response to repetitive nerve stimulation.4,9
The same behaviour is described in non-
progressive neuropathies that show
increased fibre density and unstable com-
plex in the earlier phases of reinervation;
however, stable neuromuscular transmis-
sion is observed after some months of
evolution.12,13

Single fibre EMG study in hemiparetics,
not previously referred to in the literature,
provides new information on the electro-
physiological features of this condition.
The early increment of the motor unit fibre
density is in accord with other elec-
trophysiological findings,39 and supports
the possible collateral reinervation sug-
gested by biopsy studies.15,23 The archi-
tecture of the motor unit is altered in hemiplegia.

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Listeria monocytogenes abscess in the
basal ganglia

Sir: Meningitis due to Listeria mono-
cytogenes occurs in healthy people as well
as in patients with preexisting disease. Lis-
teria brainstem encephalitis1 and two cases
of brainstem and spinal cord abscess have
been reported in otherwise healthy
people.2-3 Brain abscess by Listeria mono-
cytogenes is a rare occurrence. Five cases
have been published to our knowledge.4-8
A sixth patient is presented.

A 45-year-old left handed man received
a cadaveric renal transplant at the age of 39
yr. The cause of his renal insufficiency was
chronic poststreptococcal glomeruloneph-
ritis. Rejection was suppressed by 25 mg
azothioprine and 15 mg prednisolone daily.
The transplant functioned satisfactorily.
After two days of headache, drowsiness
and weakness of the left arm he was admit-
ted with a temperature of 39.1°C, but no
signs of meningitis. Neurologic examina-
tion showed mild hemiparesis and a left
extensor plantar response. ESR was 80
mm/h; haemoglobin 6-4 mmol/l (10-3 g/l);
leucocyte count normal. The CSF con-
tained 68 polymorphonuclear leukocytes/1
mm³, protein was 0-69 gram/l, and CSF cul-
tures remained sterile. Blood cultures
yielded Listeria monocytogenes. CT scan
on the day of admission showed a hypodense
area in the region of the right basal ganglia
with a small central ring (fig A). Initial treat-
ment consisted of iv ampicillin (12 g/24 hr)
and 10 mg daily by intrathecal injection; azothioprine was
continued. In spite of bactericidal levels of the
antibiotic in the CSF, the patient deterior-
ated. Subsequent CT scans showed an
enlarging ring after contrast enhancement
(fig B and C). On the 31st day a drain was
introduced via a right parietal burrhole. No
pus could be aspirated. Postoperatively
the hemiparesis became worse, and CT scan
showed an unchanged abscess cavity. The
azothioprine was discontinued. On the
44th day a drain was inserted through a
right frontal burrhole and aimed with a
stereotactic device. One ml of turbid fluid
was aspirated, which on culture remained
sterile. CT scan demonstrates the tip of the
drain lying in or near the third ventricle.
After the operation high fever and marked
nuchal rigidity developed. The CSF cell
count was 5000/mm³ with an elevated pro-
tein content but culture yielded no growth.
Within six days the CSF became clear and
nuchal rigidity disappeared. The condition
of the patient then improved, and 10 weeks
after admission the antibiotic treatment
was reduced.
was terminated. On discharge he could walk without support, with circumduction of the left leg. CT scan after discharge showed a small contrast enhancing nodule (fig D). After one year the patient was only slightly hindered by a minimal residual hemiparesis.

