

PAPER

Gait apraxia after bilateral supplementary motor area lesion

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Objectives: The study aimed at addressing the issue of the precise nature of gait apraxia and the cerebral dysfunction responsible for it.

Methods: The case of a patient, affected by a bilateral infarction limited to a portion of the anterior cerebral artery territory is reported. The patient's ability to walk was formally assessed by means of a new standardised test.

Results: Due to an anomaly within the anterior cerebral artery system, the patient's lesion was centred on the supplementary motor regions of both hemispheres. He presented with clear signs of gait apraxia that could not be accounted for by paresis or other neurological deficits. No signs of any other form of apraxia were detected.

Conclusions: The clinical profile of the patient and the analysis of 49 cases from previous literature suggest that gait apraxia should be considered a clinical entity in its own right and lesions to the supplementary motor areas are responsible for it.

Gait disorders are a common feature of several neurological (for example, Parkinson's disease) and psychiatric diseases (for example, schizophrenia). Disturbances of gait that cannot be readily accounted for by abnormalities often occurring in old age (for example, bone and joint degeneration, muscle wasting, or arteriosclerosis obliterans of the lower limbs), pyramidal or sensorial deficits, ataxia (cerebellar, vestibular, or proprioceptive), dystonias or dyskinesias (due to diseases involving the basal ganglia), nor by psychosis, are often referred to as "gait apraxia" (GA).^{20–24}

Meyer and Barron²³ defined GA as "the loss of ability to properly use the lower limbs in the act of walking" (p 279). The syndrome is composed of a mixture of signs including disequilibrium, locomotor abnormalities, inappropriate postural responses, and loss of gait ignition. Gait, stance, and trunk movements are all complex and highly practised.²⁵ The respective contribution of each one of these components is often hard to disentangle from that of the others (*Rumpf, Stand und Gangapraxie*, trunk, stance, and gait apraxia^{6, 26}).

Indeed, Lange²⁷ maintained that in GA ideomotor apraxia of limbs is not relevant and that all segments involved intertwine to perform a given movement. Moreover, Nutt *et al*²⁰ maintained that the severity of each individual sign constituting gait apraxia varies from patient to patient.

The trunk component of GA encompasses disturbances such as deficits of rising from sitting, sitting down, lying down, turning around while standing, or rolling over in bed. Disturbances of stance encompass deficits of balance while either standing in an upright position, walking, or in negotiating turns. They also include the loss of anticipatory and reactive postural responses resulting in falls.^{22–28} Gait disorders comprise deficits of ignition (frozen foot) and stepping, including shuffling, obstructive leg crossing,^{22–29} or leg hypokinesia. Other signs characterising gait disruption include bizarre, counterproductive, and perseverative leg movements, such as the leg's *Witzelsucht*,²⁹ dystonias, dyskinesias, and lower limbs hypotonia as well as involuntary temporary arrests of walking.

Some authors interpreted GA as a feature of frontal inertia.³⁰ Others³¹ maintained that GA be a misnomer as the deficit should not be interpreted as apraxic.^{32–34}

Gait apraxia was seen over a century ago in patients with lesions to the precentral regions of the frontal lobes who were not paretic.^{14*} The association between GA and lesions to the frontal lobes has been reiterated ever since.^{2, 23, 25, 37} Moreover, a few authors^{38–42} demonstrated that the link between trunk movement disturbances and left parietal lesions is weak with respect to other apraxias (but see Poeck *et al*³⁵ for a different view).

However, the precise nature of GA and the cerebral dysfunction responsible for it have been matters of considerable debate.^{7, 28, 44, 45} The association between gait disturbances

*The syndrome of GA has been named differently by different authors—for example, "Torso apraxie" (trunk apraxia) or "déséquilibre frontale" (frontal imbalance²). Some of the other labels conformed to the core deficit found—for example, "trepidante abasie",^{3, 4} "eingentümliche Ungeschicklichkeit" (remarkable clumsiness⁵), "bewegungstechnische Schwierigkeit" (loss of the technical requirements of the movements⁶), "Tapsigkeit" (gawky movements⁷), "Unbeholfenheit" (awkwardness⁸), slipping clutch syndrome,⁹ lower half parkinsonism,^{10, 11} gait ignition failure.¹² Some other labels referred to the authors' interpretation of GA—for example, melokinetic apraxia,¹³ "frontale ataxie" (frontal ataxia¹⁴), "gliedkinetische apraxie" (limb-kinetic apraxia, quoted by Westphal,¹⁵ "motorische Apraxie",¹⁶ "corticale apraxie",¹⁷ "innervatorische Apraxie" (innervatory apraxia¹⁸), "bilaterale motorische Apraxie",^{18, 19} frontal gait disorder,²⁰ higher level gait disorder.²¹ However, these different terms, often derived from different conceptualisations of gait disorders, define syndromes that overlap more than is usually admitted.

