BRITISH MEDICAL JOURNAL

SATURDAY 23 MARCH 1968

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No. 5594 Brit. med. J., 1968, Vol. 1. 719–780 Weekly. Price 5s.
BRITISH MEDICAL ASSOCIATION, TAVISTOCK SQUARE, LONDON W.C.1 TEL: 01 – 387 4499

Correspondence

Letters to the Editor should not exceed 500 words.

Vaccination Against Measles

SIR,—Your leading article on measles vaccine (17 February, p. 395) points out that the effectiveness of vaccination before nine months of age is likely to be inhibited by the presence of maternal antibody. This maternal antibody is a very potent force in protecting the infant against natural infection in the first nine months of life.

Has anyone considered whether the antibody acquired by the active immunization of the next generation of mothers will be carried over in sufficient quantity to protect their infants in the first nine months? Or must we face a complete break in this normal protection mechanism? The protection afforded to the school child by measles vaccine may only modify his natural attack, which, in turn, is just as infectious to his young unprotected sibling, who will be doubly at risk because of the difficulty in recognizing measles in the modified case. (This situation frequently occurs in pertussis at the present time.)

Can anyone tell me whether this problem has been worked out or is it just awaiting the next generation and their doctors?—I am, etc.,

R. M. FORRESTER.

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Royal Albert Edward Infirmary,
Wigan.

Congenital Heart Disease

SIR,—Your leading article (9 March, p. 595) is disturbing, for in my opinion it gives a dangerously misleading impression regarding the need for investigation in patients with the Eisenmenger syndrome. The syndrome can usually be diagnosed in the clinic or at the bedside, and it is often possible from the history and physical signs to differentiate atrial septal defect from ventricular septal defect or persistent ductus arteriosus in the presence of this syndrome. The helpful work of Rees and Jefferson,[1] pointing out the features in the plain chest radiograph by which these defects may be differentiated one from another, adds useful collateral evidence.

The differential anatomical diagnosis is, however, of academic interest only, for at the present time these patients are beyond the reach of surgery. Operation is contraindicated. To suggest that "cardiac catheterization and angiocardiography are therefore usually required for final diagnosis" (of the anatomy associated with the Eisenmenger syndrome) is to suggest that every such patient must be subjected to an investigation which is by no means without risk in this condition and which offers these patients no therapeutic opportunity.

A firm diagnosis of the Eisenmenger syndrome, made by the cardiologist from the clinical findings together with plain *x*-ray appearances, is at the present time sufficient. If the presence of the Eisenmenger syndrome is in doubt, then, of course, special investigation must be undertaken.—I am, etc.,

A. M. JOHNSON.

Southampton Chest Hospital,
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REFERENCE

1 Rees, R. S. O., and Jefferson, K. E., *Clin. Radiol.*, 1967, **18**, 366.

Motivation of Medical Scientists

SIR,—You quote at some length on the topic of motivation of medical scientists from the preamble to the 48th Annual Report of the Walter and Eliza Hall Institute of Medical Research (20 January, p. 132). One of the factors examined, explained, defended, and approved was the desire to win the approbation of one's peers. This laudable competitiveness is of course not confined to medical scientists, but is a fundamental driving force in virtually all fields of human endeavour, including professional, sporting, artistic, and social. It is simply another aspect of what is popularly known as "trying to keep up with the Joneses." This impulsion must have arisen very early in vertebrate evolution, and originally possibly served a sexual purpose as in the plumose display of the male bird before the female during the mating season.

It is perhaps an oblique commentary on the Welfare State that you should hold up as a new discovery what must be one of the mainsprings of human action. More than 300 years ago Milton expressed the urge very neatly:

> "Fame is the spur that the clear spirit doth raise
> (That last infirmity of noble mind),
> To scorn delights, and live laborious days."

—I am, etc.,

J. CHARLES SHEE.

Bulawayo,
Rhodesia.

Haemoglobin E and α-Thalassaemia

SIR,—In their interesting article on the combination of heterozygous haemoglobin E with α-chain deficiency of intermediate severity Dr. P. Wasi and others (7 October, p. 29) discuss why their patients have an average of 14% Hb E, 8.4% Hbγ_4 and practically no β_4^A, the remainder being Hb A with minor quantities of F. This pattern differs from the expected one—namely, Hb β_4^A in amounts comparable to those observed in α-thalassaemia intermedia (Hb H disease) and an A/E ratio of 2.5–3.0 to 1, as seen in simple AE heterozygotes. A surplus of β^E chains could be expected as well; in an earlier study on similar patients[1] no β_4^E could be detected.

The thalassaemia syndromes have demonstrated that failure to detect an expected