

Figure Photomicrographs showing immunostainings for chromogranin A (left upper, 1320 \times), prolactin (right upper, 1320 \times), growth hormone (left lower, 1320 \times) and ultrastructural detail of secretory granules (right lower, 36.600 \times).

10–25% of the tumour cells contained prolactin and it is possible that the patient's impotence was due to hyperprolactinaemia. The combination of apparently benign histological features followed by seeding through the CSF after 13 years has been described before.⁵ The tumour tissue deposits occurred in sites at which the radiotherapy dose was low or to which radiotherapy was not given. This suggests that a pituitary adenoma with potential to metastasise may respond to radiotherapy. Dissemination of tumour during surgery cannot be excluded as a cause of arachnoid seeding but such seeding has been observed in patients who have not previously had operation.

We conclude that the surgeon as well as the pathologist should be aware that "ectopic" suprasellar adenohypophyseal cells and adenomas occur in the supra- and parasellar region. The diagnosis can be made by the application of immunohistochemistry and electronmicroscopy to biopsy material. This tumour may be hormonally active so that

endocrinological studies should be performed before operation.

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Tumour necrosis factor- α in malignant melanomatous meningitis

Meningeal infiltration by neoplastic cells is an ominous prognostic sign in patients suffering from systemic cancer. After carcinoma of the breast and bronchus, malignant melanoma is the third most common primary tumour in patients with diffuse leptomeningeal metastasis.¹

Intrathecal synthesis of IgG and detection of oligoclonal immunoglobulin bands on isoelectric focussing gels of CSF in meningeal carcinomatosis, suggest immune recognition of tumour cells within the CNS.² We determined the levels of two cytokines, tumour necrosis factor- α (TNF α) and interferon gamma (IFN γ), in paired serum and CSF samples obtained from 45 patients with meningeal malignancies. Cytokines, for example, interleukins, interferons, and TNF α , are multifunctional messenger molecules currently evaluated for new approaches of im-

muno-therapy in disseminated malignancies.

Our study included CSF and serum samples, stored at -70°C after lumbar puncture and centrifugation without further processing, from patients with diffuse leptomeningeal metastasis from cancer of the breast (14), bronchus (7), ovary, cervix, prostate, kidney, stomach (1 each), and unknown origin (4), malignant melanoma (4), non-Hodgkin lymphoma (9), multiple myeloma (1), and Hodgkin's disease (1). Commercial ELISA kits were purchased from British Biotechnology, Oxford, UK, (TNF α), and Endogen, Boston, USA (IFN γ). Sensitivity was 50 ng/l for IFN γ and 40 ng/l for TNF α .

IFN γ was found in the CSF in carcinomatosis from two cases of breast cancer (322 and 899 ng/l), one case of cancer of unknown origin (639 ng/l), and one case of non-Hodgkin lymphoma (66 ng/l). IFN γ in serum was positive in two cases of cancer of the breast (56 and 901 ng/l); neither of which had detectable IFN γ in the CSF, and one case

of cancer of the bronchus (526 ng/l). IFN γ was also present in the serum of the patient with non-Hodgkin lymphoma (48 ng/l) who had IFN γ in the CSF.