*Bruns¹⁴ quoted the work of earlier authors who noted the presence of stance and gait disorders in patients with frontal tumours. Among them, he (p 138) cited Wernicke³⁵ who reckoned that a "hesitant gait has been often seen in patients with frontal tumours, it is well possible that in the future this sign will be considered as resulting from a direct insult to the frontal lobes" (p 323). Oppenheim³⁶ described gait disturbances in nine of 11 patients with frontal tumours compared with only four of 10 patients with tumours elsewhere in the brain. Bernhardt (quoted by Bruns¹⁴ p 138) reported gait disturbances in 18 of 44 patients (40%) with frontal tumours but only in five of 41 (5%) patients with extrafrontal tumours. This testifies that the link between gait disturbances and frontal lesions predates the later discussion between its apraxic or ataxic nature.

and cerebellar damage has been the only accepted knowledge for a long time. Neurosurgeons searched for tumours in the cerebellum solely on the basis of the ataxic appearance of GA,^{46, 47} only to discover postmortem that sometimes the tumour was sited within the frontal lobes instead.

Some early authors,^{16, 48, 49} following Bruns¹⁴ suggestion, maintained that the symptoms of GA could aptly be explained in terms of cerebellar imbalance. They interpreted it as due to either a frontopontocerebellar disconnection,^{50, 51} or to an increased pressure which benumbs the cerebellum.⁵² Frazier⁴⁹ maintained "that ataxia may be caused by a unilateral or a bilateral frontal lobe even in the absence of signs of increased intracranial pressure seems to admit of no contradiction" (p554). Hence the term "frontal ataxia" became popular. De Ajuriaguerra *et al*⁵³ conceded that GA may be a sign of frontal involvement, though denied that it was apraxic in nature.

On the other hand, Hartmann⁵⁴ and Kleist⁶ conceived that the GA movement disorders be a form of true apraxia, due to lesion of a specific frontal centre devoted to deploying attention to negotiate gait or to the planning of trunk and lower limb movements, respectively. Crigel and Arseni⁵⁵ studied 165 patients with either unilateral or bilateral frontal damage, 59 (36%) of whom presented with gait disorganisation. They argued that the walking disturbances due to frontal apraxia or cerebellar ataxia could be differentiated from one another (see also table 2 in Meyer and Barron²³). The concept of GA met with significant agreement^{1, 2, 19, 22, 28, 29, 56-62} and is currently listed among the direct signs of frontal lobe damage (Adams *et al*⁶³ pp 121-122; Kurlan *et al*⁶⁴).

The precise localisation of GA within the frontal lobes remains elusive. Reviews of the early literature showed that most cases of GA involved tumours encroaching on or compressing the medial aspects of the frontal cortex bilaterally.^{9, 23} However, the mapping of the symptoms onto the damage caused by a tumour is far from adequate.

Cases have also been reported of GA after vascular lesions, which should allow for a more precise localisation than allowed by tumours.^{6, 19, 23, 25, 28, 57, 65, 66} Taken together this literature confirms that lesions to the medial walls of the frontal lobes are very likely to be responsible for the appearance of GA. In particular, the lesions found in these patients lay within the territory of the anterior cerebral arteries and softened the first frontal gyrus or the gyrus cinguli, with or without involving the corpus callosum. However, the lesions in these patients were either too large or not analysed in sufficient detail to allow firm conclusions about the precise identification of the structure the lesion of which may be responsible for GA. Moreover, although the clinical syndrome had been recognised long before,³⁵ most reports of GA relied on a welter of signs never assembled in a formal diagnostic tool.

In this paper we report the case of a patient who was affected by a bilateral infarction of a portion of the anterior cerebral artery territory. The patient showed a clear GA, which was formally investigated with a new and standardised test (Della Sala *et al*, unpublished data). An unusual anomaly of the anterior cerebral artery system of the patient caused his stroke to affect only the first frontal gyri of both hemispheres. This allowed us to better delineate the boundaries of the lesion sufficient and necessary to give rise to GA.

CASE REPORT

The patient was a right handed (score 12/12 on the handedness Edinburgh inventory⁶⁷) retired man with 6 years of formal education. He was 64 years old when he was admitted to the third neurological ward of the University of Milan because of a bilateral stroke in the anterior cerebral arteries system.

