

# Mechanism of production of gait unsteadiness by tumours in the posterior fossa

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**SYNOPSIS** The hypothesis is put forward that the disturbance of gait seen with tumours of the posterior fossa is largely due to subacute dilatation of the ventricular system and not to the involvement of midline cerebellar structures concerned with balance or the coordination of truncal muscles, as is widely taught. This hypothesis is examined in the light of the clinical evidence provided by the symptomatology and treatment of 19 consecutive cases of intracranial mass lesions exhibiting truncal ataxia.

It is well known that a disturbance of gait, often termed 'truncal ataxia' or 'dysequilibrium' occurs with mass lesions situated in the cerebellar vermis, and it is widely held that its presence is of value in localizing a tumour to that region of the brain. The mechanism by which the gait disorder is produced has long been believed to be by involvement of those midline structures of the cerebellum thought to be concerned with balance and with the coordination of truncal muscles. On the basis of clinical evidence, this paper postulates an alternative theory. It suggests that the truncal ataxia of posterior fossa tumours is largely the consequence of acute or subacute hydrocephalus—dilatation of the ventricular system.

The evidence for this alternative theory may be simply summarized as follows: cases of intracranial tumour where truncal ataxia occurs will be found to have dilatation of the lateral ventricles, whether or not the tumour is situated in the cerebellar vermis. Operations which relieve the hydrocephalus will relieve the gait disturbance, even if the tumour is left undisturbed or if its removal causes greater local damage to the cerebellar vermis than that caused by the tumour before operation. It is suggested that the reason why vermis tumours have for so long been believed to cause a gait disturbance by a local effect is that the majority of tumours which give rise to an early hydrocephalus unaccompanied

by focal signs are, in fact, situated in the midline of the cerebellum—the most striking being the medulloblastomas of childhood.

Truncal ataxia may be defined as a disturbance of gait or balance which cannot be attributed to motor weakness, sensory loss, incoordination of the limbs tested individually, or to a lesion of the vestibular apparatus. It is sometimes referred to as 'dysequilibrium', the presumption in that case apparently being that it is due to a defect of the central mechanisms concerned with balancing, as opposed to one of the central coordinating system for truncal muscles, if such a distinction can be made.

When the possibility that the mechanism of truncal ataxia might be hydrocephalus occurred to the author, a search was made for clinical material to confirm or refute the theory. Over the next two years, 19 consecutive cases of truncal ataxia associated with intracranial tumours were personally observed, 17 at the Guy's-Maudsley Neurosurgical Unit and two at St Bartholomew's Hospital. Brief summaries of these 19 cases are given and their relevant features analysed before the existing literature on the gait disturbances of cerebellar lesions is discussed in relation to them (Table).

## ANALYSIS OF CASES

All the cases summarized had a disturbance of gait, varying in severity from a mild unsteadiness to a complete inability to sit or stand unaided.

