

As far as the case report of Dr Lewis and others is concerned, it would be useful to know if the patient had acute or fulminant changes of exophthalmos, for without these changes their patient would be similar to one of our own who did not respond. The lack of response in such patients is probably due to permanent fibrotic changes in extraocular muscles. In this regard it would be extremely useful to have a simply measured biochemical marker which would predict potential responders. For the moment, however, it appears that one would have to depend on the clinical indications such as those described above; this is not necessarily a compromise since it is mostly in association with these features that the threat to visual acuity occurs. Plasmapheresis is not a treatment for cosmetic discomfort but a possible alternative to orbital decompression. We agree that this procedure needs further assessment.

P DANDONA
N MARSHALL
S BIDEY
A W NATHAN
C W H HAVARD

Metabolic Unit,
Royal Free Hospital,
London NW3 2QG

Autonomic neuropathy in the Guillain-Barré syndrome

SIR,—Further to your correspondence regarding heart rate variation in diabetes mellitus and in tetraplegic patients (19 May, p 1353) we would like to report a case of Guillain-Barré syndrome in which serial tests showed the presence and then resolution of an autonomic neuropathy.

A 30-year-old man presented to the casualty department with a seven-day history of numbness paraesthesiae of both legs and hands and a left facial weakness which had been present for two days. On examination, his temperature was 37.2°C; pulse rate 124 beats/min, regular; and blood pressure 120/80 mm Hg lying down, 110/80 mm Hg standing. There was a left lower motor neurone facial palsy, a proximal weakness of all four limbs, and sensory loss of a glove and stocking distribution with absent deep tendon reflexes. Investigations: haemoglobin concentration 15.9 g/dl, white blood cells (WBC) $10.5 \times 10^9/l$; cerebrospinal fluid WBC $< 1 \times 10^6/l$; protein 2.3 g/l. Electromyography showed slowing of sensory and motor conduction.

A diagnosis of Guillain-Barré syndrome was made and corticosteroid therapy started. In view of the marked resting tachycardia serial tests for autonomic neuropathy were performed. The heart rate response to standing was conducted as described by Ewing *et al*¹ and the "30:15 ratio" derived. The variation in heart rate during respiration was recorded by continuous electrocardiographic monitoring during deep breathing (six deep breathing manoeuvres in one minute) and the E:I ratio of the mean of the longest R-R interval during maximal expiration to the mean of the shortest during maximal inspiration was calculated as described by Sundkvist *et al*.² For the first three days of his admission there was a persistent tachycardia and both the "30:15" and the E:I ratios were compatible with vagal neuropathy and autonomic dysfunction. His neurological signs were unchanged. On the fourth day, however, these ratios returned to within the normal range. This was followed by a gradual resolution of the neurological deficit.

The Guillain-Barré syndrome has been associated with a variety of circulatory abnormalities³ and vasomotor disturbances have been accepted as a sudden cause of death.⁴ Thus autonomic function should be

monitored closely in this syndrome, both to assess progress and to provide objective parameters with which to assess therapy.

M E EDMONDS
R D STURROCK

Department of Therapeutics,
Westminster Medical School,
London SW10 9TH

- ¹ Ewing, D J, *et al*, *British Medical Journal*, 1978, **1**, 145.
² Sundkvist, G, *et al*, *British Medical Journal*, 1979, **1**, 924.
³ Birchfield, R I, and Shaw, C-M, *Archives of Neurology*, 1964, **10**, 149.
⁴ Appenzeller, O, and Marshall, J, *Archives of Neurology*, 1963, **9**, 368.

Diving and hypothermia

SIR,—Another two divers in the North Sea have died from hypothermia and I should like to add to the recent contributions on this subject (5 May, p 1182; 9 June, p 1566; 25 August, p 494).

Divers under water lose heat from the body surface and through respiration, the latter in two parts—(a) the humidification of dry inspired gas and (b) absolute warming of the cold inspired gas. With a helium-oxygen respiratory mixture the absolute warming part of the respiratory heat loss becomes much more important than in air. Under normal circumstances heat supplied from the support ship to the diving bell via the umbilical cord counteracts the heat loss; but in any accident normal sources of power and heat will almost certainly be lost, as happened in the Thistle field, or at best reduced. When this happens the men are in trouble and emergency equipment should therefore be effective without requiring any outside source of power.