Clinical picture

Two days after admission, He showed a spastic paraplegia with bilateral sign of pyramidal impairment, coupled with severe

hypophonia. Neurological examination disclosed also the presence of plantar grasp, of bilateral "tonic grasping",⁶⁸ instances of groping ("magnetic apraxia"⁶⁹) particularly with his right hand, and frontal release signs, such as bilateral pal-moment and snout reflexes.⁷⁰ He also presented with motor and verbal inertia.^{71, 72}

One month after the onset, the patient was able to walk with two crutches.

Five months later his muscle stretch reflexes were still hyperactive in the lower limbs, although only minimal strength impairment was noticeable at the neurological examination. He had considerable difficulty in stopping walking on command and his gait, tentative and uncertain, was hampered by hesitation; in Liepmann's⁷³ terminology, he seemed to have lost the "Virtuosität" of the normal gait.

Fourteen months after the patient's stroke, the neurological examination disclosed only a minimal strength deficit on the right side. His walking remained very clumsy. He could not imitate foot/leg movements performed by the examiner (for example, draw a circle with the leg extended while seated, or trace a cross on the floor with either foot).

Neuroradiology

A series of MRIs and a digital subtraction angiography disclosed a particular anomaly in the patient's anterior cerebral artery system, which supplies the medial surfaces of the frontal lobes. There are no right and left anterior cerebral arteries. His anterior vascular system is characterised by the presence of three main azygous-like trunks, one anterior, one middle, and one posterior, each one of which divides into a left and a right branch. From this vascular distribution derives the rare event that small symmetric areas of the medial surfaces of the right and left hemispheres are supplied by the same single artery. The patient had an occlusion of the middle trunk just before its partition, which gave rise to a stroke damaging symmetric portions of the medial walls of both of his frontal lobes. The lesion involved the superior frontal convolution (F1) of both hemispheres, sparing the polar regions, as well as the cingulate and the corpus callosum. In particular, it involved the supplementary motor areas (Brodmann's area 6) of both sides (figs 1, 2, and 3).

General neuropsychological assessment

The patient's neuropsychological assessment has been detailed elsewhere.⁷⁴ In brief, he was fully oriented and cooperative, was not aphasic, and did not show any impairment in visual perception. His scores on tests assessing general intelligence and different aspects of memory functions were all within the normal range. When first assessed, 2 months after onset, he performed poorly on some tests assessing executive functions, such as phonological fluency, reversal learning, or selective attention, which have all been shown to be sensitive to frontal lobe damage.⁷⁵ However, 14 months after onset his psychometric profile was entirely within the limits of normality. He also showed several instances of utilisation behaviour^{76, 77} and grasp reflex, which were still persistent at this time.

Apraxia assessment

Limitation of intransitive gestures

The test devised by De Renzi *et al*⁷⁸ was employed. This is a 24 item imitation test consisting of half meaningful and half meaningless movements, half requiring the patient to hold a posture, and half the execution of a motor sequence. The score for each item ranges from 3 to 1 depending on whether the performance is flawless on the first, second, or third attempt. A score of zero is given when the performance is unsatisfactory all three times. Therefore the total score ranges from 0 to 72. Individual scores are age and education adjusted. The cut off point (53) corresponds to the worst score found in a group of 187 controls⁷⁹ and represents the 95% outer tolerance limit of the normal population.