TABLE  
CLINICAL DETAILS OF 19 PATIENTS

Case	Sex, age (a), hosp. no.	Symptoms	Signs	Radiological investigations	Treatment	Sequel
1	M 65 GM 9633	2 m—headaches 1 m—unsteady gait, confusion, weight loss	Disorientated. Papilloedema. Nystagmus on lateral and upward gaze. Mild cerebellar type ataxia of R limbs. Unable to sit up or stand	Ventriculography: hydrocephalus and mass in R cerebellar hemisphere	Metastasis removed from R cerebellar hemisphere	20 d postop.: no longer confused and walking unaided, though ataxia of R limbs unchanged
2	F 47 GM 9584	1 yr—worsening gait, unsteadiness. Vertigo produced by sudden head movements. 1 m—headaches	Mental state normal. No papilloedema. Nystagmus on lateral gaze. Gait very unsteady, broad-based	Angiography: symmetrical hydrocephalus; haemangioblastoma in superior vermis	Total removal of cystic haemangioblastoma deep in superior vermis	Immediate relief of headaches and vertigo; 2 w post-op. gait appeared normal and could walk heel to toe with minimal unsteadiness
3	M 56 GM 9568	8 m—worsening gait. 1 w—confusion	Disorientated. Papilloedema. Nystagmus on lateral gaze. Mild cerebellar ataxia of L limbs. Gait broad-based, falls backwards	Angiography: dilatation of lateral ventricles. Haemangioblastoma of L cerebellar hemisphere	Total removal of solid haemangioblastoma of L cerebellar hemisphere	2 w later confusion gone, gait greatly improved. Ataxia of L limbs unchanged
4	M 4 GM 9500	3 m—headache, vomiting	Gait unsteady, broad-based. Papilloedema	Angiography: dilatation of lateral ventricles; avascular mass in inferior vermis	Subtotal removal of astrocytoma of inferior vermis	10 d postop.: headaches gone, papilloedema resolving, gait improved
5	M 63 GM 9110	3 w—headaches, falling around. 5 d—confusion and apathy	Lethargic, confused. Papilloedema. Nystagmus on lateral gaze. No limb ataxia or weakness. Unable to stand, falls backwards	Carotid angiography: symmetrical hydrocephalus. Ventriculogram: mass in vermis	Subtotal removal of metastasis in upper midline cerebellum extending into L cerebellar hemisphere	3 d later well-orientated, walking unaided though now had moderate ataxia of L limbs
6	F 58 GM 9408	9 m—headache. Gait unsteadiness. Loss of memory	Well-orientated but distractable. Mild ataxia left limbs. Completely unable to stand unaided	Angiography: symmetrical hydrocephalus and avascular tumour behind L petrous bone	Subtotal removal of solid haemangioblastoma of L cerebellar hemisphere extending into vermis	7 d later mental state normal, no headache and walking unaided, though ataxia of L limbs was worse
7	M 25 GM 8798	2 m—headache, vomiting. 2 w—gait deteriorating	Papilloedema. Bilateral sixth nerve palsies. Mild cerebellar ataxia of right limbs. Gait very unsteady	Carotid angiogram: symmetrical hydrocephalus. Ventriculography: mass in R cerebellar hemisphere	?Total removal of medulloblastoma in outer part of R cerebellar hemisphere	8 d later walking heel to toe with minimal clumsiness. R limbs still slightly ataxic
8	F 7 GM 8972	6 w—headache, vomiting, malaise	Papilloedema. Unsteady on heel to toe walking	Ventriculography: hydrocephalus and mass on R of posterior fossa	Subtotal removal of astrocytoma of inferior vermis and R cerebellar hemisphere	No signs 2 m post-op.
9	M 61 GM 9142	2 m—headaches. Clumsy right hand. Worsening gait	Confused. Papilloedema. Mild limb ataxia L > R. Unable to sit or stand unaided	Carotid angiography: hydrocephalus. Ventriculography: mass in inferior vermis	Partial removal of vermis glioma extending into both cerebellar hemispheres	At 14 d well orientated and gait virtually normal 1 yr later had relapse of gait ataxia and confusion. Both only partially relieved by a ventriculoatrial shunt
10	F 62 GM 9453	6 m—worsening gait. Mental deterioration	Disinhibited, confused. Nystagmus on lateral and vertical gaze. Reduced R corneal reflex. Unable to stand unaided	Angiography: hydrocephalus and meningioma of R petrous apex	Ventriculoatrial shunt	8 d later well-orientated and walking with assistant

