mental data. Longitudinal surveys should allow us to find out, while the disease is running its course, whether the specific elementary defects appear in isolation first and more complex impairment only later on along with other cognitive defects (viz., impairment in attention and automatization, in behavioural planning, in intelligence, memory and even in some verbal abilities). By definition, complex behavioural disorders are multifactorial in nature and are poorly informative of the first hampered functions (such as Parkinson’s spatial defects), being admittedly more representative of the everyday coping disorders.

Summing up, we presently share a wait-and-see position on the specification, if any, of the role in cognition of the basal ganglia, at least when conclusions stem from data collected from patients with progressive neurological “subcortical” disease; we are inclined to lend great credit to eventually forthcoming longitudinal studies, starting from reasonably homogeneous and “pure” samples (viz., employing very early patients).

On the other hand, we feel rather sceptical on the general conclusions drawn from the current cross-sectional cognitive studies which yield ever increasing discrepancies. For instance, still unpublished findings of ours on the strategy-producing ability linked to the frontal network (that is, a simplified version of the “Towers of Hanoi” test) in strictly selected Parkinsonians failed to support neuropsychologically the specific frontal impairment in Parkinson’s disease. 5

Cerebral cysticercosis presenting with hemichorea

SIR: Your recent report of a patient with hemichorea due to metastatic carcinoma 1 prompts us to record a case of hemichorea which was also associated with focal lesions, in this case cerebral cysticercosis.

A 15 year old black girl presented in November 1982 with a two month history of involuntary movements of the left side of her body. The movements were gradual in onset and they were progressive. There was no recent or remote history of drug ingestion, oral contraceptives, exposure to toxins, head injury, sore throat or joint pains. She was admitted to a peripheral hospital in 1978 with severe headaches which subsided spontaneously. There was no family history of similar illness. On examination she was a mentally alert girl with left-sided facial distortions producing half smiles and half grimaces, and purposeless non-stereotyped jerks of the distal parts of her left arm and leg. Superimposed on these was intermittent flinging of the whole arm and leg. The tendon reflexes were slightly brisker on the left side but the plantar reflexes were flexor on both sides. Physical examination was otherwise normal.

The following investigations gave normal results: chest and skull radiographs, ESR, thyroid function tests, anti-streptolysin O titre, serum copper and caeruloplasmin levels, VDRL, auto-antibody screen and pregnancy test. The absolute eosinophil count in the peripheral blood was 770/mm 3. The cysticercus haemaglutination test and the fluorescent antibody test were positive in the blood in titres of 1:640 and 1:10 respectively. The corresponding titres in the CSF were 1:16 and 1:1. There were no eosinophils in the CSF. EEG showed some excess of polymorphic slow activity bilaterally. The CT scan showed numerous cysts, with calcification and central or peripheral contrast enhancement, in both hemispheres (fig). The appearance is characteristic of parenchymatous cysticercosis in the late active stage. Of particular interest, in this clinical setting, were several cysts in the basal ganglia of the right hemisphere. One was situated in the head of the caudate nucleus, encroaching on the anterior horn. At least three more were situated in the region of the anterior limb of the internal capsule and the lenticular nucleus.

The patient was treated with haloperidol.

There was some improvement in her involuntary movements. She had presented when the value of praiziquantel in treating cerebral cysticercosis was not yet established. We have since lost contact with the patient.

There can be little doubt that the patient’s neurological illness was the result of cerebral cysticercosis. While the lesions were diffuse and multiple, her left hemichorea could be explained by the cysts in, and adjacent to, the head of the right caudate nucleus. Some cases of chorea have been attributed to the neoplastic 1 and vascular 2 lesions in the contralateral caudate nucleus, putamen and other basal ganglia. Although cerebral cysticercosis has very protean manifestations, reviews of the field, 3–4 and a literature search retrospective to 1966, have yielded no reference to it as a cause of chorea. Greater availability of CT scanning may reveal this association more frequently in the Third World, where cerebral cysticercosis is a major cause of neurological illness.

References


References

Glycolytic enzymes in the CSF as tumour markers

Sir: In agreement with Twijnstra and colleagues 1 I find measurements of glycolytic enzymes in cerebrospinal fluid (CSF) such as lactate dehydrogenase (LDH) or phosphoglucoisomerase (PHI) easy to perform, readily available and economical. A drawback in the detection of meningeal metastases is their limited specificity. The following suggestions may help to reach a greater specificity.