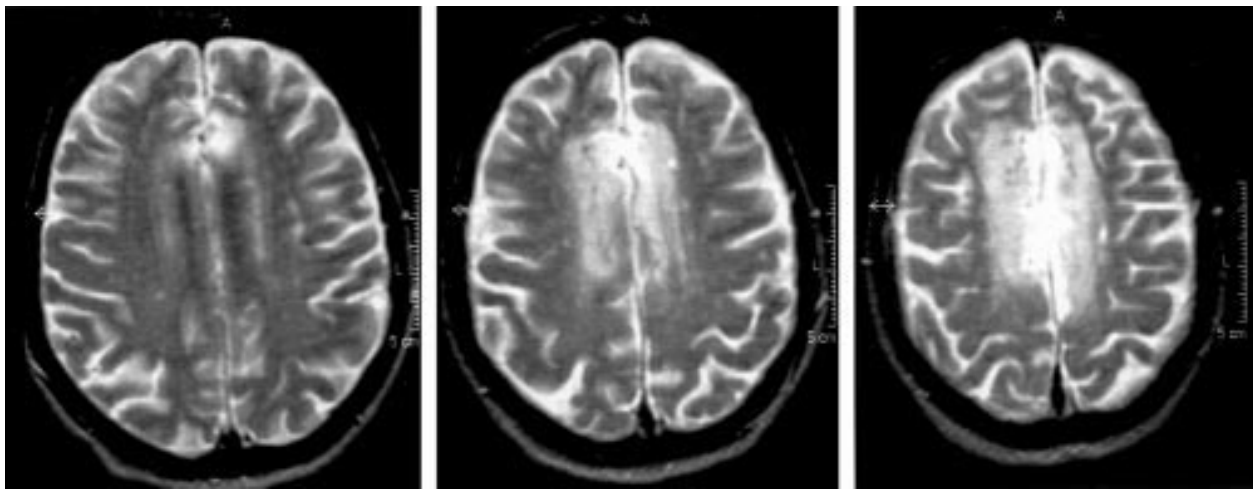


Figure 1 The patient's MRI T2 axial scans showing the lesion encroaching upon the superior frontal convolutions and centred on the SMA bilaterally.

Actual use of objects

The test described by De Renzi and Lucchelli⁶⁸ was used. Twenty objects of common use were presented one at a time to the patient. He was asked to demonstrate their use by carrying out a series of common, complex actions (for example a candle, a candlestick, and a matchbox were laid on the table and the patient was asked to light the candle). Scoring followed the published norms, score ranges from 0 to 120, cut off score is 60 (worst observed score in normal controls).

Face apraxia

Two tests assessing the imitation of the upper and the lower face movements respectively were used.⁸⁰⁻⁸¹ The upper face apraxia test included nine items (for example, "Close your eyes", "Look down"), and the lower face apraxia test was made up of 29 items (for example, "Show your teeth", "Blow"). Items were scored as pass or fail according to a set of rules derived from previous authors' attempts to define errors in facial apraxia.⁸²⁻⁸³ For both the upper and lower face tests, scoring procedures based on the rank order score were employed. The total score ranges from a worst of zero to a best score of 45.0 in the upper face apraxia test and to a best of 435.0 in the lower face apraxia test. Individual scores are age and education adjusted. Cut off scores, corresponding to the outer tolerance limit of the score distribution of 182 healthy volunteers⁸⁰⁻⁸¹ are 38 and 400 for upper and lower face tests respectively.

The patient performed within the normal range on all tests assessing ideomotor, object utilisation, and upper and lower face apraxia. His scores are given in table 1.

Gait apraxia

The patient presented with difficulties in sitting on or standing from a chair on command. He had difficulty lying supine on the examining bed and could not roll over. These difficulties were also overt in his daily life. Indeed, his wife confirmed that he needed her assistance to go to bed and to rise from it. His walking was hesitant and at times he persevered with individual components of a movement sequence. He was well aware of these difficulties and understood what he was requested to do, although he upheld that he no longer knew how to perform these movements. These problems were initially traced back to his motor inertia and central paraplegia. However, these difficulties continued after the rapid recovery from his motor deficits (probably due to perilesional oedema) was almost complete. Indeed, the improvement of motor functions made it clear that he also had problems in starting to walk, in stopping going forward, let alone in trying to walk backwards. These deficits were so severe as to be far beyond the possible sequelae of some elementary neurological deficits. He did not show any cerebellar or parkinsonian signs or symptoms. Pseudobulbar palsy was highly unlikely as he was not dysphagic nor dysarthric, showed no signs of diffuse cognitive deterioration, and the MRI disclosed no signs of subcortical lacunae.

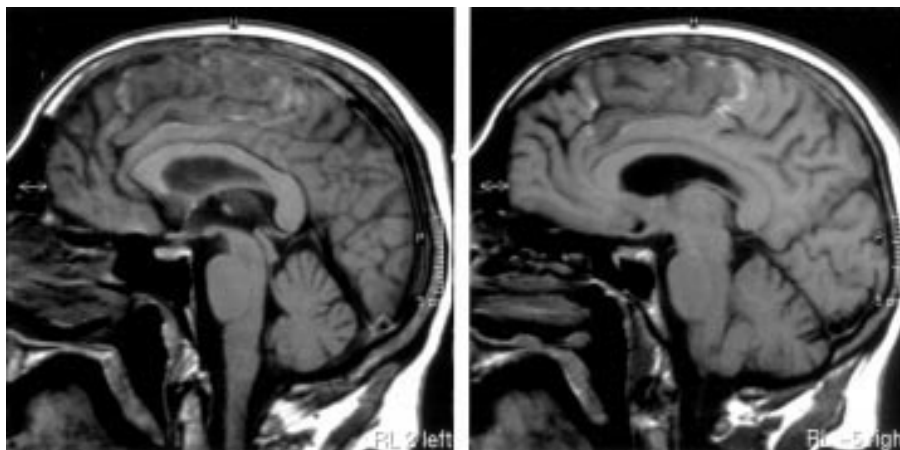


Figure 2 The patient's MRI T1 parasagittal scans. Left: parasagittal scan 2 mm off the midline. Right: parasagittal scan 5 mm off the midline.

