[1] Howarth WG. Dilatation of the oesophagus without anatomical stenosis. *Proc R Soc Med* 1919;**12**, Section 1 and 2 Laryngology:64.
[2] Smithers DW. Achalasia, hiatus hernia, muscle hypertrophy, columnar-cell oesophageal mucosa and their association with oesophageal tumours. In: Tanner NE, Smithers OW, eds. *Tumours of the oesophagus*. Edinburgh: Livingstone, 1961:61-80.
[3] Seaman WB, Wells J, Flood CA. Diagnostic problems of oesophageal cancer. *AJR* 1963;**90**:778-91.
[4] Tucker HJ, Snape WJ, Cohen S. Achalasia secondary to carcinoma: manometric and clinical features. *Ann Intern Med* 1978;**89**:315-8.
[5] Kelley ML Jr. Intraluminal manometry in the evaluation of malignant disease of the oesophagus. *Cancer* 1968;**21**:1011-8.
[6] Goldin NR, Burns TW, Ferrante WA. Secondary achalasia: association with adenocarcinoma of the lung and reversal with radiation therapy. *Am J Gastroenterol* 1983;**178**:203-5.
[7] Davis JA, Kantrowitz PA, Chandler HL, Schatzki SC. Reversible achalasia due to reticulum-cell sarcoma. *N Engl J Med* 1975;**293**:130-2.
[8] Kline MM. Successful treatment of vigorous achalasia associated with gastric lymphoma. *Dig Dis Sci* 1979;**25**:311-3.
[9] Kolodny M, Schrader ZR, Rubin W, Hochman R, Sleisenger MH. Esophageal achalasia probably due to gastric carcinoma. *Ann Intern Med* 1968;**69**:569-73.
[10] Serebro HA, Venkatachalam B, Prentice RSA, Neuman HW, Beck IT. Possible pathogenesis of motility changes in diffuse esophageal spasm associated with gastric carcinoma. *Can Med Assoc J* 1970;**102**:1257-9.
[11] Bessent CT, Lopez CA, Cocco AE. Carcinoma of the EG junction mimicking achalasia. *Md State Med J* 1973;**22** (Jun):47-50.
[12] Shulze KS, Goresky CA, Jabbari M, Lough JO. Esophageal achalasia associated with gastric carcinoma: lack of evidence for widespread plexus destruction. *Can Med Assoc J* 1975;**112**:857-64.
[13] Benjamin SB, Eisold J, Gerhardt DC, Castell DO. Central nervous system lymphoma presenting as dysphagia. *Dig Dis Sci* 1982;**27**:155-60.
[14] Sandler RS, Bozymski EM, Orlando RC. Failure of clinical criteria to distinguish between primary achalasia and achalasia secondary to tumor. *Dig Dis Sci* 1982;**27**:209-13.
[15] Menin R, Fisher RS. Return of esophageal peristalsis in achalasia secondary to gastric cancer. *Dig Dis Sci* 1981;**26**:1038-44.

The heart in the Guillain-Barré syndrome

The Guillain-Barré syndrome, or Landry's ascending paralysis, is an acute immunologically mediated predominantly motor neuropathy. It is characterised by a raised protein concentration without an increase in the cell count in the cerebrospinal fluid. The disease progresses for between two and four weeks, during which the flaccid motor paralysis tends to ascend, hence Landry's eponym. If the chest muscles are affected it may be necessary for the patient to be ventilated. In some cases disease goes on to affect the cranial nerves.

The autonomic nervous system is responsible for moment to moment regulation of heart rate, the force of myocardial contraction, and the capacitance of the blood vessels and their resistance to forward flow. The cardiac nerves therefore control cardiac output, arterial pressure, and perfusion of the tissues. The sympathetic and parasympathetic preganglionic cells receive both excitatory and inhibitory impulses from the cardiovascular centres in the medulla and from spinal neurones.

