Hyperventilation tetany: effect of carbamazepine

Schaaf and Payne first reported the use of diphenylhydantoin to treat tetany in 1966.1 We decided to try anticonvulsant therapy with carbamazepine in hyperventilation tetany. Carbamazepine was administered to 211 women and 77 men aged 18-42 (mean 26) in a dose of 600 mg daily for three months. Chvostek's sign was found in 70%, carpopedal spasms and paraesthesiae occurred in 28%, and non-specific features such as headache, fatigue, and anxiety in 72%.

Electromyography was performed on all patients after two minutes of hyperventilation and all showed repetitive double, triple, and multiple discharges typical of tetany. Carbamazepine significantly improved many of these features and Chvostek's sign disappeared in 31%.

A general reluctance to use anticonvulsant medication to treat hyperventilation tetany is probably due to the belief that tetany is due to ionic imbalance. Our results have shown that the stabilising effects of carbamazepine on neurons, probably implemented at the level of the brainstem reticular formation which is responsible for the integration of tetanic activity was the reason for the improvement. The effect on the limbic system was shown by the decrease in anxiety.

Our experience suggests that carbamazepine may be useful in the treatment of hyperventilation tetany.

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Reversible pituitary stalk enlargement in cranial diabetes insipidus

Infiltrative disease or neoplastic involvement of the pituitary stalk results in the CT appearance of stalk thickening often accompanied clinically by diabetes insipidus. Some patients, however, with cranial diabetes insipidus of no identifiable cause may also exhibit stalk enlargement.2 The natural history of this radiological abnormality in so-called idiopathic cranial diabetes insipidus has not been previously reported. We report on the follow-up of two patients with cranial diabetes insipidus whose CT findings at initial presentation revealed gross stalk enlargement.

CT scan showed gross thickening of the pituitary stalk in a 14 year old boy who presented three months after onset of symptoms. Water deprivation test with parallel assessment of thirst sensation confirmed the diagnosis of diabetes insipidus with concomitant adipsia. Treatment comprised water restriction (2 l/day) and intranasal desmopressin. Three years later a repeat CT scan showed no abnormality of the stalk. Serial CT and MRI over an eight year follow up have excluded a mass lesion.

In a second case a 24 year old female presented with a two week history of polyuria and polydipsia of sudden onset. Water deprivation testing confirmed cranial diabetes insipidus. Anterior pituitary function was intact. CT findings in this patient at initial presentation are shown in fig 1. The normal pituitary stalk (PS) is smaller than or equal to the size of the basilar artery (BA), but as illustrated in fig 2, was considerably enlarged in this patient.

MRI performed after three months of treatment with intranasal desmopressin confirmed that the stalk was still thickened. One year after presentation, however, repeat CT showed that the stalk was then of normal size (fig 3).

Structural involvement of the pituitary stalk in a number of pathological states such as histiocytosis, sarcoidosis, primary and secondary neoplasms may result in diabetes insipidus. Our patients with diabetes insipidus had gross stalk enlargement at presentation but resolution of the abnormality without treatment other than desmopressin in these cases confirms that stalk enlargement may occur in the absence of any progressive infiltrating or neoplastic process.

The MRI findings in case 2 (fig 2) three months after presentation are noteworthy. There was no abnormalities of the hypothalamus. Stalk thickening was still obvious, though less marked than at presentation suggesting a slowly resolving process. The fact that the lesion was still present at this time excludes the possibility of ischaemic infarction of the stalk since the effects of infarction
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