

Motor neglect

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SUMMARY Motor neglect is characterised by an underutilisation of one side, without defects of strength, reflexes or sensibility. Twenty cases of frontal, parietal and thalamic lesions causing motor neglect, but all without sensory neglect, are reported. It is proposed that the cerebral structures involved in motor neglect are the same as those for sensory neglect and for the preparation of movement. As in sensory neglect, the multiplicity of the structures concerned suggests that this interconnection is necessary to maintain a sufficient level of activity. Predominance of left sided neglect by right sided lesions suggests that the left hemisphere is dominant for deliberate activity; hemispheric dominance could be applied to sensory neglect where conscious awareness would play the role of deliberate activity.

The terms unilateral motor neglect or unilateral hemi-inattention are used nearly interchangeably in the classic as well as in contemporary literature and reviews. Neglect may either be global¹ or partial (dissociated), as is the case with a visual and spatial neglect, a sensory neglect, a hemicorporeal neglect and even an olfactory neglect.² Unilateral underutilisation of the limbs is frequently reported as a phenomenon associated with the "Neglect Syndrome". Its isolated appearance was mentioned in the older literature²⁻⁴ under various names. With P Castaigne,^{5,6} we have drawn attention to a disturbance of spontaneous movement involving one half of the body and having the appearance of hemiplegia, yet with normal strength and dexterity, which can be proven by prompting an extraordinary effort on the part of the patient during the examination. We have called this disorder Motor Neglect.

The purpose of this communication is to present 20 cases of motor neglect in which localisation of the lesion is sufficiently well defined to allow a discussion of the topography of the lesion and the possible pathophysiological mechanisms involved.

Patients

Motor neglect was unilateral in each of the cases and included, on the affected side, the following characteristics: underutilisation of the upper extremity for

tasks that could be performed with the "healthy side" even when this was inconvenient (for example when they required a change in position of the body); non participation or feeble participation in bimanual tasks (such as clapping, opening a bottle, buttoning or unbuttoning a garment); under- or non-participation of the hand in gesturing when speaking; lack of arm swing when walking. This spontaneous underutilisation contrasted with near normal movement and strength, when the examiner actively encouraged the patient to use the arm. In some cases, the patient described the disturbance by saying that the hand was lazy or unreliable, although the required task finally was performed correctly. The patient had to "command" the hand to perform, he had to think of using it. In other cases, when the right hand was affected, the patient would say that he had become a left hander whereas he had been right handed. The disturbance rarely affected the upper limb alone; it usually involved both extremities but predominantly the upper. In the lower extremity the disorder was manifested by a lag in movement and a reduced range of motion, and automatic movements were specially disturbed: the affected leg lagged behind the good one when walking, or the leg stayed on the bed when the patient attempted to get up, causing falls. Here, as in the case of the arm, deliberate effort would compensate for the disturbance.

In order to avoid confusing motor neglect with classical hemiplegia we have reported in this series only cases that did not have a marked reduction of muscle strength or other motor or reflex disturbance. We have included cases with hypotonia and other disorders of movement that increased or

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tended to make motor neglect more obvious, but which were not constant; these were: (1) Lack of spontaneous "placing reaction". This was almost constant. When the patient was sitting, he let his hand rest along his body or between his legs rather than putting it "normally" on his thigh or on the arm of a chair. The leg could also be left in an uncomfortable position such as behind the body, or beside the chair, sometimes "lying on the back of the toes". In some cases, it tended to upset the equilibrium. When the subject moved from one place to another, no attempt was made by the hand to avoid hitting objects (for instance, the back of a chair); and such incidents did not produce change in the patient's posture. At times, the hand could be left to drag passively on the surface of a bed or table. As the patient got into bed, the arm or leg would be left hanging out of bed. In other instances, the arm might be caught up under the body or the leg be crossed under the healthy leg in an uncomfortable position which the patient did not seem to notice. However, in all cases, a comment from the examiner or a mere exhortation to assume a better position without specification, caused the patient to rectify his posture. (2) The insufficient or delayed reaction to assume correct posture could be so severe as to be absent; in this case the patient, losing his balance, fell to the affected side. There was no attempt to avoid the fall, or to minimise the shock. (3) There was a lack of automatic withdrawal reaction to painful stimulation, which could be striking: the patient, who appreciated pain normally, did not move the limb away although he protested and attempted to use the healthy limb to retrieve the affected one or fence away the painful stimulus. This sign, at times, was difficult to interpret as some patients believed that they must stoically sustain painful stimulation. (4) In some cases voluntary gesture was faulty and it appeared as if there was an error in the appreciation of the necessary energy to reach a point in the corporeal or extracorporeal space; the movement always fell short of the target (hypometria). For instance, when the patient is asked to put his finger to his nose, he bent his head forward to compensate for the inadequate movement of the arm while, in other circumstances, he was able to raise the elbow much higher, as, for instance, when pointing at the ceiling.