Table 2 The patient's performance in the three components of the tests assessing GA 14 months after onset

Test	Patient score	Scale score range	Cut off score	Age matched controls (SD)
Trunk	11	0–22	20	22
Walking	10	0–20	17	19.9 (0.4)
Lower limbs	5	0–28	26	27.7 (0.6)
GA total score	26	0–70	65	69.5 (0.7)

Venneri, unpublished data). Most controls performed at ceiling in all the three components of the test. The score corresponding to the inner tolerance limit of the normal distribution was chosen as the cut off point in all tests.

The patient scored poorly on all three components of the task. For example, he failed to turn his head towards the left, or to bend his trunk forward, he proved unable to turn from prone to supine on the bed. He could not mime marching on the spot nor marching sidewise to the left or to the right. He failed all attempts when asked to pretend to extinguish a cigarette while seated, to kick an imaginary ball, or to wipe his feet on a mat.

His scores together with score ranges, cut off scores derived from norms, and the mean scores achieved by 26 controls aged between 60 and 69 in each component of the test, are reported in table 2.

DISCUSSION

Gait apraxia as a clinical entity

All purposeful movements involving trunk and legs, including walking, contribute to very complex performances. Not only do they need the cooperation and coordination of several muscular districts, but they also call for perfect timing and sequencing of events, which, once learnt, become a semiautomatic performance.

Several neurological diseases and psychiatric conditions can impair this complex, skilful performance. The impairment of locomotion not accountable for by any alternative cause, coupled with cerebral lesions encroaching upon areas known to be involved in its accomplishment, should be considered GA. Gait apraxia is defined as the loss of monitoring of the automatic implementation of gait mechanisms.

Gait apraxia has a long history. However, its precise nature and localisation are still defiant of exact identification. The case of the patient reported in this paper helps refine the syndrome, thanks to the standardised tests employed, and allows for a more accurate topographical definition of the responsible brain lesion, because of the unusual anomaly of his anterior cerebral arteries system.

The patient never showed any problems in negotiating difficult walks (for example, mountain routes) before his stroke. We interpreted his clinical picture as apraxic because of the lack of overt elementary motor, ataxic, or psychiatric deficits. He presented with disturbances in all the domains of GA acknowledged by Zingerle²⁸ and by Nutt *et al.*,²⁰ as the core symptoms of "frontal gait disorder"²⁰—that is, walking disturbances, disequilibrium, loss of postural reflexes, counterproductive parasitic movements. He also manifested ignition failure with his feet glued to the floor,¹² his walk was stiff and graceless and characterised by difficulty in stopping and turning. This paramount inability to halt one's own gait was described by Brun⁵⁸ (case H) as being similar to that of an automaton ("*gleich völlig einer lokomotorischen Maschine*", translation: he was akin to an automaton, p 215). Moreover, the patient failed to reproduce by imitation or on verbal command meaningful gestures (for example, a cross, a circle) with either his leg or foot, a sign seen by other authors in patients affected by GA.¹⁹

Anatomical remarks

Table 3 summarises the 49 cases we could glean from the literature reporting the localisation of GA. In 45 of these cases the localisation was precise enough to contribute to our discussion. Different authors used different labels to describe similar sets of GA signs. Moreover, they chose to emphasise different aspects of the syndrome, often according to their theoretical frame of reference. The localisation is reported as given by the authors*.

From the analysis of table 3 it is possible to deduce that GA is not a rare entity and can be due to different aetiologies. However, cases of "pure" GA are scant. With few exceptions (for example, case K³⁷) the lesion damaged the precentral aspects of the frontal lobes, with or without involvement of the genu of the corpus callosum. In 24 cases out of 45 in which the localisation was possible the lesion was bilateral. When the lesion was reported as unilateral, there seems to be some evidence for a left asymmetry (13 left cases versus eight right).

Indeed, Lange²⁷ stated that "certainly frontal lobe lesions play a key role in GA, yet it would be premature to maintain that left frontal lobe lesions are more frequently associated with GA than the right ones, and which region is more important"²⁷. Kleist²⁶ came to the same conclusion on the basis of his review of the literature and stated that the lesion responsible for GA is located "at the foot of F1, . . . ; the role of the corpus callosum is uncertain".

