

25 Years Ago in the Canadian Journal of Neurological Sciences

AQUEDUCT GLIOSIS CAUSED BY KERATIN AND CHOLESTEROL IN A CASE OF CRANIOPHARYNGIOMA

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Robert Gamble, Arie L. Liebeskind

SUMMARY: Keratin and cholesterol particles released by a craniopharyngioma were shown to be the agents involved in the production of a foreign-body granulomatous arachnoiditis and ependymitis. Some of the particles were seen embedded in the reactive subependymal glial tissue along the aqueduct of Sylvius. It was thought they were a major factor leading to the aqueductal stenosis. However, we have also considered that the change in cerebrospinal fluid hydrodynamics following the ventriculopleural shunt contributed to the occlusion of the aqueduct.

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THE ROLE OF HUMORAL MEDIATORS IN MIGRAINE HEADACHE

A. Fanchamps

SUMMARY: The classical vascular mechanism of migraine attacks demonstrated by Wolff - intracranial vasoconstriction during the prodromal stage and passive vasodilatation of extracranial arteries during the painful phase - has been confirmed by modern methods. Arterial distension is, however, not sufficient to explain the origin of the pain: to give rise to an acute migraineous pain, it must be associated with a lowered pain threshold of the receptors situated in the wall of the affected vessels. A number of humoral factors - plasmakinin, serotonin, histamine - intervene in the chain of events that culminates in migraine headache.

At the start of the attack, the blood platelets release serotonin, the mast cells in the affected area release histamine and proteolytic enzymes that split plasmakininogens to form plasmakinins. Free serotonin and histamine increase capillary permeability and favor transudation of plasmakinin into the vessel walls and perivascular tissues. The combined effect of serotonin and plasmakinin on the vessel wall receptors reduces their pain threshold. On the other hand, the bulk of the released serotonin is excreted as 5-HIAA, and plasma serotonin falls. Since serotonin has a constricting effect on the extracranial arteries and a dilating one on the capillaries, the fall in its plasma level induces hypotonia of these arteries and capillary constriction, which results in a passive distension of the arterial walls. The two factors necessary for the production of pain are thus present: a low pain threshold and vascular distension.

In addition to these three main humoral mediators, the part played by tyramine in migraine of alimentary origin, the precipitating effect of estrogen decrease in menstrual migraine and the hypothetical role of prostaglandins and prolactin are discussed.

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CORNEAL REFLEX IN HEMISPHERE DISEASE II

R. T. Ross, J. A. Johnston

SUMMARY: In man, contralateral cerebral decortication or removal of a hemisphere does not permanently abolish the corneal reflex. Destruction of the contralateral thalamus or contralateral mesencephalic trigeminal tract does abolish the corneal reflex.

In the cat however, the corneal reflex remains intact after contralateral decortication, hemispherectomy and total ablation of the thalamus. Some of these procedures diminished the corneal reflex or increased the threshold, but neither of these changes were seen for more than a few days post-operatively. In this animal however, the second reflex response, evoked by supra orbital nerve stimulation, is abolished by certain thalamic lesions and by contralateral hemi-transection of the brain stem at various levels.

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A REVIEW OF ACRYLAMIDE NEUROTOXICITY PART II. EXPERIMENTAL ANIMAL NEUROTOXICITY AND PATHOLOGIC MECHANISMS

Peter S. Spencer, Herbert H. Schaumburg

SUMMARY: The companion paper of this review examined the chemical, biochemical and toxicologic properties of acrylamide and its analogues (Spencer and Schaumburg, 1974). Mention was made of the industrial preparation and uses of acrylamide, the hazard faced by industrial workers, and the clinicopathologic features of chronic human acrylamide intoxication.

The present paper summarizes current knowledge of the effects of acrylamide intoxication in laboratory animals. The major focus here is the pattern of nerve damage in experimental acrylamide neuropathy, since this has become the most powerful experimental tool with which to explore human nervous system dying-back disease. Adhering to this theme, the paper concludes with a discussion of the pathologic mechanisms which might be involved in producing the nervous system damage seen in acrylamide neuropathy.

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THE NEURAPRAXIC LESION A CLINICAL CONTRIBUTION TO THE STUDY OF TROPHIC MECHANISMS

A. J. McComas, P. B. Jorgensen, A. R. M. Upton

SUMMARY: Four patients with neurapraxic lesions are described; in spite of the absence of impulse activity in muscle fibers no other signs of denervation could be detected. These observations are interpreted as indirect evidence for the role of a non-impulsive (axoplasmic flow) system in certain trophic phenomena. The relationship of the present findings to the spectrum of neuropathic lesions is considered.

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TREATMENT OF SUBACUTE POLYNEURITIS WITH CORTICOSTEROIDS

M. J. Newman, N. Nelson

SUMMARY: A study of the course of 17 patients with subacute polyneuritis was undertaken. Ten were given corticosteroids. Assessment of time of turn-around, that is the beginning of clinical improvement, showed that some patients responded promptly to steroids whereas a few did not. A comparison of turnaround times between the treated and untreated cases shows a statistically significant effect of steroids in hastening the onset of recovery. This is not due to bias in selection of those for steroid treatment.

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OCCIPITAL BONE CYST CAUSING LOWER CRANIAL NERVE PALSIES

M. Banna, W. G. Bradley, R. M. Kalbag, B. E. Tomlinson

SUMMARY: A 20 year-old girl presented with neck and occipital pain for six weeks, which was found to be due to a unicameral bone cyst of the left occipital condylar region.

Although there have been very few reports of simple bone cysts in bones than the long tubular bones, it appears that the present case is probably the first report of such a lesion occurring in the skull.

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