To prevent confusion with the syndrome of sensory neglect, we have excluded from our study, all patients presenting sensory disturbances including those with asomato-agnosia or denial. Some of our patients presented some degree of visual neglect from the beginning; in two cases this neglect appeared to be secondary to extension of the original lesion.

In all of the cases in this report, the precise anatomical localisation of the lesion has been assessed by the associated findings on neurological examination, surgical intervention and/or anatomopathological verification. (Cases of motor neglect associated with subdural hematoma have been eliminated from the study as they do not lead to anatomical conclusions.)

Results

Twenty cases of pure motor neglect have been observed; 15 had frontal lesions, four parietal lesions and one a thalamic lesion. All the patients considered themselves to be right handed. The low number of cases of motor neglect or parietal origin is due to the fact that we excluded patients presenting sensory neglect syndromes or other sensory disturbances that may represent elements of sensory neglect. The findings of the motor neglect syndrome were always on the opposite side to the lesion, and are summarised in table 1 for 12 patients with lesions in the right hemisphere and in table 2 for eight patients with left hemispheric lesions.

The retro-Rolandic lesions were all large glioblastomas that did not easily lend themselves to anatomic-clinical correlations. Two were essentially parietal, one parietotemporal, and one temporal but with "laminating" of the parietal area which showed multiple neuronal changes and gliosis.

The frontal lesions were in three groups:

- (A) Six small lesions (five metastases—cases 2, 3, 6, 13 and 14—and one glioblastoma—case 5) located in the white substance of the posterior part of F1 overlapping F2 in front of the pre-Rolandic sulcus.
- (B) Lesions larger in size and/or involving less well defined anatomical landmarks and comprising three cases of corticectomy of the medial aspect of the frontal lobe including the supplementary motor area and the adjacent cingulate area⁷ performed for intractable epilepsy (cases 7, 8 and 16); a case of ischaemia localised only by gamma scintigraphy in the pre-Rolantic area (case 1); a parasagittal meningioma under coronal suture (case 4); and a left frontal glioblastoma (case 15).
- (C) Flat lesions ("en plaque") involving the external cortex of the frontal lobe (meningioma "en plaque" case 10, and frontal lobectomy indenting and cutting through the back of the posterior part of F1-F2, case 9).

The thalamic lesion consisted of a nucleus of confluent lacunae in the ventro-lateral region of the thalamus, but overlapping the internal capsule and the subthalamic region.

In all of our patients, motor neglect lasted a few days to a few weeks. After that, it evolved either to

Table 1 *Motor neglect lesions of the right cerebral hemisphere*

Case no	Location of lesion	Type of lesion	Abnormal placement	Lack of withdrawal to pain	Hypometria	Visual neglect
1*	Frontal	Ischaemic	Yes	Yes	Yes	Yes
2†	Frontal	Metastasis	Yes	Yes	Yes	Yes
3†	Frontal	Metastasis	Yes	?	?	No
4	Frontal	Meningioma	Yes	No	No	No
5	Frontal	Glioblastoma	Yes	Yes	Yes	No
6	Frontal	Metastasis	Yes	Yes	No	No/Yes (-)
7‡	Frontal	Corticectomy	?	Yes	No	?
8‡	Frontal	Corticectomy	?	Yes	No	?
9	Frontal	Lobectomy	Yes	No	Yes	No
10	Frontal	Meningioma	Yes	Yes	Yes	No
11§	Parietal	Glioblastoma	Yes	Yes	Yes	No/Yes (-)
12	Thalamus	Ischaemic	Yes	Yes	No	?

