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Gait apraxia in communicating hydrocephalus

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SUMMARY Apraxia of gait in patients with communicating hydrocephalus appears in the context of a generalised motor disorder that includes defective righting reflexes, generalised increased tone to passive movements, grasp reflexes, difficulty with serial movements of the hands and defective smooth pursuit eye movements. The inability to walk does not appear to be due to a motor disorder but to release of proprioceptive supporting reactions. This mechanism is triggered by proprioceptive stimuli.

Apraxia of gait in the most severe form can be defined as an inability to walk in the absence of weakness, sensory deficit or incoordination. A detailed historical account may be found elsewhere.[2] It has been properly classified as a form of limb-kinetic apraxia[1] because the basic defect is a perseveration of posture and an inability to perform the serial movements necessary for ambulation. In the initial stage there is difficulty in starting to walk or changing the direction of walking. A defect in equilibrium is usually present but may be absent. As Meyer and Barron[2] correctly pointed out: gait apraxia is a disorder specifically of human beings and it is unlikely that an experimental animal model will ever be found. In limb kinetic apraxias the basic disturbance is the motor perseveration that hampers the performance of any movement. There is an inability to shift from one movement to another although the "motor task" remains always clear. The nature of the perseveration is not clearly understood. Perseveration may be seen as a positive or negative phenomenon in the Jacksonian sense; that is to say, as a deficit in the programming of the action or as a result of an interference of the movement by an extraneous source. Perseveration can also be analysed in the context of the Sherringtonian concept of the adequacy of the stimulus. It then becomes a symptom that is stimulus-bound, either by loss of specificity in response (dedifferentiation) or because of the lack of the naturally opposing force. Denny-Brown[1] sees motor perseveration as a positive phenomenon (release) due to overaction of tactile stimuli in the presence of an intact parietal cortex (transcortical release). Posture and movement in this context can not be clearly differentiated. They depend heavily on all kinds of sensory input. Frequently a movement that can not be activated by one modality of sensory input can be activated by another. With this line of thought the identification of the stimulus that is adequate to elicit the stereotyped behaviour is most important.

A systematic study of the motor deficits found in patients with apraxia of gait due to communicating hydrocephalus has been undertaken in order to assess several problems. (a) Is the motor disorder confined to the lower extremities or is it part of a generalised disorder? (b) Is the perseveration of posture due to an inability to programme the necessary movements or is it secondary to an interference with another motor mechanism at a lower level? (c) What stimuli are most relevant in eliciting the perseveration of posture? (d) An assessment of other neurological deficits associated with this problem. It should be stressed at the outset that apraxia of gait is not a form of ataxia but a defect at a higher level of neural organisation.

Patients and methods

Six patients with apraxia of gait were selected solely on that basis; the six patients had a clinical diagnosis of low pressure hydrocephalus. Four had a severe form and two had a mild to moderate disorder. The clinical and ancillary findings of these patients are shown in table 1. The following features were systematically studied:

(1) General changes in movements. Ability to right from the bed or to turn over.

(2) Movements of the lower extremities when the patient was lying down, sitting or standing, (a) lifting the legs, (b) heel to knee.

(3) Influence of propriceptive stimuli upon the muscle

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Table 1 *Clinical findings in six patients with communicating hydrocephalus*

Patient	*Duration of illness*	*Computed tomography*	*Radioisotopic cysternography*	*Past medical history*
1	6 months	hydrocephalus	ventricular uptake >24 h	not relevant
2	8 months	hydrocephalus	ventricular uptake >24 h	subarachnoid bleed
3	3 months	hydrocephalus	ventricular uptake >24 h	not relevant
4	7 months	hydrocephalus	ventricular uptake >24 h	not relevant
5	10 months	hydrocephalus	ventricular uptake >24 h	subarachnoid bleed
6	1 year	hydrocephalus	ventricular uptake >24 h	not relevant

Patient	*Age (yr)*	*Urinary incontinence*	*Intellectual deterioration*	*Gait apraxia*
1	61	yes	yes	severe
2	67	yes	yes	severe
3	52	yes	yes	severe
4	59	yes	no	mild to moderate
5	64	yes	no	severe
6	57	yes	yes	mild to moderate

tone of the lower extremities sitting or lying (sudden dorsiflexion of the foot).
(4) Influence of visual righting reflexes in gait (stripes were placed in the path of the patient parallel and perpendicular to the direction of walking).
(5) Influence of verbal prompting in movements.
(6) Changes in the upper extremities, (a) grasp reflexes, (b) passive movements of the extremities, (c) serial movements (fist, edge, palm)[3], (d) independent finger movements, (e) handwriting, (f) pursuit eye movements were analysed tracking a pendulum. Visual suppression of vestibular responses was investigated rotating the patient in a torsionswing fixing on a point moving along with the chair.