TABLE—continued

Case	Sex, age (yr), hosp. no.	Symptoms	Signs	Radiological investigations	Treatment	Sequel
11	F 68 GM 9352	9 m—worsening gait. Intellectual deterioration	Demented. Limitation of conjugate upward gaze. Symmetrically brisk tendon reflexes. Slight ataxia L arm. Unable to stand unaided	Pneumoencephalography: hydrocephalus and pineal mass	Ventriculoatrial shunt (no radiotherapy)	5 m later: gait and mental state normal
12	M 25 GM 9376	7 m—headache. Worsening gait	Alert, well-orientated. Bilateral papilloedema. Minimal ataxia L limbs. Gait unsteady, broad based	Angiography: hydrocephalus, and mass in superior vermis	Preliminary ventricular drain. Removal of tuberculoma of superior vermis	Gait returned virtually to normal. L-sided ataxia worse. Gait improvement maintained
13	M 55 GM 8658	9 m—headache, vomiting, drowsiness. 6 m—worsening gait	Intellect normal. Papilloedema. Gross gait unsteadiness. Slight spasticity and weakness left limbs	Ventriculography: hydrocephalus caused by anterior third ventricle (?thalamic) mass	Ventriculoatrial shunt	Gait returned to virtually normal. Spastic L hemiparesis unaltered. Relapse of headaches and gait unsteadiness 6 m later relieved by revision of shunt
14	F 68 GM 9225	1 yr—headaches. 6 m—changed personality. Worsening gait	Confused. Papilloedema. Impaired upward gaze. Slight ataxia R arm. Gross gait unsteadiness	Angiography: hydrocephalus, and avascular mass high in midline of posterior fossa	Partial removal of lymphoma on underside of tentorium	10 d later: well orientated, no headaches, gait virtually normal
15	F 61 GM 8997	2 yr—headache, apathy. Worsening gait	Confused. Weakness of conjugate upward gaze. Mild ataxia L limbs. Unable to stand unaided	Carotid angiography: symmetrical hydrocephalus. Ventriculography: indentation of posterior third ventricle	Fenestration of arachnoid cyst just below apex of tentorium	12 d later: intellectual state normal, walking unaided
16	F 57 GM 9975	2 m—headache. Intellectual and gait deterioration	Confused. Papilloedema. Ataxia R arm. Tendon reflexes symmetrically brisk. Unable to sit or stand unaided	Angiography: symmetrical hydrocephalus and mass in R cerebellar hemisphere	Ventricular drainage. Removal of metastasis from R cerebellar hemisphere	3 d later still confused but could walk with assistance. Little further improvement
17	M 58 GM 8816	1 m—headache, gait unsteadiness	Well-orientated. Unable to stand unaided	Ventriculography: symmetrical hydrocephalus, mass in L cerebellar hemisphere	Removal of metastasis from L cerebellar hemisphere	Died 2 d later
18	F 34 SBH 531854	8 m—headache and gait unsteadiness	Papilloedema. Unsteady heel to toe walking	Carotid angiography: symmetrical hydrocephalus. Ventriculography: pineal mass	Ventriculoatrial shunt	Headaches and gait unsteadiness relieved before radiotherapy
19	M 25 SBH 514369	9 m—vomiting. 4 m—gait unsteady. 2 m—headache. Diplopia	Papilloedema. Nystagmus on lateral and upward gaze. Heel to toe gait unsteady	Angiography: hydrocephalus; avascular mass in vermis	Partial removal of medulloblastoma filling fourth ventricle, to allow free flow of CSF	Virtually no abnormality of gait 14 d post-op. (before commencement of radiotherapy)

The quality of the gait disturbance was similar in all, consisting of an appearance of a greater or lesser degree of instability in the upright position. Ten patients had some lateralized limb ataxia of cerebellar type, and two showed pyramidal tract signs, but these did not appear to be of sufficient magnitude to account for the abnormality of gait and balance in the individual cases concerned.

The responsible lesions were situated as follows: midline in the posterior fossa (11), laterally in the posterior fossa (three), pineal region (three), clivus (one), thalamus (one). Their histological nature was varied and of the 17 cases where it was possible to say with certainty, 14 were intrinsic and three extrinsic tumours.

In 18 cases there was radiological evidence of

ventricular dilatation and in the remaining case, a tumour of one cerebellar hemisphere, there was clear evidence of raised intracranial pressure, and it seems reasonable to infer that hydrocephalus was present. In 15 cases the tumour was so situated that it either involved the vermis directly or it was possible that it might do so from indirect distortion. Seventeen cases showed clinical evidence of hydrocephalus—for example, papilloedema or intellectual dulling—other than the gait disturbance.