* Castaigne P. *et al.*⁵† Castaigne P. *et al.*⁶‡ Laplane D. *et al.*⁷§ Vitrey *et al.*⁸|| Laplane D.⁹(-) The visual neglect has appeared in a 2^d time.Table 2 *Motor neglect lesions of the left cerebral hemisphere*

Case no	Location of lesion	Type of lesion	Abnormal placement	Lack of withdrawal to pain	Hypometria	Visual neglect
13†	Frontal	Metastasis	Yes	Yes	Yes	Yes
14	Frontal	Metastasis	Yes	No	No	No
15	Frontal	Glioblastoma	Yes	?	Yes	Yes
16‡	Frontal	Corticectomy	No	Yes	No	No
17	Frontal	Ischaemic	Yes	No	No	No
18	Temporoparietal	Glioblastoma	?	No	No	No
19*	Temporal	Glioblastoma	Yes	Yes	Yes	Yes
20*	Parietal	Glioblastoma	Yes	Yes	Yes	No

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hemiplegia (due to spreading of the lesion) or recovery (removal of the lesion or spontaneous recovery following ischaemic injury or surgical corticectomy).

Discussion

Unilateral pure or isolated motor neglect, defined by the existence of spontaneous non-utilisation or underutilisation of the limbs on one side, with near normal strength and utilisation as a result of deliberate effort has been found in 20 patients without sensory deficit. The lesion responsible for this syndrome was either in the frontal lobe where, in six out of 15 of our cases, it was limited to the white matter in the depth of the posterior part of F1 and of overlapping F2, or was large and retro-rolandic, involving the parietal lobe. In one case, the lesion was in the ventrolateral nucleus of the thalamus. The clinical syndrome of motor neglect was identical regardless of the site of the lesion. The site of lesion could only be

suspected from disturbances sometimes associated with the syndrome of motor neglect (for example, clinical frontal lobe findings, disorder of language, hemianopsia or visuo-spatial disorders and cerebellar syndrome) and not from the motor neglect itself. Isolated unilateral motor neglect involving disorder of "automatic" placement of the limbs is in our experience, due to a contralateral lesion of the region of the foot of F1. In some cases, this finding allowed us to direct surgical investigations and intervention to the appropriate region.

Unilateral reduction in the range of motion, disproportionate to the motor deficit, is observed in patients affected with various disorders, including a left hemiparesis, hemiparkinsonism, hemichorea,¹⁰ thalamic syndrome,¹¹ and unilateral neglect.^{12,13} The reduction in the range of motion is often cited only in passing; when this phenomenon is analysed it is often done in a psychological manner, the preference for utilising the healthy hand being considered as "quite natural". Some authors have, however, offered an interpretation which takes into account

the organisation of movement, disorder of the sensory afferents,^{12,14} hemi-inattention.¹

Isolated unilateral underutilisation has already been described in a more or less explicit manner and under various names. Hartmann³ described an akinesia of the left limbs secondary to an ischaemic lesion of the right frontal white matter at the level of F2. Liepmann in 1908⁴ took the psychoparalysis on "Bruns" paralysis out of the category of apraxia: "This disturbance is not linked to loss of memory for the movement but rather to the fact that the limb is underutilised; this is why, when the movement is achieved, it is normal. This abnormality is therefore half-way between paralysis and apraxia. The seat of the disorder is probably the central cerebral cortex, and includes perhaps, the adjacent anterior and posterior cortex". Wilson in 1908¹⁵ reported an observation of "absence of initiative". The patient was a wounded man observed after the extraction of a bullet lodged into the right parietal lobe. For a few days, he displayed astereognosis with disorders of deep sensation which appeared mild, and a non-utilisation of the left hand in spite of normal strength. Thomas and Ajuriaguerra¹⁶ described similar patients in which they gave a detailed description of the deficit which is very much like our own; in the absence of any sensory or motor abnormality, the patients underutilised the limb. They interpreted this as a sign of hemispheric lesion, without giving a more precise localisation. Zoll¹⁷ reported five cases of motor neglect which, strangely, he called anosognosia following thalamic coagulation; he incriminated lesions of the ventral intermediate nucleus of the thalamus as responsible for the syndrome.

