Four examples of acute symptomatic seizures and epilepsy developing after head injuries with golf balls are described, which seem to be an exception to these clinical rules.

An 11 year old boy was struck on the right temple by a golf ball resulting in right frontal scalp contusion. His consciousness was not impaired until about 3 hours later when he became drowsy and had two focal motor seizures affecting the left arm. He was intubated and ventilated. A head CT showed a right frontal extradural haematoma with no skull fracture (figure A). The haematoma was evacuated (figure B). He was woken and extubated the next day and was discharged without neurological impairment two days later on phenytoin. No further follow up is yet available.

A 16 year old boy, who was a keen golfer with a single figure handicap, was struck on the head by a golf ball which rebounded several yards after striking him on the forehead. He experienced local pain, bruising, and swelling. Although he was never unconscious, some 4-5 hours later he developed repetitive jerking of the right face and arm. He was taken to his local casualty department where a diagnosis of serial focal motor seizures was made. His consciousness was then somewhat obtunded. A brain CT was performed which showed a small, discrete, spherical intracerebral haematoma in the left frontal lobe immediately beneath the skull at the point where he had been struck (similar in shape, but more hyperintense than the appearance in the figure B). The haematoma gave the distinct impression of a golf ball embedded in the surface of the cerebral hemisphere!

He was treated with parenteral anti-epileptic drugs and subsequently with thiopentone requiring ventilation for 48 hours while he was loaded with phenytoin.

He was maintained on phenytoin for 12 months but subsequently this was withdrawn and he has remained seizure free.

A 5 year old girl was struck on the forehead above the right eye by a golf ball struck 10 metres away. On arrival in the accident and emergency department she was fully alert, orientated, and neurologically intact. A laceration was present but there had been no apparent impairment of consciousness or vomiting. However, 90 minutes after the injury she had a generalised tonic clonic seizure lasting 25 minutes. She was intubated and ventilated. A CT scan showed a very small depressed fracture with minimal haemorrhagic contusion in the cortex of the right frontal lobe (figure C). She was woken and extubated later that day. She has had no further seizures.

A 12 year old boy was practising golf with a friend. He was struck on the front of the head by a golf ball which rebounded a considerable distance after striking him. He did not lose consciousness and had no more than localised pain, tenderness, and bruising at the site of impact. He did not seek any medical advice about the injury. Over the next 4 years he had three well documented tonic-clonic seizures that started during sleep.

A CT scan 3 years after the original injury showed a small, wedge shaped area of low density affecting the cortex close to the point at which he recalls being struck (figure D).

The heads of these four young people were all subject to contact with golf balls which at club level travel at speeds of up to 130 miles/hour. Each had post-traumatic seizures, three early and one late, despite the apparent absence of post-traumatic amnesia.

Patients 1 and 2 would indicate that this kind of injury is capable of transferring energy across the skull, independent of a skull fracture, to cause an acute extradural or cortical haematoma.⁴ In patient 4 the lesion identified at a later date by CT is consistent with the late consequences of a localised intracranial haematoma. It therefore seems reasonable to assume that the late epilepsy in patient 4 was also related to the initial golf ball injury.

In the third patient, a minor depressed fracture and contusion were again associated with an early seizure without evidence of intervening impairment of consciousness.

It does therefore seem that golf ball injuries are capable of giving rise to both acute symptomatic seizures and late epilepsy without causing post-traumatic amnesia, skull fracture or dural tear. CT evidence, however, would predict the possibility of seizures in these examples in whom the development of post-traumatic epilepsy probably results from the physical properties of golf balls and their ability to transmit considerable mechanical energy at a small site of impact. The problem is one of which spectators on golf courses (and their doctors) should be aware.

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Sensory predominant neuropathy with GM₁ antibodies, conduction blocks, and orbital pseudotumour

Two male patients developed multifocal sensory neuropathy with high titre IgM anti-GM₁ antibodies (up to 1: 64 000) and infiltrative orbitopathy. Nerve conduction studies showed multiple motor conduction blocks and evidence of a similar phenomenon in sensory nerves. Both patients deteriorated after corticosteroid administration but benefited substantially from intravenous immunoglobulin therapy. Our findings suggest the existence of a predominantly sensory subtype of multifocal motor neuropathy (MMN) and challenge the postulated motor specificity of anti-GM₁ antibodies.

Anti-GM₁ antibodies have been implicated in the aetiology of multifocal motor neuropathy (MMN) and are assumed to be specific for this disease when occurring at high titres.^{1,2} We report on two patients with high titre IgM anti-GM₁ antibodies and electrophysiological features typical of MMN presenting with severe sensory neuropathy.

Patient 1 was a man who developed asymmetric numbness of limbs and difficulty in performing fine motor movements around the age of 55. Sensory deficits showed a multifocal pattern (multiple mononeuropathy) and involved proximal limb regions, trunk, and

face. The course of illness was steadily deteriorating with some episodes of prominent disease progression usually preceded by minor infections. After 10 years he was unable to write, needed assistance for dressing and walking, and complained of diplopia. Neurological examination showed profound loss of all sensory modalities in the arms and legs and pseudoathetosis of the fingers and wrist. Deep tendon reflexes were preserved and muscle strength was normal. The patient showed marked protrusion and downwards and outwards deviation of the left eye with a complex impairment of all eye movements.