Six patients had their hydrocephalus relieved by temporary or permanent ventricular drainage before direct interference with the causative lesion. In all these the former led to improvement in the abnormality of gait: complete resolution in two (cases 11 and 18), moderate relief in two (cases 12 and 13), and partial relief in two (cases 10 and 16). Complete or partial removal of the tumour relieved the hydrocephalus in 13 cases and in seven of these the operation involved, of necessity, considerable damage to the midline structures of the cerebellum. Yet of these seven cases, the disorder of gait recovered totally or nearly so in six and in the remaining case there was partial recovery. In six cases removal of the tumour was apparently achieved without significant trauma to the cerebellar vermis. The gait disturbance recovered totally or almost so in four of these. One case died too soon post-operatively to allow this function to be assessed and in one patient (case 16), a preliminary ventricular drain had already produced a partial recovery of gait which the removal of the tumour, a metastasis in the cerebellar hemisphere, did nothing further to improve.

#### DISCUSSION

It is widely taught that a disturbance of gait in the absence of other significant focal neurological signs will localize an intracranial tumour to the cerebellar vermis, and that the vermis contains structures concerned with maintaining balance and coordinating truncal muscles. Authoritative support for this belief may be found in the literature—for example, Holmes (1922), Weisenburg (1927), and Dow (1969).

It is largely based on observations made before the second world war, for as Brodal (1969) has aptly remarked, 'in recent years relatively few

authors have been interested in the subject' (of cerebellar symptomatology). Gordon Holmes's (1917) study of patients with posterior fossa missile wounds is probably the most comprehensive clinicoanatomical study of this subject. He came to the conclusion that 'activities which require the cooperation of homologous bilateral muscles are represented in or near the vermis'. Bailey and Cushing (1925) in their study of childhood cerebellar medulloblastomas attributed the gait ataxia which may be an early symptom of these tumours to a postulated origin in the flocculonodular lobe, the main cerebellar receiving area for vestibular fibres. This was supported by Raaf and Kernohan (1944) who found resemblances between medulloblastoma cells and certain embryonal cell rests in that part of the cerebellum.

Animal studies involving resections of the caudal vermis were found to cause transient gait disturbances. Dow (1938) resected the nodulus and uvula in monkeys, finding that the gait remained unaffected if a preceding labyrinthectomy had been performed. Carrea and Mettler (1947) resected the flocculus alone in monkeys. This produced a less marked and briefer gait unsteadiness. The same authors produced a severe gait disturbance, together with gross ataxia of all limbs, by resecting the vermis of the anterior lobe and lobulus simplex. These findings may relate to the somatotopical representation of the termination of the spinocerebellar pathways demonstrated by Adrian (1943) in this part of the cerebellum. The hindlimbs are represented most rostrally in this area and it is of interest that alcoholic cerebellar degeneration, which is characterized by the early development of an ataxic gait, is largely limited to the anterior vermis (Victor *et al.*, 1959). In this condition, unlike the gait ataxia attributed to flocculonodular lobe lesions, the disturbance of gait appears attributable to incoordination of the legs tested individually, correlating with the fact that the degeneration begins in the most rostral part of the anterior lobe vermis.

Thus the evidence is that vermis lesions at two sites can produce gait disturbances. Lesions of the caudal vermis (vestibulocerebellum) may lead to dysequilibrium unaccompanied by limb ataxia, while anterior lobe lesions disturb gait due to ataxia of the individual legs, a type of

gait disturbance which does not concern us here. Little is known of the functions of the more central vermis lying between these two extremes (Brodal, 1969), though it is known that visual and auditory stimuli may evoke action potentials there (Snider and Stowell, 1944). Despite the assertions of Bolk (1906) and Weisenburg (1927) it has not been shown that truncal and limb girdle muscles have a special representation in the vermis.

It is not the intention here to dispute that ablations of the caudal vermis disturb gait or that the vestibulocerebellum is concerned with balance, but it is asserted that the clinical gait symptoms observed to accompany posterior fossa tumours are to a large extent (though maybe not completely) the consequence of acute hydrocephalus.