Scrutiny of the descriptions of the cases reported in table 3 (tumours and abscesses) discloses that the medial aspects of the frontal lobes are involved in most of them. Accordingly, Frazier⁴⁹ reported that 49% of 105 cases of patients with a frontal tumour upon whom he operated showed one or more "pseudocerebellar" signs of "frontal ataxia" (p 548). He also maintained that in most cases the lesions occupied the superior aspects of the frontal lobes, whereas none encroached upon the olfactory groove or the frontal poles. However, the growth of malignant tumours permits only flawed localisation.

Stroke and injury cases (table 3) should allow for a better relation between symptoms and site of lesion. Unfortunately, in several cases, the information available is too gross to attempt any fine grained localisation. Only three cases describe with some certainty a circumscribed lesion of the medial aspects of F1 (case K⁸; case JS⁵⁶; case HP¹⁹). In some further cases the corpus callosum was damaged, pointing to medial lesion (case T³⁷; case I⁶⁶). Moreover, von Monakow¹⁸ (pp 547–52) reported on the case of patient Koch affected by a post traumatic cyst invading the whole left frontal lobe but F1. This woman did not show GA, upholding the view that GA be associated with F1 lesions.

Gait apraxia has been mentioned also in three cases affected by Pick's disease (case Zorn^{8 27}; case AC²⁵), one affected by not-defined brain atrophy (case Gloor⁸), and two with neurosyphilis (case LS⁸⁴; case No 2²³). All these cases had a predominantly frontal atrophy, further emphasising the association between GA and frontal damage. Moreover, Rossor *et al.*²⁷ described GA in two demented patients reporting also PET data showing clear medial frontal hypometabolism in both patients.

Lastly, some indirect evidence supporting the frontomedial hypothesis of GA comes from the finding that gait abnormalities associated with normal pressure hydrocephalus have been

*The six patients with gait ignition failure reported by Atchison *et al.*¹² are not listed in table 3 because the authors explicitly maintain that the symptom they describe should be differentiated from GA. However, the authors acknowledged that gait ignition failure is due to frontal damage. We see no reasons to keep it separate from other manifestations of GA. **"Sicherlich spielt auch fuer sie das Stirnhirn eine wesentliche Rolle, ob das linke vorwiegend und in welchen besonderen Anteilen, läßt sich noch nicht näher feststellen".²⁷

Table 3 Localisation of lesions of patients presenting with signs compatible with the diagnosis of GA gleaned from the literature and subdivided according to aetiology

