"All tibial foot" with sensory crossover innervation between the tibial and deep peroneal nerves

One of the most common and well studied innervation anomalies in the upper limbs is the Martin-Gruber anastomosis.^{1,2} In the lower limbs, the anomaly is uncommon except for the accessory deep peroneal nerve.^{1,2} Recently, an exclusive innervation of the extensor digitorum brevis by the tibial nerve, "all tibial foot" has been reported.³⁻⁵ We experienced a similar patient with "all tibial foot", who, in addition, showed sensory anomaly.

A 23 year old man with encephalitis had nerve conduction studies (NCSs) to exclude coexistent peripheral neuropathy. The studies were normal except for the anomalous innervation in the bilateral lower limbs. Peroneal nerve stimulation at the ankle, fibular head, and popliteal fossa elicited only a negligible compound muscle action potential (CMAP) over the extensor digitorum brevis. The accessory deep peroneal nerve was not demonstrated by stimulation behind the lateral malleolus.^{1,2} A normal CMAP from the extensor digitorum brevis was elicited by stimulating the tibial nerve at the ankle and popliteal fossa (figure A). Although CMAP of the anterior tibial muscle was normally elicited by stimulating the common peroneal nerve at the fibular head, a small CMAP was recorded also by the stimulation of the tibial nerve at the popliteal fossa (figure B).

Sensory studies of the sural, superficial peroneal and medial plantar nerves were normal. Stimulation of the deep peroneal nerve at the ankle gave rise to a normal antidromic sensory nerve action potential (SNAP) in the skin between the first and second toes, where an obvious SNAP was recorded even after the stimulation of the tibial nerve behind the medial malleolus (figure C).

Our patient had "all tibial foot" for the motor innervation, the anomalous dual innervation of the anterior tibial muscle, and the sensory coinnervation of the skin between the first and second toes by the tibial and deep peroneal nerves. Findings were similar on both sides. We speculate that, in our patient, the deep peroneal nerve becomes almost pure sensory after branching motor fibres for the anterior tibial muscle, and that the extensor digitorum brevis is innervated by the tibial nerve. Further, the tibial nerve had a motor branch for the anterior tibial muscle and a sensory branch to supply the area typically innervated by the deep peroneal nerve. Although rare, we should keep in mind this anomaly in the practice of nerve conduction studies.

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(A) Stimulation of the tibial and deep peroneal nerves at the ankle and popliteal fossa. Recordings from the extensor digitorum brevis. (B) Stimulation of the tibial and deep peroneal nerves at the popliteal fossa. Recordings from the anterior tibial muscle. The second negative peak (◆) is probably made by the volume conduction from the simultaneously contracting gastrocnemius muscle (◆◆). (C) Stimulation of the tibial and deep peroneal nerves at the ankle. In either case, sensory nerve action potential (arrow) was recorded from the skin between the first and second toes.

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IgG Anti-GT1a antibodies which do not cross react with GQ1b ganglioside in a pharyngeal-cervical-brachial variant of Guillain-Barré syndrome

IgG anti-GT1a antibody which cross reacts with GQ1b ganglioside is associated with ophthalmoplegia in Guillain-Barré syndrome and in Fisher's syndrome, a variant of Guillain-Barré syndrome.¹ Pharyngeal-cervical-brachial weakness is another regional variant of Guillain-Barré syndrome originally described by Ropper.² Its symptoms resemble those of botulism and diphtheria and are characterised by marked oropharyngeal, neck, and shoulder weakness with areflexia only in the arms. We report on a patient with this variant who had high titres of serum IgG anti-GT1a antibody which did not cross react with GQ1b.

A 55 year-old woman was admitted due to sudden onset ptosis, trouble with swallowing, and nausea and vomiting. One week before admission she had had an upper respiratory infection with diarrhoea which lasted for several days. On admission, dilated pupils and ocular paralysis were noted. Fever of up to 40°C lasted for a week. Her neurological symptoms were at their worst on day 5. At that time, she was alert but could not open or move her eyes or swallow anything. Her pupils were dilated with sluggish light reflexes. The tip of her tongue did not protrude from her mouth. The Medical Research Council grades for the facial muscles ranged from 3 to 4; neck flexion and extension were graded 2; the deltoid and the biceps brachii and triceps muscles 4. Muscle strength in the lower limbs was normal. Deep tendon reflexes were absent in all the limbs, and the Babinski sign was negative. There was no sensory disturbance or cerebellar ataxia. She complained of headache, nausea, and vertigo on turning her head, but no meningeal signs were present. Her white blood cell count (15 600/ μ l) and serum C-reactive protein (9.0 mg/dl) were raised. Routine laboratory findings for liver and renal functions were normal. On day 5 her CSF had a raised protein concentration of 80 mg/dl, and

cells were 7/ μ l. Motor conduction velocity in the right tibial nerve on Day 11 was slightly reduced (37 m/s), whereas the motor and sensory conduction velocities in the right medial nerve were normal. A stool culture was negative for *Campylobacter jejuni*. Botulism and diphtheria were excluded because her neurological symptoms occurred a week after the onset of diarrhoea. Results of the nerve conduction and CSF studies showed abnormal values. Guillain-Barré syndrome was diagnosed. A daily intramuscular injection (8 mg/day) of dexamethasone was given for 3 days then tapered off over the next 20 days. Two weeks after admission, her internal ophthalmoplegia and dysphagia had disappeared. One month later, ocular movements were almost normal, ptosis remained only in the right eye, the strength of the neck muscles had increased to 4, and deep reflexes became detectable. All these symptoms disappeared within 3 months of onset.

