Letters

"phénomène des orteil" was present, and in the third case "tickling the plantar surface of the foot gives reflex movements of the whole limb". As Babinski was concerned in this paper with demonstrating that there is such a thing as paraplegia in flexion without involvement of the cortico-spinal tract, he seems to have failed to realise that he had recorded different forms of plantar response, one of which was the Babinski response, all present with normal cortico-spinal tract. On his own premises, his Case 2 proved his supposition about the significance of "le phénomène des orteil' to be wrong".

Babinski reported in 1899 three cases of paraplegia in flexion. Though the title runs "Sur une forme de paralysie spasmodyque consécutive à une lésion organique et sans dégénération du système pyramidal", but actually, the first case is "une tumeur paraît, s'être développée aux dépens de l'extrémité du plexus choroïde du 4e ventricule", the second case is "dans la région dorsale supérieure une tumeur ovoïde, grosse comme un œuf de moineau, qui distend le sac de la dure-mère et comprime la moelle" and the third case is "à l'œil nu ne on ne voit nettement sur la moelle qu'une large plaque de sclérose siégeant à droite dans le faisceau antéro-latéral, aux niveaux des émergences des racines antérieures; cette plaque occupe toute l'épaisseur du manteau blanc et envahit un peu la corne antérieure ... Au microscope les coupes des régions cervicales inférieure et dorsale supérieure montrent dans le point indiqué, une plaque de sclérose multiloculaire parfaitement typique; les limites de cette plaque sont nettes et la sclérose est absolue en ce sens qu'il n'existe plus aucune trace de myeline dans toute son étendue, mais les cylindraxes sont conservés, quoique tuméfies; ...".

Nathan and Smith stated: "Case 2 proved his supposition about the significance of "le phénomène des orteil to be wrong". Apparently that is not what Babinski was trying to say. In the second case-report he said: "La compression est considérable et la moelle a pris à ce niveau la forme d'un croissant; elle n'a d'ailleurs pas subi une réduction de volume notable; elle est surtout déformée. En ce point il existe une sclérose considérable caractérisée par la disparition de la grande majorité des tubes de myeline, par l'épaississement des travées névrogliales et par les alterations hyalines des vaisseaux. Mais on voit une grande quantité de cylindraxes qui sont conservés malgré leur dénudation et leur tuméfaction. Il reste même un certain nombre de tubes encore pourvus de leur myeline". In the third case Babinski said: "Le chatouillement de la plante du pied provoque des mouvements très étendus de flexion du pied sur la jambe et d'extension des orteil sur le métatarses".

According to that mentioned above, there is no reason to think that Babinski's case-report in 1899 and his statement on "le phénomene des orteil" are in any way discrepant.

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Nathan and Smith reply: We are pleased to see Dr Jin-yu Hsia's interest in Babinski's work and our paper on the Babinski response. The title of Babinski's paper is Sur une forme de paralysie spasmodyque consécutive à une lésion organique et sans dégénération du système pyramidal.1 In this paper, Babinski was establishing that with a normal pyramidal system, one could get paraplegia in flexion without increased tendon reflexes and without clonus. His observations on the kind of plantar response obtained in these three cases was incidental. What we drew attention to in our paper2 was that the patient whom he reported as Case 2 had the "phénomène des orteil" (Babinski response) with what Babinski wrote was a normal pyramidal system. We pointed out that Babinski did not draw any conclusions from the fact that he had described his response with a normal pyramidal system.

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References

SIr: I would like to comment on the rheological findings reported by A Brunetti et al, Rheological and fibrinolytic findings in multiple sclerosis (J Neurol Neurosurg Psychiatry 1981;44:340). In this study whole blood viscosity is found to be significantly increased in patients suffering from multiple sclerosis. Plasma viscosity and haematocit changes cannot account for this increase. Hence the authors draw the conclusion that the increase in whole blood viscosity is brought about by a loss of the red cells' deformability. This is further supported by the authors' observation that the relative viscosity is also increased in multiple sclerosis.