Respiratory heat loss could be reduced by means of a heat exchanger which also acts as a condenser humidifier. A soda-lime-filled canister, as used in anaesthetics but with the addition of insulation, could fulfil this need. The soda-lime granules would initially act as a passive-condenser humidifier and heat exchanger, but as the expired carbon dioxide reacted with the soda lime to produce heat and moisture the system would gradually change to one providing a positive heating effect. Of course, as the soda-lime became exhausted the canister would revert to a passive heat and moisture conserver. A single canister should nevertheless provide about six hours of positive heating and CO₂ absorption.

The provision of the insulated canisters would encroach on the space in the diving bell but a space of 30 × 15 cm diameter per man might not seem excessive for an emergency. The other problems would be (a) fitting the canister into the diver's breathing system; (b) a slight increase in resistance to breathing; and (c) a gradual increase in dead space as the soda lime became exhausted.

E LL LLOYD

Department of Anaesthetics,
Royal Infirmary,
Edinburgh EH3 9YW

"Herbal" medicines and rheumatoid arthritis

SIR,—Dr P G J Forster and others (4 August, p 308) rightly warn against the use of Chuei-Fong-Tou-Geu-Wan pills apparently imported from Holland, for the treatment of

rheumatoid disorders. However, they did not need to refer to a newspaper as a source of reference, since the matter has received wide publicity in the Dutch medical press.

After the first warning that analysis of such pills had shown the presence of indomethacin and dexamethasone, and that cases of Cushing's syndrome and withdrawal symptoms after stopping treatment had been reported,¹ a case similar to Dr Forster's was published,² involving a patient who had been using 12 pills daily containing an average of 75 mg indomethacin, 112 µg dexamethasone, and small quantities of diazepam and hydrochlorothiazide. These pills had been manufactured by the Nan Lien Pharmaceutical Co Ltd in Hong Kong. Similar pills from the Singapore subsidiary of the same firm were shown to be composed of prednisone 0.4 mg, hydrochlorothiazide 4 mg, chlorthalidone 0.5 mg, chlorpheniramine maleate 1.3 mg, and thiamine disulphide 5 mg; whereas the pills marketed by the Shou Sing Pharmaceutical Co Ltd in Taiwan contain varying (0.6-6 mg) amounts of phenylbutazone, about 8 mg of aminopyrine, and some thiamine. Moreover, the Yi Chung Tai Medical Manufacturing Co Ltd in Taiwan markets Chi-Shi-Ton pills containing 8.1 mg paracetamol, 0.26 mg ethaverine hydrochloride, 2 mg chlorzoxazone, 0.55 mg diazepam, 1.9 mg caffeine, and some thiamine.³ In none of these cases was the presence of these drugs mentioned on the package or the directions folder. Obviously patients suffering from rheumatoid arthritis run great risks in the unauthorised use of such adulterated pills.

Though marketing—mostly by small Chinese dealers—of these pills in the Netherlands is alleged, and since October 1978 has been officially condemned, a total embargo on this dangerous practice seems almost impossible to enforce. Patients should therefore be clearly warned against the use of any Chinese so-called herbal pills. This is not the only example of its type; similar instances (Amborum Spezial F, Tsai-Tsao-Wan, "nose pills," and Swasahar) have been reported from Germany, Australia, and the United States.⁴

L OFFERHAUS
M N G DUKES

Division of Pharmacotherapy,
Central Drug Inspectorate,
Ministry of Public Health and
Environmental Hygiene,
Leidschendam, The Netherlands

H M SMITS

State Institute for Drug Research,
Leiden, The Netherlands

¹ Offerhaus, L, *Nederlandsch Tijdschrift voor Geneeskunde*, 1978, **122**, 1633.

² Uitdehaag, C M J, *Nederlandsch Tijdschrift voor Geneeskunde*, 1979, **123**, 1009.

³ Central Drug Inspectorate, *Nederlandsch Tijdschrift voor Geneeskunde*, 1979, **123**, 1347.

⁴ Dukes, N M G, (editor), *Side Effects of Drugs Annual 3*, Amsterdam, Excerpta Medica, 1979.

SIR,—It might be of interest to your readers that in addition to the Chinese herbal medicine Chuei-Fong-Tou-Geu-Wan, on which Dr P J G Forster and others report (4 August, p 308), four more exotic drugs—Amborum spesial F, Tsaitsowan, Nose Pill, and Swasahar—claimed to be pure herbal remedies, were found by the German Medicines Inspection Institute (Deutsches Arzneiprüfungsinstitut, DAPI), an independent laboratory of the pharmaceutical profession, to contain dexamethasone or prednisolone or both. The