Author(s), year	Case	Age	Sex	Localisation
Traumas:				
Feuchtwanger 1923 ¹¹⁰	A.Li	32	M	Metal splinter in the L and R F
	MK	20	M	Bullet in the R F
Kleist 1934 ⁸	K	NR	M	Bullet through L and R areas 6+gCC
Tumours and abscesses:				
Bruns 1892 ¹⁴	I	NR	NR	"Sarcoma" L and R medial F
	II	54	M	Large subdural haematoma L F
	III	36	M	"Sarcoma" L F2.
	IV	60	M	Glioma R F2, F3
van Vleuten 1907 ⁹⁷	AL	55	M	"Sarcoma" L gCC+L F1+L cingulum
Hartmann 1907 ⁵⁴	WF	57	M	Glioma gCC+L and R+cingulum
Vincent 1911 ⁴⁶	Th	54	W	Glioma RF
Brun 1921 ⁵⁸	H	25	M	Glioma R medial F+CC
von Monakov 1914 ¹⁸	E	59	W	Glioma L medial F1+gCC+R cingulum
	W	58	W	Metastases medial L and R F+gCC
Dimitz and Schilder 1922 ⁵⁰	MW	38	W	Glioma L and R F1 F3 polar+gCC
Pözl 1924 ²⁹	HK	18	W	Abscess R F polar
			M	Glioma R F polar
Marie <i>et al</i> 1924 ¹¹¹	MH	56	M	Glioma R F1, F2
Gerstmann and Schilder 1926 ²²	FW	56	M	Glioma L medial F
Klein 1928 ⁴⁴	JG	52	M	Glioma R F+basal ganglia
Adie and Critchley 1927 ¹¹²	AL	54	M	Glioma R F1
van Bogaert and Martin 1929 ²	RB	47	W	Abscess L F1, F2
	MK	34	W	Glioma L F orbitopolar
Sittig 1929 ¹		39	F	Tumour around CC
Kleist 1934 ⁸	M	NR	NR	Glioma R F1 (area 6), L F1+CC
	S	NR	NR	Glioma L and R F1+gCC
Bell, 1934 ¹¹³	LD	53	W	Glioma R F+gCC
	CP	31	W	Granuloma anterior CC+hydrocephalus
Frazier 1936 ⁴⁹	5	4	M	Fibroblastoma of the falx L and R
Petrovici 1968 ²⁵	MA	28	M	Astrocitoma L F*
Strokes:				
Liepmann 1900 (quoted by Lange 1936 ²⁷)	T	48	M	L*
Abraham 1907 ⁵⁶ ; Brun 1921 ⁵⁸	JS	58	M	L: TPO+F1+CC
Westphal 1907 ¹⁵	NR	58	M	R: F and L: O+Th
Kleist 1907 ⁶	NR	44	M	L and R: F*
Kroll 1910 ⁵⁷	K	49	M	L: supramarginal and angular gyri.
	T	45	M	L: F2+supramarginal girus+gCC
Bonhöffer 1914 ¹⁹	HP	51	M	L: F1, F2+orbital+CC
Mingazzini and Ciarla 1920 ⁶⁶	I	60	M	R: internal capsule+L gCC
Schuster and Pinéas 1926 ⁹⁹	ML	70	W	L*
Maas and Sittig 1929 ¹¹⁴	AR	70	W	L and R.*
Zingerle 1934 ²⁸	MP	61	M	L and R.*
Eihelberg 1951 ¹¹⁵	27	59	W	ACA atherosclerosis (angiography) L>R+hydrocephalus
Meyer and Barron 1960 ²³	1	71	M	Anterior L and R*
	4	49	M	ACA L and R*
	7	59	M	ACA L and R.*
Petrovici 1968 ²⁵	IF	70	W	ACA L*
	MM	63	M	Multiple infarcts L and R*
Nutt <i>et al</i> 1993 ²⁰	NR	83	M	Multiple strokes*
	NR	68	M	Periventricular vascular disease*
	NR	81	M	NR

*Postmortem not available; ACA, anterior cerebral artery; CBD, corticobasal degeneration; CC, corpus callosum; F, frontal; F1, F2, F3, first, second, third frontal gyrus; gCC, genu corpus callosum; L, left; R, right; TPO, temporo-parietal-occipital; NR, not reported.

traced back to the compression of the areas adjacent to the frontal horns of the lateral ventricles.⁸⁵⁻⁸⁷

Neuropsychological remarks

Our patient showed no deficits in any other praxis domain. His performance on tests assessing upper limb ideomotor apraxia, utilisation apraxia, and facial apraxia was well above the threshold of normality. This finding points to a dissociation between GA and other kinds of apraxia. Durwen *et al*⁴¹ examined the case of patient IL who had a stroke in the territory of the middle cerebral artery, and presented with oral and ideomotor apraxia but not gait apraxia, a mirror picture of that presented by our patient. They argued that for GA to emerge a lesion within the territory of the anterior cerebral artery is necessary. Moreover, several authors³⁸⁻⁴⁰ have argued for a relative preservation of trunk movements compared with

ideomotor apraxia of the upper limbs, after parietal lesions (see further discussion in Howes⁴² and Poeck *et al*⁴³). Taken together, these findings point to a dissociation between GA associated with frontomedial lesions and ideomotor apraxia associated with (left) parietal lesions.

Indeed, lesions of frontomedial regions, in particular of the supplementary motor areas do not elicit ideomotor apraxia⁸⁸ (for reviews see Laiacina *et al*⁸⁹ and Marchetti and Della Sala⁹⁰). Watson *et al*⁹¹ reported the only two cases of bilateral apraxia after a lesion in the left supplementary motor area of which we are aware. Only one of them (case 1) was assessed at some length. However, the patient of Watson *et al* showed an asymmetry in performance: 13 months after the lesion, his apraxia was present only on the left hand,⁹² which leaves us with the suspicion that this patient's apraxia might be due to a lesion of the corpus callosum. The other patient with a supplementary

motor lesion studied in some detail had a crossed apraxia,⁹⁰ and therefore the rarity of her praxis organisation should be considered with some caution.