However the factors affecting whole blood viscosity at shear rate 230 s−1 are not clear enough to allow this conclusion to be drawn without direct measurement. Firstly the patients were on steroids which has some effect on red cell deformability.1 Secondly an absence of correlation between relative viscosity and red cell deformability recently has been reported.2 Therefore it is possible that red cell deformability is decreased in multiple sclerosis—some membrane abnormalities have already been reported3—but this can only be established by a method measuring this factor more directly.

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References

GIROLAMI et al reply:
Sir: We have read with interest the comments by Ernst et al about our paper on "Rheological and fibrinolytic findings in Multiple Sclerosis". We agree with the assumption that red blood cell deformability could be determined using a specific method. However there is no sure method to date of evaluating such a parameter. Differential centrifugation and several filter methods have many pitfalls and variables and yield discrepant results.
Whole blood and plasma viscosity determinations even at elevated shear rates using the Brookfield or another adequate rotational viscometer, appear definitely more reliable that any deformability test so far developed. In our paper we have only claimed that relative viscosity, namely the ratio between whole blood and plasma viscosity, appeared increased in patients with multiple sclerosis indicating probably that red blood cell deformability was decreased. Of course this is true only if one admits the existence of a relationship between relative viscosity and red cell deformability. The possibility that steroid therapy might have affected significantly the viscosity determinations seems remote. In a group of patients with long standing Cushing's syndrome we have failed to show any significant change of whole blood or plasma viscosity.

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GIOVANNI M PATRASSI
LUIGI RICCHIERI
BRUNO TAVOLATO
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Childhood bacterial meningitis

Sir: A recent report described several cases of childhood bacterial meningitis with complications. In this series, the incidence of cerebral infarction was high and factors possibly implicated in the aetiology of this complication were discussed. Further consideration of this serious sequel to meningitis may be of interest.

Elevated levels of fibrinogen degradation products (FDP) in the cerebrospinal fluid (CSF) of patients suffering from viral or bacterial meningitis have been observed. In cases with complications (including one case with hemiplegia), elevated levels of FDP persisted in the CSF for several weeks. The authors concluded the origin of the FDP to be either the serum, with leakage into the CSF via a blood-CSF barrier damaged by inflammation, or from arteries damaged directly, as a consequence of the associated arteritis. Others, however, have suggested nervous tissue damage to be the source.

Whatever the origin of FDP in the CSF, these substances may play a crucial role in the pathogenesis of cerebral infarction. FDP are known to have several biological actions. They may increase platelet aggregation; they potentiate the action of many substances including peptides on smooth muscle and in addition to an independent contractile effect on human intracranial arteries, they enhance the effect of 5-hydroxytryptamine. FDP have also been shown to increase microvascular permeability—an effect abolished by β-receptor agonists, suggesting a possible therapeutic role for such drugs.

It seems possible that FDP play a central role in the pathogenesis of a variety of conditions in which cerebral arterial spasm may occur. If this is so, it may be of interest to measure FDP concentrations in the CSF of patients with meningitis to observe whether there is a correlation between high or increasing levels of FDP and the onset of complications such as cerebral infarction.

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University Department of Neurosurgery
Manchester Royal Infirmary

References


Matters arising: GIROLAMI et al reply:

Antonio Girolami, Giovanni M Patrassi, Luigi Ricchieri and Bruno Tavolato

*J Neurol Neurosurg Psychiatry* 1982 45: 185-186
doi: 10.1136/jnnp.45.2.185-b

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World Congress on Mental Retardation of the International Association for the Scientific Study of Mental Retardation, Toronto, Canada, 22-26 August, 1982. Address for information: IASSMD, Kinsmen Building, York University Campus, 4700 Keele Street, Downsview, Ontario, Canada M3J IP3.

CORRECTIONS

The authors of the paper “Physiological basis for enduring vestibular symptoms” (Journal of Neurology, Neurosurgery and Psychiatry 1982;45:126–130) were PRudge and BR Chambers.

Matters arising

The letter from Dr Edzard Ernst (Journal of Neurology, Neurosurgery and Psychiatry 1982;45:185) should have been entitled “Rheological and fibrinolytic findings in multiple sclerosis”.


individual opinions, with which the editors do not always concur: the well known divergence of opinion on the value of the various surgical techniques to treat tremor, spasmodic torticollis and dystonia illustrate the enthusiasm of the neurosurgeon and the modulated scepticism of the editors.