Results

The patient with gait apraxia frequently showed general changes in his movements that were readily recognised. The results of the test are described in tables 2 and 3. When the patient was lying on the bed, he moved little or not at all; sometimes he grasped the clothing. There was a striking inability to rise from bed. The patient was completely unable to sit up on his own. When he attempted to move, he might raise his head or move his body to one side and then declare that he could not move. He might manage to sit up but then failed to swing his legs over the edge of the bed, and he could not use them for any other purpose. If he managed to sit up, either with help of by himself, he showed no attitudinal defects, keeping his body and his trunk in a normal position, but his equilibrium was precarious and he frequently swayed to either side and might actually fall. In the most severe cases of gait apraxia the patient was unable to stand up and readily fell unless the physician prevented him from so doing.

If the patient was supine, it could be easily shown that he had no weakness or incoordination of the lower extremities. He could readily lift either leg and keep it in the air for several minutes. Heel knee tests were performed with minimal clumsiness. He would readily make a circle in the air, and make the movements necessary for riding a bicycle. He could kick an imaginary ball without any difficulty. Tactile stimulation of the sole of the feet elicited no response but dorsiflexion of the feet frequently caused stiffening of the lower extremities (table 2).

If the patient was in the sitting position, and the lower extremities were not supporting the body weight, it could be demonstrated that he was able to perform many movements without much trouble. He frequently could make tapping movements with either foot, make a circle on the ground or the figure of eight, or kick an imaginary ball. All these movements were no longer possible when the

Table 2 *Results of tests*
Movements of the lower extremities

Patient	*Lying*	*Sitting*	*Standing*
1	normal	normal	unable
2	normal	normal	unable
3	normal	normal	unable
4	normal	normal	unable
5	normal	normal	unable
6	normal	normal	unable

Patient	*Sudden dorsi-flexion of foot*	*Walking over stripes*	*Ability to right or turn over on bed*
1	Stiffening of the leg	unable	unable
2	Stiffening of the leg	unable	unable
3	Stiffening of the leg	unable	unable
4	Stiffening of the leg	poor	poor
5	Stiffening of the leg	unable	unable
6	Stiffening of the leg	poor	poor

*Lifting the legs, heel to knee, kicking a ball, making a circle in the air, tapping movements, riding a bicycle.

Table 3 *Results of tests*

Patient	*Influence of verbal prompting in movements*		*Grasp reflex*	*Serial movements of the hands*	*Independent fine finger movements*	*Handwriting*
	Standing	*Sitting*				
1	no	yes	no	poor	poor	poor
2	no	yes	yes	poor	poor	poor
3	no	yes	yes	poor	poor	poor
4	no	yes	yes	poor	poor	poor
5	no	yes	yes	poor	poor	poor
6	no	yes	no	poor	poor	poor

patient stood up. He then appeared hopelessly glued to the floor, unable to take a step. In attempting to walk he might shuffle with short steps. His equilibrium was poor and he might fall while attempting to move. If the patient was supported by a person and he put his weight on one extremity relieving the other one, he could then perform the same activities with the non supporting leg as if he were lying down or sitting. Verbal prompting did not induce change in the behaviour of the patient. Stripes placed on his path either perpendicular or parallel to the direction of walking did not improve his performance. Both lower extremities could be felt to have a greatly increased tone (table 2).

More surprising was finding a variety of difficulties with motor performance in the upper extemities. A simple grasp reflex was induced by tactile stimuli of the palm of the hand almost invariably (table 3). Occasionally visual input would precipitate grasping responses (visual grasping). Proprioceptive stimuli most frequently elicited a grasping response. Increase tone of the "counterholding" type was almost always found in these patients. It too was also induced by both tactile and proprioceptive stimuli although the proprioceptive stimulus appeared to be a more powerful one. Hence any attempt to move any extremity resulted in contraction of the entire extremity. Motor perseveration frequently hindered any activity in these patients. Motor perseveration could easily be demonstrated by asking the patient to perform a series of movements that might or might not have a goal. Closing and opening the hands could be performed, although the movement was slow and the patient frequently failed to open his hands completely. With Luria's test[3] for serial movements (fist, edge, palm) the patient invariably perseverated with some movements. He might do well initially, but then he continued to make a fist or to strike on the edge of the hand. Characteristically here, verbal input (repeating the words fist, edge, palm) would improve his performance. However such patients did not have an impairment of visuo-spatial orientation and they might copy different positions of the hand of the examiner without any trouble. When both hands were to be used in an alternating fashion, for example making a fist with one hand while the other was open and then reversing this position, the patient was quite unable to perform the task; in this case verbal help was of no avail. Fine movements of both hands could be performed although they were performed slowly. None of these patients showed evidence of ideomotor apraxia, being able to perform complex commands without hesitation. Motor perseveration would sometimes interfere with the movement but the final result was altered little. Handwriting was altered in a very specific way. The hand would stiffen when attempting to grab the pencil. If the patient was asked to write his name he would perseverate writing it and was unable to write anything else, unless given a new sheet of paper. If the patient was instructed to draw a circle and a cross, he perseverated in drawing the circle or the cross and was unable to shift from one motor task to another (table 3).