The clinical evidence for relating vermis tumour gait ataxia to vestibulocerebellar damage is based mainly on medulloblastomas. Yet by the time of operation, let alone at necropsy, the tumour is generally involving such an extensive area of the cerebellum and the fourth ventricle that it is not possible to identify it as having arisen in the flocculonodular lobe. The author examined the records of all childhood medulloblastomas treated by a single surgeon (Mr J. J. Maccabe) at the Guy's-Maudsley Neurosurgical Unit. In all cases there was both preoperative gait ataxia and radiological evidence of hydrocephalus. Yet the gait recovered rapidly after operation, which relieved the hydrocephalus by unblocking the fourth ventricle while, if anything, inflicting further damage to the vermis. The onset of a gait disturbance coincided with or succeeded other undoubted symptoms of hydrocephalus, such as headache or lethargy, yet if it were due to flocculonodular lobe damage it might be expected that it would have appeared at an earlier stage, before the fourth ventricle had become blocked.

The evidence that, in man, traumatic lesions of the vermis give rise to a gait ataxia is not convincing. Although Holmes (1917) believed that the vermis is concerned with control of truncal muscles, he was forced to admit that he had never seen a wound confined to the vermis, and that where a wound involved the vermis in addition to a lateral lobe(s) there appeared to be no additional loss of control of gait other than that

attributable to incoordination of individual limbs.

Weisenburg (1927) stated revealingly that, though in his experience mistakes in localizing a lesion to the cerebellum have seldom occurred, yet where they have, it has usually been where a hydrocephalus has been caused by, say, a tumour in the third ventricle. An unsteady gait may be a symptom of adult normotensive communicating hydrocephalus (Ojemann, 1972) and even the dominating feature (Chawla and Woodward, 1972). Colloid cysts of the third ventricle may produce a severe gait disturbance which recovers when the cyst is removed and the hydrocephalus is relieved (Little and MacCarty, 1974).

It may well be asked why such an evident feature as gait ataxia has not previously been recognized as one of the symptoms of ventricular dilatation. There are probably several reasons. There is no doubt that hydrocephalus may be unaccompanied by any disorder of gait, especially if its onset is gradual, and early in life. Thus cases of congenital hydrocephalus are seldom characterized by an abnormal gait, though sometimes the latter is prominent. Also it has seemed to the writer that where gait ataxia is present in children with posterior fossa tumours, it is less striking and more easily overlooked than in comparable adult cases.

Until the wide recognition of adult-onset communicating hydrocephalus in the last decade, most cases of hydrocephalus of relatively acute onset recognized in adult life were due to tumours. Many of these were so situated that they produced in addition to the gait ataxia lateralized symptoms such as some spastic weakness or limb cerebellar ataxia. These features were probably often insufficient to account for the gait disturbance, but this was possibly overlooked and a false attribution of the difficulty in walking made to relatively minor clinical abnormalities. Those patients with tumours so situated in the vermis region as to produce hydrocephalus but no focal features had their unstable gait attributed to vermal damage, as that symptom was well known to be pathognomonic of a lesion in that part of the brain!

However, in many cases it seems likely that there is a local component contributing to the gait ataxia, for this symptom is often only par-

tially relieved by relief of the hydrocephalus. In case 9 the original partial resection of a glioma of the caudal vermis led to a virtually complete recovery of gait, but when the tumour recurred a year later with a relapse of the gait ataxia, a ventriculoatrial shunt led to only a partial remission. Presumably the gait disturbance now owed more to local brain damage than to hydrocephalus, whereas initially the reverse may have been the case.

It might be objected that recovery of gait after removal of a vermis tumour is merely a reflection of the known capacity of the cerebellum to recover from destructive lesions but this does not account either for improvement after shunting alone, or for the rapidity of gait recovery postoperatively in comparison with the length of time preoperatively that the symptom was present, as in case 2.

Theoretically, hydrocephalus might produce a disturbance of gait from downwards pressure on the rostral vermis by a dilated suprapineal recess, but this does not account for the gait ataxia of third ventricle tumours. Such a situation might be expected to give rise to signs of compression of the quadrigeminal plate which were not noted in these cases. A further objection is that a rostral vermis compression might be expected to give rise to a gait disturbance due to incoordination of the legs tested individually and so by definition be excluded from the category of gait ataxia being discussed here.

There seems to be no special anatomical reason why ventricular dilatation should disturb gait. Possibly it is due not to damage to any particular site in the cerebral hemispheres, but to a diffuse dysfunction of the white matter and hence of the general coordinating structures of the brain at their highest level.

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