hibitor and Fast Red TR salt solution in 3% aqueous dupanol as colour index. The results are summarized in the Table.

Genetic Disorder	Number Examined	Abnormal Cases	%
β -thalassaemia trait	156	27	17.3
Hb A ₂ increased (>3.5%)	156	25	16.0
β -thal. (Hb F > 5%)	156	2	1.3
G-6-PD deficiency	155	8	5.2
Atypical ACAH	187*	13	6.9

* Including 32 Cypriot students of Athen's University.

The prevalence of the β -thalassaemia trait was high in the sample examined. With a prevalence of 17.3% the birth rate of homozygous β -thalassaemia can be estimated to be 0.8%. A relative birth rate of 0.6% was also found among Cypriots in London.⁵

Considering the severity of thalassaemia major these figures demonstrate its seriousness as a public health problem in Cyprus. Since at present there is no other way of reducing the birth rate of patients with thalassaemia major except by genetic counselling, it is suggested that the whole population at risk should be screened for the thalassaemia trait and careful genetic counselling provided to all prospective couples whenever both the prospective father and mother are heterozygous.

On the other hand, the prevalence of clinical manifestations associated with G-6-PD deficiency, namely, favism and severe neonatal jaundice, seems to be higher than that expected on the basis of the rather low prevalence of G-6-PD deficiency (5.2%).⁶ It should be noted in this respect that in addition to G-6-PD deficiency other unknown factors, probably genetic, may be responsible for precipitating clinical manifestations in sensitive individuals.

Interestingly we found a relatively high prevalence of atypical ACAH; this is generally believed to be geographically distributed rather homogeneously with a prevalence of about 4%.⁷ The higher prevalence observed in this study (6.9%) may partly explain why a number of the first cases of suxamethonium apnoeas and some of the rare abnormal ACAH genotypes were described in Cypriots residing in England.—We are, etc.,

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Herpes Simplex and Temporal Lobe Epilepsy

SIR.—We were interested in Dr. Constance A. C. Ross's letter (8 July, p. 112) concerning herpes simplex virus and temporal lobe epilepsy. Following a report¹ that patients

with an aggressive psychosis had a very high titre of antibody to herpes simplex virus compared with controls we conducted a small survey of sera from nine cases of temporal lobe epilepsy, 14 cases of idiopathic epilepsy, and 23 normal controls, matched for age and sex. When we used the fluorescent antibody method to detect herpes virus-specific IgM and to measure the titre of IgG against herpes simplex virus there was no significant difference in antibody titre between the three groups, and herpes virus-specific IgM was not found in any serum.

The results do not exclude past damage by herpes simplex virus but make continuous active infection unlikely. We agree with Dr. Ross that antibody titres should be checked on paired sera.—We are, etc.,

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Virus-specific Antibodies in Multiple Sclerosis

SIR.—Some patients with multiple sclerosis were found by the fluorescent antibody method to possess measles virus-specific immunoglobulin M (IgM).¹ A second survey made on 57 sera from multiple sclerosis and 57 from individuals in normal health, matched for age and sex, has confirmed these findings and has added some information on virus-specific immunoglobulin G (IgG), detected by the same method.

IgM specific for measles virus-infected cells.—The incidence of IgM staining by sera from multiple sclerosis on measles virus-infected cells appears in Table 1. The staining is weak and the pattern diffuse, slightly brighter at the cell margin, and leaves unstained known intracellular aggregates of virus antigen. It was not seen in cells infected with the viruses of mumps, herpes simplex, varicella, vaccinia, or rubella. It is not removed from serum by normal brain tissue. It is removed by absorption with crudely pelleted measles virus and from about half the sera by absorption with aggregated human gamma globulin. This may indicate secondary staining by rheumatoid factor, but nevertheless is immunological.²

Table 1—Number of Sera Showing IgM Staining of Measles Virus-infected HEp₂ cells

Multiple Sclerosis (56 sera)		Controls (56 sera)	
Absorption with aggregated gamma globulin		Absorption with aggregated gamma globulin	
Before	After	Before	After
22	9	1	0

Titres of virus-specific IgG.—As may be expected,³ we found a significant increase of measles virus-specific IgG in multiple sclerosis patients as compared with control subjects (Table 2). We were surprised to find an equally clear increase of herpes simplex virus-specific IgG, but none against

rubella virus. Titres against mumps or herpes zoster viruses were low and not different in the two groups.

Table 2—Geometric Mean Titres (reciprocal) of Virus-specific IgG in 57 Pairs of Sera

	Multiple sclerosis	Normal controls	Significance χ^2 (df = 1)
Measles virus	31.4	18.3	9.03 : P < 0.01
Herpes-simplex virus	40.8	17.5	7.19 : P < 0.01
Rubella virus	25.9	25.7	0.55 : P > 0.40

Virus-specific IgG in cerebrospinal fluid.

—Serum and cerebrospinal fluid taken together from 25 patients and 17 normal subjects of about the same age as the patients, were tested for the presence of antibody to the viruses of measles, mumps, and herpes simplex and some, 16 multiple sclerosis and 11 control, for IgG to rubella virus. Sixteen of 25 patients had measles virus-specific IgG in cerebrospinal fluid and 8 of these 16 also had IgG specific for herpes simplex virus. No rubella IgG was found in the C.S.F. No virus-specific IgM was detected in any C.S.F. and no virus-specific IgG in controls.

The presence in serum of IgM specific for a minor component only of measles-infected cells may be related to a report (personal communication, Dr. E. Norrby) that some multiple sclerosis sera have more antibody against measles virus haemolysin than against virus haemagglutinin. It could also represent a cross-reaction with some other infectious agent or tissue or haemolysin.

Similar examination of cerebrospinal fluids from other chronic or subacute diseases of the central nervous system will show whether the particular IgM reacting with measles virus is characteristic of multiple sclerosis and also how often such fluids contain immunoglobulins specific for more than one virus.—We are, etc.,

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E.N.T. Advances

SIR.—In a review (22 July, p. 243) Mr. John Ballantyne raised the question whether there was a place for books which discuss trends of modern development in a specialty. I do not intend to dispute whether this is so or not, but I would like to take issue with the examples given in favour of his argument. He pointed out that omissions of most recent advances are inevitable in books of this type and gives as an example the lack of reference to electrocochleography, which, in his opinion is "almost certainly destined to become the best objective test of cochlear function for many years to come."

Electrocochleography was in a very experimental stage when I wrote the chapter on the deaf child in this book, but were I to write it today I still might have omitted cochleography. Many "objective" tests have