Associated neurological findings In all these cases there was no incoordination of the cerebellar type. Interestingly four of the six patients did not have pyramidal signs, and Babinski signs were absent in five. There was no deficit of cutaneous or proprioceptive modalities. No visual impairment was present and visuo-spatial disorientation was found only in one case. A non-fluent aphasia frequently mild with verbal perseveration was observed in two cases. Blepharospasm was seen in one patient. Snout and sucking reflexes sometimes were present. The jaw jerk was normal in five of the patients. Other infrequent neurological findings were, mild hemiparesis, avoiding responses, urinary incontinence but no clear pseudobulbar symptoms. The dementias found in these patients were frequently of a global type with impairement of orientation, memory and judgement but without focal cortical signs. There was no evidence of ideomotor apraxia or ideational apraxia. Agnostic difficulties were not present. Smooth pursuit eye movements were saccadic in all of the patients, and all of them failed to suppress the vestibulo-ocular reflex when rotated fixing on a target moving along with the patient.

Discussion

Denny-Brown[4] has shown that the fundamental organisation of the motor systems in mammals is that of quadrupedal progression. The rhythmic movements required to move the weight of the body from one limb to another are chiefly integrated in the spinal cord. The mechanisms involved were superbly analysed in the dog by Sherrington[5] and they include reciprocal innervation, successive induction, rebound, switching phenomenon and afterdischarge. Supraspinal influences ensure the coupling of locomotion with posture and righting. Intact posterior roots are not essential for locomotion and it has long been believed that the movements of locomotion are of "central" origin.[7] It may appear that the same mechanisms operate in bipedal locomotion in human beings. Ontogenetically bipedal locomotion in human beings is usually acquired between 10 and 14 months of age.[6]

In physiological terms, gait apraxia most likely represents a defect in the supraspinal modulation of locomotion, for in the human the elemental mechanisms of stepping are influenced greatly by supraspinal structures. However, this hypothesis is not altogether satisfactory, because it does not explain why more complex patterns of movements may be preserved while a more elemental mechanism is lost. The fundamental question is whether the disorder represents a problem in the central programming of the movement or whether there are mechanisms that interfere with the normal systems. Apraxia of gait is a disorder of the serial order of motor behaviour. Seen in this context, the basic problem appears to be the perseveration of posture. The nature of perseveration becomes then the fundamental problem. It is clear that apraxia of gait is part of a more diffuse defect of motor function. In the most severe forms the righting reflexes are abnormal and equilibrium poor. These findings are difficult to understand because righting and standing are automatic movements integrated at relatively low subcortical levels. What is the nature of the defect in the lower extremities? We found a great difference in the performance of the patient if he was supine, sitting or standing. If the legs were not used to support the body weight, they could be used quite easily for many types of movements. Serial movements such as tapping or riding a bicycle could be easily performed. Those involving an imaginary object such as kicking a ball were also done without difficulty. Movements with a goal, such as those made attempting to draw a circle or an eight with the lower extremities were carried out even if the sole of the feet was in contact with the ground However, when the patient attempts to put any weight on the extremities he would immediately become "locked." No effective stimulus could make the patient move in these circumstances. Dorsiflexion of the foot would elicit a general stiffening of the limb. Several important conclusions can be drawn from these facts. First of all, the movements necessary for bipedal locomotion are not lost because the patient can perform them as long as the lower extremities are not used as an antigravity pillar. Tactile or contactual stimuli on the sole of the feet are of no importance in eliciting the perseveration of posture. Proprioceptive stimuli of the lower extremities are the effective stimulus that "locks" the lower limbs. So the disorder of gait in those cases is not directly due to a deficit or defect in the programming of the movements for locomotion, but it seems secondary to a mechanism that interferes with serial movements. That mechanism is triggered by proprioceptive stimuli. We think that apraxia of gait is due to release of positive proprioceptive supporting reactions. In addition these patients have grasp reflexes, poor righting reflexes, generalised gegenhalten, difficulty with serial movements of the hands, clumsiness performing independent finger movements, motor perseveration in writing, and defective smooth pursuit eye movements. The nature of these defects can not be explained on the same basis as the gait apraxia.

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