We have explored the existence of motor neglect due to parietal lesions and upper frontal lesions^{5,6} in the absence of motor and sensory deficits and in the absence of other elements of the syndrome of neglect. Recently, Valenstein and Heilman,¹⁸ observed unilateral hypokinesia and motor extinction of the left side after haemorrhage affecting the right caudate nucleus. Although the technique of examination was very different from the one we used, the cases were probably similar. However, the lesions were far from being limited to the caudate nucleus, but affected the entire anterior limb of the internal capsule and thus resembled the frontal lesion present in some of our patients.

It is only recently that motor neglect has been studied in animals. However, in the past several studies dealt indirectly with the subject. The unilateral or asymmetric destruction of the posterior columns¹⁹ or of the pyramidal tract²⁰ causes an under-utilisation of the corresponding limbs which can be partially ameliorated by creating or aggravating contralateral lesions. Unilateral hypokinesia was

described after contralateral lesion of the tegmentum of the upper pons and of the mesencephalon,²¹ by a lesion located at any point of the rubro-olivo cerebellar loop²² and by an ipsilateral hemicerbellectomy.²³ Humphrey²⁴ published a review of disorders of motility observed in man and animals affected by parietal lobe lesions. He reported a marked decrease in the frequency of spontaneous, purposeful movements and of visual tactile placing responses of the arm and hand contralateral to the site of the lesion. He stated that "it may be premature to extract from the numerous and sometimes varying descriptions of the parietal lobe syndrome, a particular set of disturbances that appears to be primarily attentive or motor in nature". However, Watson *et al*²⁵ showed, thanks to cross-conditioning stimulation, that the motor neglect, observed in the monkey after a lesion of the arcuate fasciculus or of the mesencephalic reticular formation, is "induced by the loss of the intention to make the correct motor response for a perceived stimulus and not by sensory or sensory attentional defect". One wonders whether the motor behaviour of unilateral neglect syndromes following either frontal lesions²⁶ parietal lobe,²⁷ anterior cingulate gyrus²⁸ or mesencephalic reticular formation^{29,30} are all dependent upon the same mechanism. The topographic analogy that exists between the structures which when injured result in unilateral neglect in animals and the structures whose lesions result in motor neglect in man is striking. It is all the more so because in addition to the cases presented here, we have also found cases of motor neglect associated with other neurologic signs that indicated that the lesion responsible was probably, though not certainly (which made us exclude them from this study) located in the mesencephalic area.³¹

The motor neglect mechanism cannot be attributed either to a sensory disorder or to a sensory neglect syndrome (patients with these findings were excluded from this study). The hypothesis of a hemispheric hypo-arousal initially put forward by Heilman¹ does not allow for the explanation of unimodal neglect. Yingling *et al*³² partially removed this difficulty by showing that the stimulation of specific parts of the nucleus reticularis thalami abolished the corresponding evoked cortical responses (visual/auditory or tactile). However, we are not sure that these findings apply to unimodal or predominantly unimodal neglect due to frontal or parietal cortical lesions. It is even less clear that they apply to motor neglect.

