Myocardial Infarction in Epileptics

SIR,—Patients on long-term phenytoin and/or phenobarbitone therapy have a high incidence of osteomalacia. They also have a lower than normal circulating concentration of vitamin D metabolites.[1 2]

In a recent paper (14 September 1974, p. 647) I reported a significant association between vitamin D consumption and the incidence of myocardial infarction. In later studies I have found an association between vitamin D intake and serum cholesterol levels.[3] On these grounds we might expect epileptics to have a lower than normal incidence of myocardial infarction. Experienced neurologists in Norway say that they have not seen a single chronic epileptic die of myocardial infarction. I have so far been unable to find any work concerning this in the literature. My object in writing this letter is to stimulate some reaction to this question or appropriate action on this problem.—I am, etc.,

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1 Stamp, T. C. B., *et al.*, *British Medical Journal*, 1972, **4**, 9.
2 Haussler, M. R., *Nutrition Reviews*, 1974, **32** 257.
3 Lindén, V., *Scandinavian Journal of Social Medicine*, in press.

Developmental Screening and Assessment

SIR,—Three excellent papers within the past few months[1-3] have demonstrated not only the feasibility but also the desirability of monitoring the development of children in general practice. Many specialist paediatricians, however, are highly cynical about the ability of general practitioners to perform this function and some community physicians are of the same view, perhaps feeling that one of their functions is being usurped. I think that both are labouring under an illusion and this is partly due to a confusion of terms. General practitioners have talked too often about developmental *assessment* when they meant developmental *screening*. I have myself been guilty of this error.[4] We must from now on have a clear definition of our terms.

Assessment occurs at assessment centres, places like the Wolfson Institute, the Newcomen Clinic, and others, where expert developmental paediatricians, psychologists, otologists, etc., can investigate a child in depth in a session which may last a day or a whole week in order to arrive at a definitive diagnosis where possible and to formulate a plan of management for a handicapped child.

Developmental screening occurs in well-baby clinics in general practice or in the old local authority clinics, attempts to identify at the earliest possible stage those children who should go forward for assessment. It is a technique that is in the process of maturation and there are many varieties of it. The tests must be quick yet accurate, with the lowest possible rate of false negatives; they must be acceptable to child, parent, and clinician; they must involve the minimum of specialized equipment, and there are many other criteria they must satisfy. But they are intended to identify delay in development, and not make a definitive diagnosis (though this will often be possible). The great advantage which the clinician doing developmental screening has is that, seeing so many normal children and understanding the range of normality, the abnormal is usually glaringly obvious. This is the antithesis of the view generally held by specialists who claim that because one seldom sees the abnormal it will be missed when it appears; those doing developmental screening know that the opposite is the case.

Let us therefore work together on this and not against each other, for there is plenty of work for us all to do. When the screening is done properly the assessment centres will be far busier than they now are. It will be many years before developmental screening becomes a routine part of primary paediatric care, though I am certain that one day this will be the case, and in the meantime there is the need of a greater number of clinic doctors willing and able to undertake this work.—I am, etc.,

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1 Starte, G. D., *Practitioner*, 1974, **213**, 823.
2 Bain, D. J. G., *Health Bulletin*, 1974, **32**, 189.
3 Rowlands, P., *Update*, 1975, **10**, 379.
4 Hooper, P. D., and Alexander, E. I., *Practitioner*, 1971, **207**, 371.

Deoxyuridine Suppression Test

SIR,—In a recent study (20 July 1974, p. 148) the deoxyuridine (dU) suppression tests[1 2] were performed on 110 marrow samples aspirated at our hospital over a period of 12 months and it was found that this test provided a simple, rapid, and useful method of diagnosing vitamin B_{12} or folate deficiency. It was also found that the addition of 50ng-1μg pteroylglutamic acid (PGA) per ml of marrow culture (together with the dU) partially corrected the abnormal suppression shown by two folate-deficient patients but did not correct the abnormal suppression shown by three vitamin B_{12}-deficient patients. The latter observation appeared to provide a new method of using the dU suppression test to distinguish between vitamin B_{12} and folate deficiency. Subsequent experience with marrow aspirates from 12 other folate-deficient patients (with haemoglobin levels between 5·7 and 14·1 g/dl) has shown that the addition of 50ng-1μg PGA per ml of marrow culture corrects the abnormal dU suppression only in moderately or severely anaemic folate-deficient patients. The minimum concentration of PGA which partially corrects the abnormal dU suppression in all folate-deficient patients appears to be 10 μg per ml of marrow culture. This concentration of PGA has no significant effect on the dU-suppressed values in all normoblastic and some vitamin B_{12}-deficient marrow cultures.

It has been previously shown[2 3] that the addition of 1 μg of cyanocobalamin per ml of marrow culture partially corrects the abnormal dU suppression shown by vitamin B_{12}-deficient marrows but does not correct the abnormal suppression shown by folate-deficient marrows. Over the past 12 months we have successfully distinguished between vitamin B_{12} and folate deficiency by studying both the effect of adding 1 μg of cyanocobalamin and the effect of adding 10 μg of PGA per ml of marrow culture on the dU-suppressed value. Examples of results obtained in folate- and vitamin B_{12}-deficient patients are given in the table. The only major problem encountered with this test has been the uncertainty as to whether a dU-suppressed value which is partially corrected by both cyanocobalamin and PGA necessarily indicates the existence of depleted folate stores in addition to a deficiency of vitamin B_{12}.

We have found the dU supression test of particular value in diagnosing folate deficiency in patients in whom the red cell folate level was within the normal range. For instance, the dU supression test demonstrated the presence of a secondary folate deficiency in three patients with megaloblastic haemopoiesis and normal serum vitamin B_{12} and red cell folate levels. One of these patients suffered from a severe dapsone-induced haemolytic anaemia, another from hereditary spherocytosis, and the third from disseminated tuberculosis complicating acute myeloid leukaemia. In all three patients the dU-suppressed value returned to normal and haemopoiesis became normoblastic after therapy with 5 mg folic acid daily for three days.—We are, etc.,

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1 Metz, J., *et al.*, *British Journal of Haematology*, 1968, **14**, 575.
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Doubts about Lignocaine

SIR,—Lignocaine has been so fashionable that other approaches to the prevention of ventricular ectopic activity in acute myocardial infarction have been inhibited in recent years. Now that doubts about the worth of this popular remedy have been made public through your leading article (1 March, p. 473), it may become easier to conduct clinical trials of other routes.

Case No.	Haemoglobin (g/dl)	Mean Corpuscular Volume (fl)	dU-Suppressed Values* (%)			Conclusion
			Without added B_{12} or PGA	With 10μg PGA/ml†	With 1μg B_{12}/ml	
1	6·8	132	26·4	11·5	30·0	Folate-deficient
2	5·7	106	59·6	23·3	53·8	
3	9·6	105	27·6	15·6	26·3	
4	12·0	102	11·5	6·9	10·9	
5	15·1	98	20·6	11·1	19·0	Vitamin B_{12}-deficient
6	13·4	109	22·1	21·1	16·0	
7	13·3	104	17·4	17·4	13·0	

*Normal range = 5·37 ± 3·88 (2s.d.)%
†The dU-suppressed value is reduced by at least 20% in all folate-deficient patients
Conversion: SI to Traditional Units—Mean corpuscular volume: 1 fl = 1 μm^3