Autonomic dysfunction may occur in the syndrome, particularly in those who show weakness of the respiratory muscles.[1] Vasomotor control may thus be disturbed, sometimes with hypertension (perhaps because the carotid sinus is affected) and sometimes with postural hypotension. Abnormal sweating and pupillary disturbances may occur. Patients with the Guillain-Barré syndrome may die suddenly, and since necropsies have not shown a cause and arrhythmias are a recognised complication[2] of the disease, death has been attributed to abnormalities of the cardiac nerves. Both bradycardia and paroxysmal tachycardia may occur and the patient may need to have a demand pacemaker inserted.[2-4] In a survey of patients treated at the Mayo Clinic the syndrome was fatal in about 20% of children whose trunk and respiratory musculature was affected.[4] The authors of that paper suggested that cardiac monitoring should be instituted whenever assisted ventilation is needed (as would be wise in any acutely ill patient requiring mechanical ventilation).

Some deaths might be avoided if we had a test to predict potentially dangerous impairment of the cardiac autonomic system. A reduction of the normal beat to beat variation in heart rate may occur in the Guillain-Barré syndrome and is thought to indicate that the vagus nerve is affected.[5] Persson and Solders have described a study of six patients with the syndrome in whom the electrocardiogram was recorded during two minutes of normal breathing and, when possible, during two minutes of deep breathing, after which consecutive R-R intervals were plotted against time and the variation in R-R interval in relation to the mean R-R interval calculated both for normal and for deep breathing.[6] The recordings were repeated serially and compared with control recordings. All six patients showed similar results, with a gradual decrease in beat to beat variation, which became maximal two to four weeks after the onset of symptoms. This pathological regularity of the heart was most pronounced in those with the most severe muscle paralysis. Vagal paresis was suggested by finding an almost fixed sinus tachycardia of over 100 beats a minute. Three patients later showed periods of fluctuation in blood pressure or bradycardia, or both. Improvement in R-R variation paralleled clinical recovery. The most abnormal findings were in the two patients who needed mechanical ventilation, while control studies in two other patients on ventilators but with no neurological disease showed no abnormality.

Although variations in R-R interval tend to decrease with increasing heart rate, and sinus arrhythmia is usually associated with sinus bradycardia, the Swedish workers did not believe that the tachycardia by itself was responsible for the decrease in R-R variation and the simple observation of tachycardia alone would not suffice as an indication of impaired vagal function because the differences in relative R-R interval variation were greater in the serial studies than changes in the recorded heart rate. Possibly, therefore, serial measurement of the R-R interval in patients with severe disease affecting the trunk and respiratory musculature may identify those patients at risk of developing fatal arrhythmias.

CELIA M OAKLEY

Consultant Cardiologist,
Royal Postgraduate Medical School,
London W12 0HS

[1] Lichtenfield P. Autonomic dysfunction in the Guillain-Barré syndrome. *Am J Med* 1971;**50**:772-80.
[2] Clark E, Bayliss RIS, Cooper R. Landry-Guillain-Barré syndrome: cardiovascular complications. *Br Med J* 1954;ii:1504-7.
[3] Favre H, Foex P, Guggisberg M. Use of demand pacemaker in a case of Guillain-Barré syndrome. *Lancet* 1970;i:1062-3.
[4] Emmons PR, Blume WT, DuShane JW. Cardiac monitoring and demand pacemaker in Guillain-Barré syndrome. *Arch Neurol* 1975;**32**:59-61.
[5] Frison JC, Sanchez L, Garnacho A, Bofill J, Oliveró R, Miquel C. Heart rate variations in the Guillain-Barré syndrome. *Br Med J* 1980;**281**:649.
[6] Persson A, Solders G. R-R variations in Guillain-Barré syndrome: a test of autonomic dysfunction. *Acta Neurol Scand* 1983;**67**:294-300.

Correction

Assessment of pituitary function

We regret that the 13th line of the 8th paragraph of Dr T D R Hockaday's leader on "Assessment of pituitary function" (10 December 1983, p 1738) was incorrect. It should have read ". . . growth hormone releasing hormone deficiency) on single shot."