Patient 2, a 68 year old man, reported an insidious onset and gradual worsening of asymmetric sensory loss and painful patchy dysaesthesia. At a neurological examination 15 years after disease onset, pinprick sensation, vibration, and proprioception were prominently affected in his arms and legs. There was severe ataxia of gait. Postural maintenance and voluntary movements strongly depended on visual guidance. Muscle strength was normal except for slight bilateral paresis of the tibialis anterior and intrinsic hand muscles (4+/5 MRC scale). There was no evidence of autonomic nerve or pyramidal tract dysfunction. Tendon reflexes were absent. The patient had an incomplete left side third nerve paresis and ipsilateral visual loss.

General physical examination was normal in both patients, as were repeated screenings for neoplastic and connective tissue diseases. Enzyme linked immunosorbent assays (ELISAs)³ showed IgM antibody activity against GM₁, asialo-GM₁, and GD_{1b}, presumably recognising the Gal(β1-3)GalNAc group (table). Clonality of ganglioside antibodies was not investigated. Serum immunoelectrophoresis did not show monoclonal gammopathy. All the following laboratory indices were normal or negative: creatine phosphokinase, erythrocyte sedimentation rate, renal and liver function, antinuclear antibodies, thyroxine, vitamins B₁₂, B₆, B₁₂, folic acid, urine porphobilinogen, and serum cryoglobulins. Cerebrospinal fluid was acellular with 52 mg/dl and 90 mg/dl protein, respectively (normal <50 mg/dl).

Nerve conduction studies showed multifocal slowing of motor nerve conduction velocities and F wave latencies (table). Despite near normal muscle strength we found motor conduction blocks at sites not prone to compression. Sensory nerve potentials could not be elicited in the median, ulnar, radial, or sural nerves. Electromyography showed fibrillation activity, generalised fasciculations, and features of chronic neurogenic damage. Magnetic resonance imaging of the brain, spinal cord, and dorsal nerve roots did not show relevant abnormalities. Both patients had non-progressive orbital infiltration with slight gadolinium enhancement suggestive of ectopic lymphoproliferative tissue. Lesion extension was most pronounced at the apex orbitae and fissura orbitalis superior and caused compression of the left optic and oculomotor nerves in one patient and mechanical interference with eye movement in the other. Analogous orbital infiltration has been described in patients with paraproteinemic neuropathies and antibody mediated autoimmune diseases such as myasthenia.

Administration of methylprednisolone at dosages of 40 to 60 mg/day was followed by marked deterioration of sensory ataxia in both patients. By contrast, substantial and rapid benefit was achieved by means of intravenous immunoglobulin (IVIg) therapy (0.4

Ganglioside antibody patterns and electrophysiological characteristics

Variable	Patient 1	Patient 2
Antibody titres:		
IgM GM ₁	1:64000	1:4000
IgG GM ₁	—	—
IgM asialo-GM ₁	1:32000	1:8000
IgG asialo-GM ₁	—	—
IgM GD _{1a}	—	—
IgG GD _{1a}	—	—
IgM GD _{1b}	1:500	1:32000
IgG GD _{1b}	—	—
IgM GT _{1b}	—	1:2000
IgG GT _{1b}	—	—
IgM GQ _{1b}	—	—
IgG GQ _{1b}	—	—
IgM sulfatide	—	—
IgG sulfatide	—	—
Nerve conduction studies:		
Right median nerve:		
DL (ms)/NCV ₁ /NCV ₂ (m/s)	4.5/48/56	4.6/32/65
F wave latency (ms)	33.0	43.6
Motor conduction block	+	+
SNAP	Absent	Absent
Right ulnar nerve:		
DL (ms)/NCV ₁ /NCV ₂ (m/s)	2.6/56/50	3.0/35/45
F wave latency (ms)	32.5	43.9
Motor conduction block	+	—
SNAP	Absent	Absent
Right peroneal nerve:		
DL (ms)/NCV ₁ /NCV ₂ (m/s)	6.6/42/39	5.2/33/31
F wave latency (ms)	86.9	62.5
Motor conduction block	+	+
SNAP	Absent	Absent

Antibody titres presented are those before treatment (baseline). SNAP=sensory nerve action potential; DL=distal latency; NCV=nerve conduction velocity in the following segments: median nerve: wrist-elbow (NCV₁, normal >50m/s), elbow-axilla (NCV₂, normal >56m/s); ulnar nerve: wrist-elbow (NCV₁, normal >50m/s), elbow-axilla (NCV₂, normal >55m/s); peroneal nerve: ankle-fibula neck (NCV₁, normal >42m/s), fibula neck-popliteal fossa (NCV₂, normal >41m/s).

g/kg body weight/day for five consecutive days). Gait normalised, fine motor movements and ophthalmoplegia markedly improved, and motor conduction blocks partially resolved. In addition, low amplitude sensory nerve action potentials reoccurred.