It seems more logical to regard motor neglect as the result of a defect of movement preprogramming and organisation. It is striking that the structures, in which lesions are liable to result in motor neglect are

concerned with the preparation or planning of movement: the parietal association areas,^{33,34} secondary motor area,³⁵ the premotor cortex,²⁴ the thalamus, and notably the ventrolateral nucleus.³⁶⁻⁴⁰ These structures and the motor areas are, physiologically and anatomically, largely interconnected.²⁴ Therefore, one can hypothesise that motor neglect is a disorder related to a defect in the "triggering" of these structures which prepare and programme movement and that the interconnections between these different structures are necessary to maintain each one at a sufficient functional level. It is possible that the underutilisation observed in the partial and asymmetrical lesions of the sensitive afferents¹⁹ or of the pyramidal tract²⁰ or in Parkinsonism may be due to the same mechanism of the lowering of the functional level of all the structures of a hemisphere participating in the organisation of motricity. As regards the motor neglect itself, the reversibility of this disorder in situations of emergency, when it becomes urgently necessary to act, proves that other possibilities for a "preparatory" motor system do exist. The transitory character of the motor neglect in the presence of non-progressive lesions (surgical corticectomy) shows also the existence of alternative systems.

Valenstein and Heilman¹⁸ offer two possible explanations for the hypokinesia and motor extinction they observed. First they compared the disturbance to the unilateral hypokinesia induced in animals by lesions of the dopaminergic system. However, as already pointed out, the lesions of the patients are far from being limited to the caudate nucleus but also affect the anterior limb of the internal capsule and tend to resemble the frontal lesions of our patients. The second possible explanation was that the activation system of the healthy hemisphere helps the diseased hemisphere (in their cases the right hemisphere) in planning motor actions except when it is occupied with its own tasks. This interpretation is compatible with the one we now propose.

In the present series of cases of motor neglect, the method of patient selection was responsible for the weak preponderance of patients with right hemispheric lesion and left motor neglect. Cases in which some form of sensory neglect was present were excluded with the purpose of demonstrating the independence of pure motor neglect. However, if we had included all cases of motor neglect observed, regardless of the type of associated neurological findings, we would have found a higher incidence of right hemispheric lesions as all other authors have in unilateral neglect. In order to explain this prevalence, Heilman and Watson¹ suggested that the corticoreticular loop, which they thought to be the anatomical support of the unilateral hemispheric

arousal, is more discretely organised in the right hemisphere, a statement they do not support with facts. Recently, Heilman and Vanden Abell⁴ have offered another hypothesis according to which the right hemisphere has a dominant role in attention. It remains to be demonstrated whether this hypothesis is well founded, and if it is, whether it offers a real explanation.

In the circumstances in which our patients were examined it was verbal exhortation which made apparent the dissociation between motor ability and automatic utilisation of the affected limbs. Thus, rather than speaking of a voluntary effort, as is usual, it would probably be more accurate to suggest that the reason for improvement is a motivation set up from language. The role of language appears to play an important role during examination; even when the patient is not given explicit instructions, he feels motivated by the presence of the examiner and attempts to guess what it is that the observer wants from him. Therefore, he has a tendency to reproduce or repeat behaviour that has been required from him previously. Even outside of the examining situation, the role of language in voluntary effort is naïvely, but strongly, shown by the patients who explain that they must order the lazy hand to perform.

There are several other reasons for believing that in all deliberate motor acts of man, the dominant hemisphere plays a preponderant role. The evidence is drawn from observations made in patients with hemisphere disconnection: following a division of the cerebral commissures, not only "all patients showed some degree of left-sided apraxia to verbal commands" for an extremely variable period of time (one day to more than 5 years)⁴² but also "there was a tendency to use the left hand less than usual under ordinary circumstances. Special effort and stimulation were often necessary to bring the left hand into action".⁴³ In a case of Marchiafava-Bignami disease with hemisphere disconnection syndrome, Barbizet *et al*⁴⁴ have observed the same paucity of gesturing with the left limbs. In addition a specific study of the patient's behaviour while performing everyday routine movements could lead to the assumption that the left hand was not, or was imperfectly aware of what the patient wanted to do deliberately, even under the influence of a primary drive, as in drinking or smoking. Similar observations seem to have been made by Schott *et al*.⁴⁵ They write: "during the examination, the patient denies being left-handed and wants to use his right hand, and in everyday gestures the left hand acts, but every so often it opposes and resists the right hand, which is always more efficient." Akelaitis⁴⁶ described similar behaviour, although he curiously interpreted it as

being of epileptic or psychiatric origin. This role of language dominance in motor intention of deliberate actions agrees with the existing correlation between the preferred utilisation of the right limbs and the speech dominance of the left cerebral hemisphere, in right-handed individuals. Inhibition of motor performances of the right hand, but not of the left, by interference of verbal tasks has been reported in right-handed individuals.⁴⁷⁻⁵⁰ This strongly suggests that motor organisation of the right arm is language dependent.