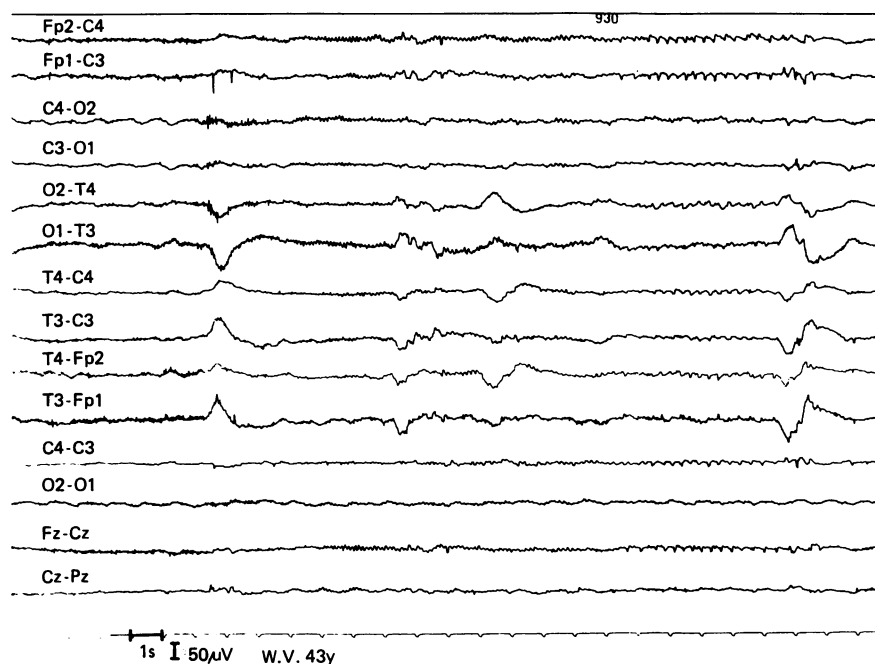
TNF α was detected in three of four CSF samples but not in the serum of patients with meningeal infiltration from malignant melanoma (61, 78, and 166 ng/l) or in CSF in other neoplastic diseases. TNF α was, however, detected in sera from three patients with meningeal carcinomatosis (breast, 534 ng/l; bronchus, 46 ng/l; unknown origin, 118 ng/l) and one patient with non-Hodgkin lymphoma (48 ng/l). None of the CSF or serum samples contained both IFN γ and TNF α .

TNF α may mediate inflammation and tissue destruction in bacterial meningitis, particularly severe meningococcal disease,³ inflammatory demyelination,⁴ and tumour cell cytotoxicity⁵ in vitro.

To our knowledge, this is the first report on TNF α in meningeal malignancies. New approaches to an immunotherapy of malignant melanoma are evolving rapidly, and are based on specific immunogenic features of this malignancy, for example, inhibition of tumour cell proliferation in vitro by IFN γ and TNF α .⁶ Although elevated levels of TNF α in the CSF of patients with malignant melanomatous meningitis are still a preliminary finding, the lack of similar results in a large control group of other meningeal malignancies confirms the existence of special interactions between melanoma cells and the host's immune system. This warrants further investigation.

Partial self induced seizures: an uncommon motivation for auto-induction

Some epileptic patients may self induce their seizures. In most instances, these are people affected by photosensitive primary generalised epilepsy, in whom self induction is by intermittent photic stimulation (IPS) or, more rarely, pattern stimulation or eye closure. Documented reports have increased recently due to the availability of IPS from



Spontaneous seizure occurring only with subjective symptoms (somatognosic illusion affecting left upper arm), accompanied by rhythmic, low voltage discharge, most prominent on right frontal or fronto-central areas.

various sources of artificial light (TV, video games). In these cases, patients generally explain self induction with vague motivations and most authors consider auto-induction as a compulsive pleasure seeking behaviour.

Conversely, reports of partial self induced seizures are extremely rare.¹⁻⁴ We have recently observed a patient with spontaneous and self induced simple partial seizures. He was a 43 year old, right handed man without familial neurological disorders and with unremarkable personal history apart from the sporadic occurrence of depressive episodes. At the age of 36 he was involved in a car accident during which he experienced a minor head injury with a brief loss of consciousness. After a few months he began to suffer from ictal episodes, generally diurnal and often occurring many times a day. These attacks began with a subjective sensation which the patient described as an altered perception of the left arm ("as if my arm changed its size, or moved, or were crossed by waves"); this sensation was often followed by a violent adduction of the legs, a rapid versive movement of the head and trunk towards the left side, and, successively, rhythmic clonic jerks of the left eyelid. The whole episode occurred with preserved consciousness and lasted about 30 seconds. These episodes were interpreted as psychogenic and treated with benzodiazepines with poor results. The patient discovered accidentally that he was able to induce these attacks by rubbing his left eye. As physicians were generally puzzled by the description of the episodes, he began to provoke his seizures during medical examinations, to prove the truth of his ailments.