A careful analysis of case reports describing patients with anarchic hand and lesions to the supplementary motor areas⁹³ showed that ideomotor apraxia is extremely rare, if at all present. On the contrary, fleeting comments concerning difficulty that patients with anarchic hand may have controlling their gait or leg movements abound.⁹⁰⁻⁹³ These problems have been sometimes labelled (bilateral) “alien foot”,⁹⁴⁻⁹⁶ although their resemblance to GA symptoms is staggering. The opposite situation is also encountered: patients are reported showing a paramount GA who also presented with clear episodes of anarchic hand behaviour. For example, Van Vleuten⁹⁷ related that his patient AL (table 3) while being examined could not refrain from compulsorily unbuttoning his shirt with his left hand even when instructed not to do so. Kleist⁶ (table 3) also described a patient with multiple frontal strokes who was unable to inhibit endless spontaneous movements of his right hand which interfered with the actions he was requested to carry out. Our patient had a bilateral supplementary motor area lesion and, besides his overt GA, presented with a clear utilisation behaviour syndrome, which has been interpreted as a double anarchic hand.⁷⁴

In summary, lesions to one supplementary motor area could give rise to contralateral anarchic hand and contralateral GA (which, however, will be clinically hard to detect as such given that the nature of the actions performed require bilateral cooperation). Bilateral lesions to the same areas might produce utilisation behaviour and a full blown GA. Moreover, lesions to the medial areas of the frontal lobes are also likely to be associated with grasp reflex (*tonische Perseveration des Greifen*¹⁶; *Zwangsgreifen*⁹⁸; *Nachgreifen*⁶⁸⁻⁹⁹) which our patient showed from onset and throughout the follow up. On the other hand, ideomotor apraxia would be seen mainly after left parietal lesions.¹⁰⁰

The problem remains of the possible compensatory mechanism from the intact hemisphere in unilateral cases. Follow up studies of GA are rare. Our patient was still severely apraxic over a year after his stroke. The only other case with a longitudinal follow up we could cull from the literature is that of patient FZ described by Zingerle.²⁸ He was a 79 year old man, with alcoholism, arsenic poisoning, and possibly a right hemispheric stroke, whose severe GA improved considerably after a few months. The possibility that a unilateral GA might get better, whereas a bilateral one would not should remain speculative at this stage.

A cognitive interpretation

The puzzle of GA interpretation has haunted early authors and has yet to be resolved.²⁰ Hartmann⁵⁴ maintained that the frontal lobes contain a “*Merkzentrum für die höhere Koordination der Bewegungsbilder*” (an attention centre for the coordination of movement representations). Gerstmann and Schilder²² and Klein⁴⁴ revamped the concept of *gliedkinetische Apraxie*¹³ (also known as “innervatory”⁶ “cortical”¹⁷ “melo-kinetic”¹⁰¹ or “limb-kinetic”,¹⁰²⁻¹⁰³) to account for GA. Bonhöffer,¹⁹ Denny Brown,⁹ and Meyer and Barron²³ claimed it to be due to a contralateral frontal lesion. Kleist⁶ (p 952, table 3) maintained that area 6 contained a centre for the organisation of complex movements performed with trunk and lower limbs, such as dancing, hopping, or swapping the bearing of a walk. He also argued that this centre was non-lateralised (see also Liepmann⁵ pp 97–9). The link between supplementary motor areas and innervatory patterns has been recently advocated by Rothi *et al*¹⁰⁴⁻¹⁰⁵ though referring it to a mechanism necessary to code any gesture output lexicon into a space-time supramodal plan.

Our patient shows GA coupled with utilisation behaviour.⁷⁴ We held his bilateral supplementary motor areas lesion

responsible for both motor disturbances. Among their other functions¹⁰⁶ the two supplementary motor areas would supervise two apparently heterogeneous tasks—namely, locomotion and context independent utilisation. We support the hypothesis that bilateral GA, as found in our patient, should be considered a “motor” rather than an “ideomotor” apraxia. In fact, his deficits were present in all attempts independent of the context and not only on imitation.

Finally, we contend that GA has its own specificity, the diagnosis of which is possible only after having excluded other possible causes of gait disturbances. To account for this specificity we capitalise on a hypothesis put forward by Pözl.²⁹ He deemed that the change of the locomotor technique from four to two footed upright position, was paralleled by the phylogenetic enlargement of the frontal cortex (Fuster¹⁰⁷ pp 174–81). He also reckoned that the coordination of gait in humans piggybacked on this process, and the frontal lobes took it over. This could have given rise to an anatomical segregation of praxis abilities. Those devoted to hand movements and the skilful use of tools would be centred on the left parietal lobe. Those required for the complexity of the mostly automatic gait movements would be carried out by the frontal lobes, in particular by the supplementary motor areas, which in this frame would serve a self monitoring function.¹⁰⁸

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