We propose another possible explanation of left lateralisation of motor neglect: different lesions of the systems that programme and organise movement²⁴ might cause a defect in the initiation and follow through of motion; if the lesion is located in the right hemisphere, verbal incitation and/or the deliberate will of the subject elaborated by the left hemisphere may compensate, at least partially, for the motor disturbance, as long as the corpus callosum is intact. On the other hand, in the left hemisphere language and movement are so intricately organised that it makes the occurrence of the characteristic dissociation of motor neglect less likely. The predominance of left sided motor neglect could not be verified in lesions located at a subcortical level, for example in the thalamus.¹⁷ One can assume that at this level, the same alternative processes through verbal exhortation and deliberate voluntary effort may be at play in the left as well as the right hemisphere. On this hypothesis, it is assumed that the left hemisphere, responsible for language, plays a dominant role in the programming and organisation of movement, through deliberate conscious effort; and that movement of the right limbs and language are intimately related because their organisation has developed simultaneously; this may be either genetic, educational or both.

One must stress that the language in question is not necessarily a very elaborate one. Encouragements at a very simple linguistic level improve the performance of the "neglected" hand. There are in fact reasons to believe that the left hemisphere plays, from birth, a predominant role in the discrimination of sounds in the mother's language;⁵¹⁻⁵² the role of maternal language in inciting movement is quite evident. Several sorts of evidence support the idea that the mechanism of motor and sensory neglect are closely related: frequency of association, topographic proximity of lesions causing both of them; secondary appearance of visual neglect in two of our patients due to the growth of the tumour responsible for the initial motor neglect. Thus the hypothesis that we propose to explain motor neglect could also be applied to sensory neglect. Awareness, that is conscious awareness, would play the role of

deliberate and purposeful effort in motor neglect, assuming that it has the same relationships with the left hemisphere and language.

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Since acceptance of our manuscript, a study on "None sensory neglect from parieto-temporal lesions in monkeys" by Valenstein E, Heilman KM, Watson RT and Van Den Abell T has been reported. It confirms our suggestion that the motor behaviour of neglect observed in parietal lesions should be interpreted as "non sensory".

References

- ¹Heilman KM, Watson RT. Mechanisms underlying the unilateral neglect syndrome. In: *Advances in Neurology* New York: Raven Press 1977;18:140.
- ²Friedland RP, Weinstein EA. Hemi-inattention and hemisphere specialization: introduction and historical review. In: *Advances in Neurology* New York: Raven Press 1977;18:1407-31.
- ³Hartmann F. Beiträge zur Apraxielehre. *Motschr. J Psychiatry Neurol* 1907;21:71-111, 248-70.
- ⁴Liepmann H. *Aufsätze aus dem Apraxiegebiet*. Berlin: S. Karger, 1908.
- ⁵Castaigne P, Laplane D, Degos JD. Trois cas de négligence motrice par lésion rétro-rolandique. *Rev Neurol (Paris)* 1970;122:233-42.
- ⁶Castaigne P, Laplane D, Degos JD. Trois cas de négligence motrice par lésion frontale prérolandique. *Rev Neurol (Paris)* 1972;126:5-15.
- ⁷Laplane D, Talairach J, Meininger V, Bancaud J, Orgogozo J. Clinical consequences of corticectomies involving the supplementary area in man. *J Neurol Sci* 1977;34:301-14.
- ⁸Vitrey M, Trouillas P, Aimard G. Un nouveau cas de négligence motrice. *Lyon Médical* 1974;232:283-5.
- ⁹Laplane D. Considération sur la place de la négligence motrice dans les désorganisations du mouvement. In: *Livre Jubilaire PF Girard*. Lyon SPCM eds. 1976.
- ¹⁰Thomas A, de Ajariaguerra J. Observation anatomoclinique d'un cas de chorée molle. *Presse Médicale* 1945;53:575.
- ¹¹Castaigne P, Cambier J. La séméiologie des lésions thalamiques. *Presse Médicale* 1969;72:1101-3.
- ¹²Critchley M. *The parietal lobes*. New York: Hafner 1953.
- ¹³Heilman KM, Valenstein E. Frontal lobe neglect in man. *Neurology (Minneapolis)* 1972;22:660-4.
- ¹⁴Denny-Brown D, Meyer JS, Horenstein S. The significance of perceptual rivalry resulting from parietal lesion. *Brain* 1952;75:433-71.
- ¹⁵Wilson SAK. A contribution to the study of apraxia. *Brain* 1908;31:164-216.

