck to temple. At the surface the pain had gone and his condition was as shown in the photographs. His discomfort was further relieved by the aspiration of another 8 ml. of air. It was apparent that he must have sustained a minute fracture into his maxillary sinus, the ostium of which was presumably sufficiently obstructed by the residual inflammation of his recent coryza for the air to be forced out through the fracture instead of draining through the ostium. Regrettably two series of x-rays failed to reveal the location of the fracture. Later the same day he reported a few specks of blood in his nasal secretions.

During the following five days the swelling gradually disappeared, leaving only the bruising of his original injury. He passed from my care shortly after, and was returned to diving about six weeks later with no recurrence.

A similar case of swelling of the right cheek and side of forehead, accompanied by pain over the maxillary sinus, is reported in a civilian diver in July 1968, following a dive to 100 feet. Again there was no demonstrable maxillary sinus fracture on x-ray (Personal communication from J. M. Young, Surgeon Lieutenant Commander, Royal Naval Physiological Laboratory, Alverstoke.)—I am, etc.,

D. R. LEITCH.

H.M.S. Reclaim,
B.F.P.O. Ships.

Delayed Neurological Effects of Electrical Accidents

SIR,—Your leading article (18 January, p. 135) prompts me to report a case of electrical injury of interest because of the neurological sequelae and the initial presentation in coma—a cause rarely listed in the differential diagnosis of the unconscious patient.

A 61-year-old man was admitted having been found unconscious in the engine-room of his houseboat. On examination he was stuporous, but responded to painful stimuli. The upper limb reflexes were absent, with marked spasticity in the right arm. There were no other abnormal neurological signs. There was a reddened area of skin over the left side of the lower back and a small superficial burn over the left elbow, but no other signs of external injury. Temperature 98.4° F. (32° C.), B.P. 130/80, pulse 80 per minute, sinus rhythm, heart sounds normal, no

murmurs. Examination of the chest and abdomen was normal. Lumbar puncture revealed clear C.S.F. under normal pressure with less than two leucocytes cu. mm., protein 50 mg./100 ml., sterile on culture, C.S.F. and serum W.R. negative.

His conscious level improved over the next 12 hours, but he remained disorientated and confused for a further 24 hours. During this time he complained of severe muscle spasm in the back and lower limbs. Forty-eight hours after the initial episode he was noted to have marked weakness in both arms, mainly involving the long extensors of the wrists and fingers. There was sensory blunting to touch and pinprick over the lateral aspects of the right forearm and hand. The upper limb reflexes remained absent. There were no abnormal signs in the lower limbs or cranial nerves. Electromyography confirmed the weakness was of neural rather than muscular origin. A normal conduction velocity in the right ulnar nerve supported the clinical diagnosis of a spinal cord and not a peripheral nerve lesion.

The patient recalled later that his last memory before the episode was of going to turn off the 240-volt A.C. mains supply. The electrical system was examined subsequently and found faulty. During the next six weeks the muscle weakness gradually improved, though he still experienced difficulty in fine movements of the hands. The sensory loss resolved except for impairment to touch and pinprick in the right index finger.

I should like to thank Dr. S. R. F. Whittaker for his permission to report this case.

—I am, etc.,

R. N. ALLAN.

Department of Medicine,
Royal Postgraduate Medical School,
London W.12.

Draining Loop Colostomy

SIR,—In his letter (18 January, p. 187) Mr. W. M. Keynes refers to the obsolescence of Paul's tubing and this I agree. The time-honoured glass rod can also be eliminated by the simple device of making the skin incision to accommodate the loop not linear but in V-shape. The apex of the V is drawn under the loop and the edges of the flap are sutured back around the limbs of the colostomy. This skin bridge supports the loop and thus disposes of the discomfort of a glass rod; moreover, the recession of the loop into the abdominal wall, a complication which sometimes occurs after removal of a glass rod, cannot take place.—I am, etc.,

St. George's Hospital
Medical School,
London S.W.1.

BRYAN N. BROOKE.

Management of Acute Salicylate Poisoning

SIR,—Dr. R. C. Mac Keith's letter (18 January, p. 185) suggests that a blood pH done in the casualty department would probably be life-saving in salicylate poisoning. Two recent authorities^{1,2} state that the change in the pH may be minimal and may not reflect accurately the degree of acidosis present in severe salicylate poisoning.

The most important investigation in salicylate poisoning is a rapid estimation of the plasma salicylate level. There are two simple sideroom techniques available now^{3,4} which allow such an estimation to be obtained