Listeria monocytogenes is a facultative intracellular parasite. Defence depends mainly on cell-mediated immunity. Tripathy et al. studied the effect of cytotoxic drugs on the defence against such infection in mice. Azathioprine, given as a single dose at the time of infection suppressed the immune response. The occurrence of Listeria monocytogenes infections during corticosteroid therapy and in patients with cancer, has been reported, and central nervous system infections have been observed after kidney and heart transplantations. Four out of six patients with brain abscess due to such infection (including the present case) were renal transplant recipients. Two patients died and four survived the infection. The two fatal cases were treated with antibiotics only, as was one of the surviving patients, who was treated for six months. The present case demonstrated enlargement of the lesions in spite of early antibiotic treatment. Successful non-surgical treatment of early detected focal brain infection has been reported several times. The failure of antibiotics alone in our patient probably was related to his altered immune status. The surgical treatment in this case deserves comment. Needle aspiration instead of excision was chosen because of the risks of the latter method in basal ganglia abscesses. A rather unconventional approach, by means of a stereotactic apparatus, was tried the second time. This caused perforation of the abscess and drainage into the ventricles, a well-known complication in the treatment of basal ganglia abscesses. Drainage of the abscess into the ventricular system seems to have cured the patient, possibly by exposure of the abscess contents to antibiotics in the CSF. The more accurate positioning of the drain by means of a stereotactic device or preoperative computed tomography is probably an improvement in the treatment of centrally localised brain abscesses.

Dr RM van der Heide (Department of Infectious Diseases), REH van Acker and S Surachno co-treated the patient.

References


Matters arising

Viruses and Alzheimer’s disease

Sir: I was interested in the letter from Drs Mann et al describing the results of their search for herpes simplex virus antigen in samples of brain from cases of Alzheimer’s disease and Parkinson’s disease. Although they place more emphasis on the negative results in 40 cases, I found their positive result in the biopsy specimen from one case of Alzheimer’s disease of considerable interest. Disappointingly they do not illustrate their positive staining and they do not state whether prior absorption of antibody with the virus abolished staining on an adjacent section.

In the course of a study on the distribution of herpes simplex virus antigen in cases of herpes simplex encephalitis using the immunoperoxidase technique my attention was drawn to a particularly heavy involvement of the granule cells of the dentate fascia of the hippocampus. I therefore examined this layer of cells in a series of control cases among which were cases of Alzheimer’s disease. Sections were taken from a block from approximately the mid point of one hippocampus from 12 patients with clinical and pathological features of Alzheimer’s disease, and a small focus of positive staining for herpes simplex virus was found, involving three contiguous cells within the granule cell layer of the dentate fascia (fig). The positive staining was present in the cytoplasm and not in the nuclei of these cells. Staining of an adjacent section with antibody preabsorbed with herpes simplex virus gave negative results, but this could conceivably have been due to absence of the same cells within this section. Other cases of Alzheimer’s disease and all the other control cases examined gave negative results. This observation, taken together with other preliminary observations such as that of Mann et al, the report of Nagington et al of a case of herpes simplex encephalitis from which herpes simplex virus was only isolated after temporal lobe tissue had been co-cultivated for 18 days (suggesting the presence of latent infection), and the finding of herpes simplex virus genome in brain tissue from some patients with Alzheimer’s disease, multiple sclerosis and normal subjects, warrants further investigation. My own observations on the distribution of herpes simplex virus within the brain in herpes simplex encephalitis suggested that the infection had arisen within one cerebral hemisphere, possibly at a site related to the central connections of the olfactory system.

A possible hypothesis consistent with these observations is that herpes simplex virus produces a latent (or at least incomplete) infection of the olfactory bulbs, of more central connections of the olfactory tracts, or of the granule cells of the dentate fascia of the hippocampus (where the unusually close proximity of the neuron cell bodies may play a role). At this site, in the majority of those infected no pathological consequences ensue except that in later life the metabolism of neurons closely connected with these cells is altered and neurofibrillary tangles develop. On very rare occasions in persons with such an infection, perhaps triggered by some unidentified event, whole virus is again produced and overt infection results with development of acute encephalitis. Also rarely in the presenile age group, but more commonly in the senile age group, the latent infection generates metabolic alterations leading to neurofibrillary tangle formation not only in the hippocampus, but in a more widespread distribution in the neocortex, giving rise to Alzheimer’s disease.

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References


Mann et al reply:

In response to Dr Esiri’s letter we illustrate (figure) the area of positive immunoperoxidase reaction product, described in our earlier letter, in the case of a 59-year-old woman with Alzheimer’s disease...