- ¹⁶ Thomas A, de Ajariaguerra J. *Etude sémiologique du tonus musculaire* Paris: Flammarion 1949.
- ¹⁷ Zoll JG. Transient anosognosia associated with thalatomy: is it caused by proprioceptive loss? *Confin Neurol* 1969;**31**:48–55.
- ¹⁸ Valenstein E, Heilman KM. Unilateral hypokinesia and motor extinction. *Neurology (NY)* 1981;**31**:445–8.
- ¹⁹ Gilman S, Denny-Brown D. Disorders of movement and behaviour following dorsal columns lesion. *Brain* 1976;**99**:397–418.
- ²⁰ Gilman S, Marco LA. Effects of medullary pyramidotomy in the monkey. *Brain* 1971;**94**:495–514.
- ²¹ Poirier LJ. Experimental and histological study of mid brain dyskinesias. *J Neurophysiol* 1960;**23**:534–51.
- ²² Laroche L, Bedard P, Boucher R, Poirier CJ. The rubro-olivo-cerebello-rubral loop and postural tremor in the monkey. *J Neurol Sci* 1970;**11**:53–64.
- ²³ Gilman S. The mechanism of cerebellar hypotonia; an experimental study in the monkey. *Brain* 1969;**92**:621–38.
- ²⁴ Humphrey DR. On the cortical control of visually directed reaching: contribution by precentral motor areas. In: Talbott RE and Humphrey DR, eds. *Posture and Movement*. New-York: Raven Press 1979:51–112.
- ²⁵ Watson RT, Miller B, Heilman KM. Non sensory neglect. *Ann Neurol* 1978;**3**:505–8.
- ²⁶ Welch K, Stutteville P. Experimental production of unilateral neglect in monkey. *Brain* 1958;**81**:341–7.
- ²⁷ Heilman KM, Pandya DN, Geschwind N. Trimodal inattention following parietal lobes ablation. *Trans Am Neurol Assoc* 1970;**95**:259.
- ²⁸ Watson RT, Heilman KM, Cauthen JC, King FA. Neglect after cingulectomy. *Neurology (Minneapolis)* 1973;**23**:1003–5.
- ²⁹ Reeves AG, Hagaman WD. Behavioral and EEG asymmetry following unilateral lesions of the forebrain and midbrain of cats. *Electroencephalogr Clin Neurophysiol* 1971;**30**:83–6.
- ³⁰ Watson RT, Heilman KM, Miller BD, King FA. Neglect after mesencephalic reticular formation lesions. *Neurology (Minneapolis)* 1974;**24**:294–8.
- ³¹ Degos JD, Verroust J, Bouchareine A, Serdaru M, Barbizet J. Asterixis in focal brain lesions. *Arch Neurol* 1979;**36**:705–7.
- ³² Yingling CD, Skinner JE. Gating of thalamic input to cerebral cortex by nucleus reticularis thalami. In: Desmedt JE, ed. *Attention, Voluntary Contraction and Event-related Cerebral Potentials*. Progress in Neurophysiology. New York: Karger 1977;**1**:70–96.
- ³³ Kornhuber H. *Handbook of sensory physiology*. Springer Verlag: vol. VI, part. 2, *Vestibular system*, 1974:600.
- ³⁴ Mountcastle VB, Lynch JC, Georgopoulos A, Sakata H, Acuna C. Posterior parietal association cortex of the monkey: command function for operation within extrapersonal space. *J Neurophysiol* 1975;**38**:871–908.
- ³⁵ Tanji J, Taniguchi K. Does the supplementary motor area play a part in modifying motor cortex reflexes? *J Physiol (Paris)* 1978;**74**:317–8.