Although the authors have considered the possible influence of age and sex on enzyme activity in the CSF, they have apparently not taken into account the state of the blood brain barrier (BBB) and the enzyme levels in the blood. In evaluating plasma protein concentrations in CSF such as IgG, it is common practice nowadays to correct the CSF value for the serum derived fraction. A similar approach was recently undertaken for the estimation of another tumour marker, carinoembryonic antigen in CSF. 2

CSF/serum ratios range from 1/230 for albumin to 1/500 for IgG. The permeability of the BBB for these plasma proteins is governed by their molecular radii. 3 By analogy, for LDH, an enzyme with 140 000 D molecular weight, one would expect a quotient of about 1/360. With a serum value of 240 U/l (the upper limit of normal with standardised methods) one then arrives at 240/360 = 0-7 U/l for the LDH activity in the CSF. However, the normal value given by Twijnstra et al 1 lies at 10 U/l. Therefore, in great contrast to plasma proteins, only approximately 7% of the LDH activity in CSF originates from the blood under normal conditions. Accordingly corrections for serum LDH may appear unnecessary. While this is probably true for controls with normal LDH and intact BBB, it is different for cancer patients. Meningeal affection often causes considerable impairment of the BBB and in the presence of systemic metastases LDH serum activity may be elevated several fold. Errotnously high CSF values may result especially when both conditions coincide. For PHI a correction formula analogous to Tourtellotte's calculation of the IgG synthesis rate has been proposed. 4

It would be interesting to know if the three control persons with pathological CSF values above 26 U/l in Twijnstra's patients 1 displayed elevated serum LDH and/or BBB disturbances. Likewise the observed augmentation of CSF LDH in older subjects may be connected to the greater permeability of the BBB for people aged over 60 years. In addition, simultaneous measurement of CSF and serum enzyme activities often reveal a beneficial effect of the CNS-directed therapy, when at the same time the systemic cancer cannot be controlled. 3

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References


Shoulder abduction fatiguability

Sir: I read with considerable interest the recent article by Nicklin et al entitled Shoulder abduction fatiguability. 1 Having previously read and admired the work of two of the authors, I was encouraged to observe that their work continues in the application of hand-held dynamometry to assess the neurological patient. I have, nevertheless, several concerns with what the authors have recently presented in this journal.

My chief concern is their apparent failure to take into account the influence of gravity during testing. By either testing shoulder abduction against gravity and not correcting for its influence or mixing the results of tests performed against gravity (in sitting) with those of tests performed with gravity eliminated (when supine) a potential source of error was introduced. Winter et al calculated that a failure to correct for gravity effects resulted in an absolute percentage difference of 2.4% (range -6.5 to 26.0) for knee extension. 2 The potential error associated with a failure to take gravity into account can be illustrated as follows: Suppose a subject's arm places 15 Newtons of force on a dynamometer, at its point of application. That is, with the arm abducted to 90° and the elbow flexed to 90° but resting on the dyna- mometer, a force of 15 Newtons is registered because of gravity. Next, suppose that the seated subject generates 140 Newtons of abduction force when tested as suggested by the authors. A 6.0% decline in this force over a series of 10 contractions would be 8.4 Newtons. This value, 8.4 Newtons, is only 54% of 155 Newtons, the actual force produced (140 Newtons + 15 Newtons to hold the arm against gravity). Thus, the fatigue index is decreased by (6.0 – 5.4)/6.0 = 0.1, or simply by including the weight of the arm. Now let us assume that the same subject is affected with a disorder that renders her weak. Gravity still results in 15 Newtons of force from the arm. The subject now generates 30 Newtons of force while seated. A 6.0% decline in this force equals 1.8 Newtons, which is 40% of 45 Newtons (30 Newtons + 15 Newtons to hold the arm against gravity). Thus, the fatigue index is decreased by (6.0 – 4.0)/6.0 = 0.33 or 33.3% by including the weight of the arm. Granting that this is a highly hypothetical situation, the result showing a failure to correct for gravity, particularly in a weak arm, could be quite serious.

My second concern is with the muscle group selected by the authors for their study. Although good reasons probably exist for the authors' choice of the shoulder abductors, these muscles are probably more difficult than some others to test accurately. In a study in which I tested supine subjects, I found that forces obtained during repeated tests of shoulder abduction, unlike forces obtained during most other actions, differed significantly from one another. 3 I have observed that subjects tend to flex the trunk toward the contralateral side during shoulder abduction testing. This tendency, which is particularly apparent when subjects are tested in sitting, magnifies actual force production. Even the subject in the authors' figure 1 seems to be flexed to the right during testing.