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LEADING ARTICLES

PAPERS AND ORIGINALS

PRELIMINARY COMMUNICATIONS

MEDICAL MEMORANDA

MIDDLE ARTICLES

BOOK REVIEWS 752

NEWS AND NOTES

CURRENT PRACTICE

CORRESPONDENCE 762

OBITUARY NOTICES 768

SUPPLEMENT

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Correspondence

Letters to the Editor should not exceed 500 words.

E.E.G. Signs of Death

SIR,—Your recent leading article (11 May, p. 318) draws attention to the need for reliable methods of assessing cerebral function in the apparently moribund patient and suggests that the role of the electroencephalogram in such assessment should be investigated as a matter of urgency. Dr. G. Pampligione's letter (1 June, p. 557), while endorsing this view, appears to obscure the central issue.

It is difficult to justify his proposal to establish an independent mobile E.E.G. unit. The research which so urgently needs to be carried out will probably require serial studies both of the E.E.G. and of other variables, and these can best be performed in a single centre. There is no shortage of moribund patients and it should be unnecessary to collect clinical material from a number of different hospitals. Until the necessary research has been completed no useful clinical service would be provided by an electroencephalographic flying squad offering ill-founded and largely speculative opinions in the middle of the night.

Your correspondent asserts that "the bibliography is rather more extensive than your leader implies." This is true to the extent that a large number of authors have reported that following acute cerebral anoxia the E.E.G. is often abnormal or unrecordable. However, the number of studies which propose, and seek to test, electroencephalographic criteria for predicting the outcome of such catastrophes are extremely few and none is included in the eight references cited by Dr. Pampiglione. Nevertheless, the poverty of the literature on this subject is emphasized by the fact that several of the sources which he quotes are of only marginal relevance to the E.E.G. diagnosis of death and that the majority are reports of verbal communications consisting largely of anecdote or of opinion and published in society proceedings. It will be unfortunate if electroencephalographers, by claiming knowledge which they do not yet possess, delay adequate investigation of this pressing problem.—I am, etc.,

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Hyperventilation in Children

SIR,—Your leading article (4 May, p. 255) calls for some comment. Most non-cardiorespiratory causes of hyperventilation in children are accompanied sooner or later by a significant disturbance in consciousness, which aids differentiation. In addition, the depth of ventilation is often increased disproportionately to the rate, which is a clue to a non-cardiorespiratory aetiology. Your statement, that in states of overbreathing due to salicylate poisoning and diabetic ketoacidosis "treatment may be urgently needed," is indeed an understatement. Children with these conditions who reach the stage of overbreathing are seriously ill, often with a significant disturbance of serum electrolytes and the acid–base state and must have urgent laboratory assessment and intensive treatment.

Salicylate poisoning in adults may present in the stage of respiratory alkalosis and pass gradually into metabolic acidosis. In children, however, the acidotic stage rapidly supervenes, and of more than 20 children seen with hyperventilation due to salicylism metabolic acidosis was always present on admission to hospital, sometimes only a few hours after ingestion of the drug.

The differentiation of the various cardiorespiratory causes of hyperventilation may be very difficult. Thus a child with salicylate poisoning might be dehydrated owing to hyperventilation, vomiting, and impaired fluid intake, and the urine may also reduce Benedict's solution and suggest the diagnosis of diabetic ketoacidosis. Glycosuria is of course not diagnostic of the diabetic state, but may be present in other causes of hyperventilation such as encephalopathies—for example, that due to lead intoxication and other intracranial disturbances such as haemorrhage. One must also remember the uncommon combination of diabetic ketoacidosis and renal insufficiency, when glycosuria may be absent, and it is possible for a diabetic child to have salicylate poisoning. Uraemia and septicaemia must also be considered in the differential diagnosis.

Therefore when one is confronted with a hyperventilating child, especially in the presence of an altered state of consciousness, the non-cardiorespiratory causes must be considered. Screening with Phenistix and Dextrostix may be helpful, but an urgent full laboratory assessment is essential, including blood sugar, serum salicylate, blood urea, serum electrolytes, acid-base state, blood *p*H and urinalysis, if these children are to be correctly diagnosed and treated.—I am, etc.,

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Antibiotic Cover in Dentistry

SIR,—In a most interesting paper Dr. R. J. Vecht and Dr. Celia M. Oakley (25 May, p. 455) reported three cases of endocarditis arising in patients suffering from hypertrophic obstructive cardiomyopathy, and they rightly drew attention to this risk and to the need to provide the usual antibiotic cover before dental and other manipulations of these patients.

Comment is required, however, on the inference that the onset of the disease was related to the performance of dental treatment in Case 1. It is not usually considered necessary to provide cover for routine conservative dentistry on patients at risk to subacute bacterial endocarditis, since this type of procedure does not cause a significant bacteraemia unless performed on infected teeth, which in such patients should be treated by extraction. Is it possible that the dental treatment mentioned included scaling or root canal therapy, both of which may cause bacteraemia and require prophylactic