I found this a most valuable text, controversial, up to date, and in most sections balanced and authoritative. The rather obsessiionally long classifications (for instance one and a half text pages on myoclonus; 18 alleged patterns of drug related fluctuations and dyskinesia) tell us that their present basis is insecure and suggest that in time they will disappear as new information brings with it simplicity. Remember the anatomical nightmares of basal ganglia pathways we had to learn in the days of stereotactic thalamotomy? If the chapters on dopamine receptors and tardive dyskinesia were too brief or too specialised to be of general help to neurologists, this is a minor criticism.

I would venture the view that this is the outstanding work on Parkinsonism and movement disorders written to-date. The editors point out that it is not totally comprehensive, but as a statement of the position in the Parkinson game, some 15 years after Cotzias’s paper it is a first class compilation which I would warmly commend to all neurological departments.

JMS PEARCE


The Human Brain and Its Universe by the anatomist Kuhlenbeck is a book on philosophy and not on anatomy. Kuhlenbeck possesses a vast amount of learning and a deep understanding of mathematics, physics and philosophy. The main subject matter is consciousness and the data that our consciousness gives us. Of those who have thought about these questions—and all philosophers and many neurologists and psychologists have done so—Schopenhauer is one who is commonly neglected in English-speaking countries. Kuhlenbeck here speaks up for this philosopher. He clarifies his concept by showing that Schopenhauer’s use of the word “will” was a mistake. Schopenhauer used “will” to mean Kant’s Ding an sich, for matter regardless of any human being apprehending it and of any human way of apprehending it.

It is unfortunate that no English-speaking editor has helped Kuhlenbeck with his English, which is a mixture of a word-for-word translation of German and the long-winded Teutonic writing that the Americans have acquired from earlier German science and later refugees from Germany. But it is relieved every now and then by acid remarks about the author’s well-known contemporaries.

PETER W NATHAN


The two volumes, The World of Natural Sciences and Its Phenomenology and The Brain and Its Mind, overlap to such an extent that both should not have been published. Whole sentences and identical lengthy quotations appear in both of them. This book needed strict editing but clearly it got none.

The main subject of the book, as of the previous volume, is one that neurologists usually disregard: “How can material, ie physical spacetime events” produce mentation, or, “how do physical events become transmuted into consciousness, or how does matter give rise to mind?” An example is quoted by Kuhlenbeck: Ziehen wrote: “There is no pathway from the visual cortical cells that leads to the sensation of red.” But Kuhlenbeck’s style of writing is such that it is difficult to get his message.

Like Vol. 1, the book is mostly on philosophy; in addition, there are thoughts and facts from everywhere and anywhere, relevant and irrelevant. One cannot conceive of whom the book was written. The parts of the book on neurology are at very different levels. The reader is informed what a synapse is and told about dendrites and neurons. At the same time, he is supposed to know what “the brain stem’s tegmentum” is, whereabouts the central tegmental tract is, to understand gating mechanisms, and to know what neuropeptides are. He is told that “medullated neurites (‘white fibers’) are coated by a laminated lipid wrapping representing the myelin sheath produced by oligodendrogli a respectively by Schwann cells”; and at the same time he is presumed to understand “Cerebral grisea are not only influenced by hormones but also to some extent produce hormones”.

Kuhlenbeck makes no special contributions to the elucidation of this problem that comes from neurology and that could only have been contributed by a distinguished neuroanatomist. And so the reader loses heart as he ploughs through 346 pages of assertions on every subject in the world, occultism, spiritualism, Swedenborg, Buddhism, Berger’s views on the encephalogram and philosophy, cerebral localisation, and Marx and Engels.

PETER W NATHAN

Notice

The 11th Annual Meeting of the International Neuropsychological Society will be held 2–5 February, 1983 in Mexico City, Mexico. For information write to: Dr Manfred Meier, Arrangements Coordinator, Box 390 Mayo, University of Minnesota Medical School, Minneapolis, MN 55455, USA.

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