- ³⁶ Evarts EV. Activity of ventralis lateralis neurones prior to movement in the monkey. *Physiologist* 1970;**13**:191.
- ³⁷ Joffroy AJ, Lamarre Y. Single cell activity in the ventral lateral thalamus of the unanesthetized monkey. *Exp Neurol* 1974;**42**:1–16.
- ³⁸ Massion J. Rôle possible de l'activité rythmique du noyau ventro-latéral dans la prédétermination centrale du mouvement. *J Physiol (Paris)* 1968;**60**:497–8.
- ³⁹ McPherson JM, Rasmussen DD, Murphy J. Activities of neurones in "motor" thalamus during control of limbs movement in the primate. *J Neurophysiol* 1980;**44**:11–28.
- ⁴⁰ Umbach W, Ehrhardt KJ. Microelectrode recording in the basal ganglia during stereotaxic operations. *Confin Neurol* 1965;**26**:315–7.
- ⁴¹ Heilman KM, Van den Abell T. Right hemisphere dominance for attention. The mechanism underlying hemispheric asymmetries of inattention (neglect). *Neurology (Minneapolis)* 1980;**30**:327–30.
- ⁴² Gazzaniga MS, Bogen JE, Sperry RW. Dyspraxia following division of the cerebral commissures. *Arch Neurol* 1967;**16**:606–12.
- ⁴³ Sperry RW, Gazzaniga MS, Bogen JE. Interhemispheric relationships: the neocortical commissures; syndrome of hemisphere disconnection. Vincin PJ, Bruyn GW, eds *Handbook of Clinical Neurology*. Amsterdam N Holland Pub Co: 1969;**4**:273–90.
- ⁴⁴ Barbizet J, Degos JD, Lejeune A, Leroy A. Syndrome de dysconnexion interhémisphérique avec dyspraxie diagnostiqué au cours d'une maladie de Marchiafava-Bignami. *Rev Neurol (Paris)* 1978;**134**:781–1006.
- ⁴⁵ Schott B, Michel F, Michel D, Dumas R. Apraxie idéomotrice gauche avec main gauche anomique. Syndrome de déconnexion calleuse. *Rev Neurol (Paris)* 1969;**120**:359–65.
- ⁴⁶ Akelaitis AJ. Studies on the corpus callosum. IV. Diagnostic dyspraxia in epileptics following partial and complete section of the corpus callosum. *Am J Psychiatry* 1944;**101**:594–9.
- ⁴⁷ Kinsbourne M, Cook J. Generalized and lateralized effects of concurrent verbalization on a unimanual skill. *Q J Psychology* 1971;**23**:341–5.
- ⁴⁸ Hicks R. Interhemispheric response competition between vocal and unimanual performance in normal adult males. *J Comp Physiol Psychol* 1975;**89**:50–60.
- ⁴⁹ Lomas J, Kimura D. Intrahemispheric interaction between speaking and sequential manual activity. *Neuropsychologie* 1976;**14**:23–4.
- ⁵⁰ Bowers D, Heilman KM, Satz P, Alzman A. Simultaneous performance on verbal, non verbal and motor tasks by right handed adults. *Cortex* 1978;**14**:540–56.
- ⁵¹ Kasman-Entus A. Hemispheric asymmetry in processing of dichotically presented speech and non speech stimuli by infarcts. In: Segalowitz ST and Gruber FA, eds. *Language Development and Neurological Theory*. New York: Academic Press 1977.
- ⁵² Bertoncini J, Mehler J. Quelques remarques à propos de la perception chez le nouveau-né. *Revue du Praticien* 1981;**31**:387–96.