In the literature, there is circumstantial evidence of a pathogenic role of high titre IgM anti-GM₁ antibodies in the mediation of motor conduction blocks in MMN.^{1,2} Often, the antibody attack is directed at the Gal(β1–3)GalNAc epitope of GM₁. This carbohydrate group, however, is also an obligatory constituent of GD_{1b} (cross reactivity), which was implicated in the aetiology of sensory neuropathies. GD_{1b} distributes on human dorsal root ganglion cells and paranodal myelin and shows comparable immunostaining with anti-GM₁ antibodies with Gal(β1–3)GalNAc specificity and antibodies to disialosyl residues of GD_{1b}.⁴ This and various other findings challenge a motor specificity of anti-GM₁ antibodies and, instead, suggest a vulnerability of part of the sensory system. Actually, nerve conduction studies in previous series of patients with MMN showed numerous sensory abnormalities, as did pathological evaluations of sensory nerve biopsy specimens.^{5,6,7} We report on a predominantly sensory variant of MMN found in two male patients. Both had IgM anti-GM₁ and anti-asialo-GM₁ antibodies at titres high enough to be assumed specific for MMN.^{1,2} Despite near normal muscle strength both of our patients had multiple motor conduction blocks. Rapid clinical improvement after IVIg therapy despite a more than 10 year history of illness tempts us to speculate on a similar phenomenon in sensory nerves (sensory conduction block).

Good response to therapy argues against a pathogenetic significance of high titre GD_{1b} antibodies in patient 2, because the rare GD_{1b} associated sensory axonal neuropathy (sensory ganglionopathy) is mostly irreversible. In addition, this patient showed multiple con-

duction blocks and slowing of nerve conduction velocities rather than predominantly axonal damage and had no monoclonal gammopathy, which is usually present in cases with the GD_{1b} associated neuropathy.

We propose that multifocal neuropathies with conduction blocks and high titre anti-GM₁ antibodies have a clinical range from predominantly sensory to predominantly motor variants and suggest that all these variants be subsumed under the term “multifocal motor-sensory neuropathy (MMSN)”. This concept is of clinical relevance in that all phenotypes share the same therapeutic peculiarities including good response to IVIg and ineffectiveness (most cases) or even unfavourable effects of corticosteroids, which are first line drugs in the treatment of other immune neuropathies. Our report aims to increase awareness for sometimes prominent sensory involvement in MM(S)N and to facilitate early and accurate diagnosis in such patients.

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CORRESPONDENCE

Alterations of muscarinic acetylcholine receptor subtypes in diffuse Lewy body disease: relation to Alzheimer's disease

The article by Shiozaki *et al*¹ demonstrating significantly less muscarinic acetylcholine receptor binding sites in the temporal cortex in dementia with Lewy bodies than in Alzheimer's disease and different upregulation of the m1 and m2 receptor subtypes suggests differences in the manner of degeneration of the cholinergic system between both dementing disorders that may be of basic and practical therapeutic relevance. The more severe reduction of ChAT activity in the neocortex in dementia with Lewy bodies than in Alzheimer's disease, the higher upregulation of the postsynaptic m1 receptor in dementia with Lewy bodies, and the higher level of the presynaptic m2 receptor subtype in Alzheimer's disease suggest a severe depletion of presynaptic projection neurons in dementia with Lewy bodies but their relative preservation or upregulation in Alzheimer's disease.¹ These data are in line with previous—including personal—findings on cell loss and shrinkage in the cholinergic magnocellular posterior part of the nucleus basalis of Meynert in dementing neurodegenerative disorders.² In Parkinson's disease (brain stem type of dementia with Lewy bodies), cell depletion in the nucleus basalis of Meynert averages 30% to 40% without correlation with age or duration of the illness. It is much higher in demented patients with Parkinson's disease (similar to Alzheimer's disease range 50% to 70%) than in non-demented patients (0% to 40%), who show neuronal loss similar to or only slightly higher than age matched controls. Cell loss in the nucleus basalis of Meynert in non-demented patients with Parkinson's disease is usually associated with little or no cortical Alzheimer's disease pathology, whereas in severely demented patients with Parkinson's disease, heavy cell depletion in the nucleus basalis of Meynert is often, but inconsistently, accompanied by severe cortical neuritic Alzheimer's disease pathology suggesting threshold levels of cholinergic forebrain impairment and deficit for the development of dementia.² Even more severe depletion of the nucleus basalis of Meynert with 75% to 80% loss of large cholinergic neurons is found in dementia with Lewy bodies (figure). There were no major differences in cell loss in the nucleus basalis of Meynert between dementia with Lewy bodies with “plaque only” Alzheimer's disease (two cases) and with “true” Alzheimer's disease (eight cases with Braak stages V or VI). Lewy bodies and neurofibrillary tangles in the nucleus basalis of Meynert neurons were seen in eight brains of patients with Lewy body disease.³