metry may be due to a "disinhibition" rather than a slow reorganisation homologous with the emergence of spasticity.

1 Bolton B, Carmichael EA, Sturup G. Vasoconstriction following deep inspiration. *J Physiol* 1936;**86**:83–94.
2 Gilliatt RW, Guttmann L, Whitteridge D. Inspiratory vasoconstriction in patients after spinal injuries. *J Physiol* 1948;**107**:67–75.
3 Cole JD, Mani R, Sedgwick EM. Cutaneous vasomotor reflexes following spinal cord injury in man. *J Physiol* 1985;**369**:134P.
4 Oberg PA, Tenland T, Nilsson GE. Laser-doppler flowmetry: A non invasive and continuous method for blood flow evaluation in microvascular studies. *Acta Med Scand Suppl* 1983;**687**:17–24.
5 Johnson RH, Spalding JMK. *Disorders of the autonomic nervous system*. Oxford: Blackwell Scientific Publications, 1974:120.
6 Chevallier PE. *De la paralysie des nerfs vaso-moteurs dans l'hemiplegie, These de Paris*. Paris: E Martinet, 1867.
7 Ellis LB, Weiss S. Vasomotor disturbance and oedema associated with cerebral hemiplegia. *Arch Neurol Psychiatry* 1936;**36**:363–72.
8 Gowers WR. *A manual of diseases of the nervous system*. London: J and A Churchill Ltd, 1886;**2**:77.
9 Kennard MA. Vasomotor disturbances resulting from cortical lesions. *Arch Neurol Psychiatry* 1935;**33**:537–45.
10 Blumberg H, Wallin BG. Direct evidence of neurally mediated vasodilatation in hairy skin of skin of the human foot. *J Physiol* 1987;**382**:105–21.
11 Hagbarth K-E, Hallin RG, Hongell AA, Torebjork HE, Wallin BG. General characteristics of sympathetic activity in human skin nerves. *Acta Physiol Scand* 1972;**84**: 164–76.
12 Wallin BG, Stjernberg L. Sympathetic activity in man after spinal cord injury. *Brain* 1984;**107**:183–98.

A classic description of subacute combined degeneration of the spinal cord

Amongst the early descriptions of cord lesions in association with anaemia was Lichtheim's,[1] but confusion with tabes and other degenerative diseases prevailed when Leichtenstern[2] described the same affection as "Progressive Pernicious Anaemia in Tabetic patients." The most assiduous account was in a 70 page paper by JS Risien Russell, FE Batten and James Collier[3] from Queen Square. They described nine patients with autopsies in seven. The illness was in three stages:

"1 A stage of slight spastic paraplegia with slight ataxy and marked subjective sensations in the lower limbs.

2 A stage of severe spastic paraplegia with marked anaesthesia of legs and trunk.

3 A stage of complete paraplegia; absent knee jerks; absolute anaesthesia; rapid wasting and loss of faradic excitability in the muscles of the paraplegic region; increase of superficial reflex excitability; absolute incontinence of both sphincters and oedema of the lower extremities and trunk.

A lengthy detailed description, illustrated by sensory charts follows. The disease appeared in the fourth and fifth decades, average age 40; four of their seven necropsy cases were female and there was a slight family history in two cases. In respect of the anaemia they noted:

". . . some of the most typical cases presented no anaemia throughout the course . . . others only late in the disease, while in other cases anaemia was an obtrusive symptom from the first and preceded the nervous symptoms by many months."

The course was rapid, duration four and three months respectively in cases 2 and 6, or chronic in cases 5 and 9. There were many odd features we would not today recognise as part of the illness. Cranial nerve palsies, convulsions and pyrexia developed in certain instances. "Lightning pains in the legs occurred, and . . . in all cases a severe constant dragging pain beneath the lower costal margin, always unilateral . . . Herpes occurred in two cases and cutaneous haemorrhage, in a third, in nerve root distribution . . . Most patients died within four months of losing the ability to walk. Duration varied between three months and thirty months, averages under nine months."

Pathologically: "the stress of the disease fell on the mid-dorsal region of the cord . . . very marked destruction of the white matter all round the periphery . . . not in any way limited to the long tracts. "The posterior columns, which are generally most affected, may be completely sclerosed . . . Lesions as high as the upper medulla or mid pons" (stained by Marchi and Weigert-Pal methods) . . .

Both "a focal destructive lesion and a system degeneration" were recognized . . . In most cases the peripheral nerves were found to be normal or showed only very slight changes, but case 1. showed very considerable degeneration.

These pathological studies are detailed and excellently illustrated. They repay minute study, rather than abbreviation here. Six theories of causation are elaborated, with a final preference for an unidentified toxin which damages both the blood and cord.

It was not until 1926 that Minot and Murphy discovered the "liver factor" which reversed pernicious anaemia, prolonged life, and later was found to contain the vital cobalamin—vitamin B_{12}.

JMS PEARCE

1 Lichtheim L. Verhandl des VI. Congresses f. innere Med 1887; *Neurol Centrabl* 1887;**6**:235.
2 Leichtenstern *Deutsch Med Woch* 1884;**10**:849.
3 Russell JSR, Batten FE, Collier J. Subacute Combined Degeneration of the Spinal Cord. *Brain* 1900;**23**:39–110.