Neurological and funduscopic examination and brain CT scan with contrast enhancement were normal. Basal EEG showed diffuse, low voltage fast activity, without interictal paroxysmal activity. During the recording the patient experienced a spontaneous ictal episode, limited to subjective abnormal sensation in his left arm. The last part of the attack was accompanied by a rhythmic, low voltage discharge, progressively decreasing in

frequency (from 9–10 to 3 Hz) and increasing in amplitude, most prominent on the right frontal or fronto-central areas (figure). During a further polygraphic recording, as no spontaneous episodes were observed, the patient was asked to provoke an attack. He rubbed his left eye and, after about 10 seconds, experienced the abnormal sensation, followed by a sudden diffuse muscle contraction with adduction of the legs and by clonic movements of the left eyelid. In the last part of the episode a focal rhythmic discharge, having the same frequency of the eyelid jerks, was seen on the right frontal region. He was treated with carbamazepine (CBZ) at a dose of 10 mg/kg/day. Both spontaneous and self induced episodes disappeared in a short time. One year later the patient was still free of seizures.

The site of origin of the seizures was difficult to localise, as neuroradiological evaluation was normal and examination with special electrodes was not available. The early ictal semeiology (somatognosic illusion), however, suggested a right parietal or temporo-parietal origin,⁵ while the unilateral eyelid jerks implied a successive spreading of the discharge to a limited area of pre-rolandic cortex. In all documented cases of self induced partial seizures patients induced their seizures because of the resulting pleasant experience ("hedonistic motivation"). In the case reported by Van Reeth¹ the seizures were accompanied by an extremely enjoyable sensation; the patient was able to induce the attacks by means of a casually discovered mechanism (inhaling rapidly and deeply while smoking a cigarette) and considered them as a pleasant diversion. The patient reported by Spadetta and Giachheddu² provoked his seizures by means of apnoea or Valsalva manoeuvre; the reason given for inducing the attacks was the intense feeling of pleasure which accompanied them ("as if I were in heaven"). Jacome *et al*³ reported a patient with gelastic seizures who was able to precipitate his seizures by rapidly hyperextending his trunk and neck, and

provoked them because of the intense sexual pleasure which he experienced before losing consciousness. The patient described by Klass⁴ self induced her pleasant trance-like states, followed by the stereotypical vision of a dancing couple by playing records of music and concentrating intensely. In our case, conversely, the seizure was not experienced as a pleasant episode, and the reason for self induction indicates a peculiar utilitarian motivation: the patient, who casually discovered the possibility of provoking his seizures, used the triggering manoeuvre to persuade the sceptical physicians of the truthfulness of his disturbances.

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F-response during cataplexy

Cataplexy is one of the cardinal symptoms of the narcolepsy-cataplexy syndrome and is characterised by brief, sudden episodes of muscle weakness without loss of consciousness. Although the H-reflex is known to be abolished or clearly diminished during cataplexy,¹ the pathophysiology of the atonia is not well studied. We report the results of the continuously recorded F-responses before, during, and after cataplexy and sleep attack.

The patient was a 77 year old woman who had been suffering from daytime sleep attack, cataplexy, sleep paralysis, and sleep hallucination since the age of 15. The cataplexy lasted seconds or minutes and was often triggered by a sudden emotional surge, such as excitement and surprise. During cataplexy, all limbs were flaccidly paralysed and the deep tendon jerks could not be elicited. Standard polysomnographic recordings showed sleep onset REM sleep, increased REM sleep, and frequent sleep apnoea. The results of nerve conduction study of the right tibial and sural nerves, brainstem auditory evoked potential, and brain computed tomography were normal. The H-reflex was not obtained from the soleus muscle by the electrical stimulation of the tibial nerve. The symptoms were clearly improved by oral administration of imipramine 90 mg per day.

For the recording of polygraphy during cataplexy and sleep attack, the relaxed patient lay on a couch in a semidark, warm and quiet room. In addition to electroencephalography (EEG), electro-oculography (EOG), and submental electromyography, F-response was continuously recorded from the right flexor hallucis brevis muscle by the percutan-



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