Serum antiganglioside antibody titres were determined using an enzyme linked immunosorbent assay (ELISA). On day 11, the titres of anti-GM1, GM2, GD1a, GD1b IgG, and IgM antibodies were <500; whereas the titre of the IgG anti-GT1a antibody had increased to 16 000 and that of the anti-GQ1b antibody was 500 (normal ranges were set at <500). No IgM antibodies to GT1a or GQ1b were detected. Thin layer chromatography with immunostaining confirmed that her serum IgG reacted with GT1a but not with GQ1b or GD1a (figure). An absorption study showed that her IgG anti-GT1a antibodies were not absorbed by GQ1b, GD1a, or GM1 (data not shown). Three months after onset, the respective serum titres of the anti-GT1a and anti-GQ1b IgG antibodies had decreased to 500 and <500.

The regional distribution of her symptoms was the same as that of the pharyngeal-cervical-brachial variant of Guillain-Barré syndrome, except for the areflexia of the legs. Based on clinical observations of three patients, Ropper² described this variant as sparing the power and reflexes in the legs. One of his patients, however, had generalised areflexia. We therefore classified our patient as having the pharyngeal-cervical-brachial variant of Guillain-Barré syndrome. Her headaches, nausea, and vertigo suggest the involvement of a central component.

Serum antiganglioside antibodies are present in patients with Guillain-Barré syndrome. Some of these antibodies are associated with certain clinical variants or signs of this syndrome. Chiba *et al.*¹ detected IgG anti-GT1a and anti-GQ1b antibodies in most of a group of patients with Fisher's syndrome or

Guillain-Barré syndrome with ophthalmoplegia and showed that the anti-GT1a antibodies in these patients cross reacted with GQ1b. Our patient with the pharyngeal-cervical-brachial variant of Guillain-Barré syndrome also had increases in IgG anti-GQ1b and anti-GT1a antibodies that paralleled the clinical course. The anti-GT1a antibody titre in our patient, however, was much higher than the anti-GQ1b antibody titre; moreover, the anti-GT1a antibody did not cross react with GQ1b. Mizoguchi *et al.*³ also detected IgG anti-GT1a antibodies which did not cross react with GQ1b, as well as anti-GD1a antibodies in the serum of a patient with this Guillain-Barré syndrome variant. Furthermore, O'Leary *et al.*⁴ reported IgG anti-GT1a and anti-GQ1b antibodies in three patients with acute oropharyngeal palsy. The presence of IgG anti-GT1a antibodies in these patients and in ours was associated with the emergence of acute polyneuropathy with marked lower cranial nerve involvement. IgG anti-GT1a antibodies which do not cross react with GQ1b may be closely related to the proclivity of Guillain-Barré syndrome to manifest oropharyngeal palsy or to the cause of its pharyngeal-cervical-brachial variant. The IgG anti-GT1a antibody present in Fisher's syndrome may recognise a structure common to GT1a and GQ1b gangliosides,¹ whereas in the pharyngeal-cervical-brachial variant it would react with another epitope specific to GT1a.

This research was supported in part by grants in aid from the Ono Medical Research Foundation, Uehara Memorial Foundation, Ciba-Geigy Foundation (Japan) for the Promotion of Science, the Nakabayashi Trust for ALS Research, the Ryoichi Naito Foundation for Medical Research, and by a Research Grant for Neuroimmunological Diseases from the Ministry of Health and Welfare Japan.

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Dexamethasone is not necessarily unsafe in primary supratentorial intracerebral haemorrhage

Controversy surrounds the role of steroids in the treatment of intracerebral haemorrhage.¹ Theoretically, the short term use of dexamethasone is justified because it lessens the damaging effects of cerebral oedema, decreases intracranial pressure, and strengthens the blood-brain barrier. However, the possible benefit has to be weighed against the risk of complications, especially infections and gastrointestinal haemorrhage.²

Thin layer chromatography with immunostaining. Thin layer chromatography plates stained with (A) orcinol/sulphuric acid for hexose; (B) IgG from our patient, subsequently stained by peroxidase conjugated antihuman IgG antibodies; (C) IgG from a patient with Bickerstaff's brain stem encephalitis. Lane 1: bovine brain ganglioside mixtures. Lane 2: the GD1a and GT1a fraction separated from bovine brain ganglioside mixtures by Q-sepharose column chromatography. Lane 3: fraction enriched with GT1a and GQ1b. The plates were developed with C-M-0.2% calcium chloride in water (5:4:1 by volume).