This method seems a useful corollary to conventional histological methods for diagnosing Hirschsprung's disease, since the diagnosis rests on showing the *presence* of abnormal fluorescent nerves rather than the *absence* of ganglion cells. Whereas many serial sections must be prepared for the latter technique, the fluorescent method requires relatively few sections to demonstrate the abnormal nerve filaments and trunks.

A full report of this work is to be published separately.

Department of Zoology, University of Melbourne, Parkeville, Victoria 3052, Australia.
B. J. GANNON
G. BURNSTOCK.

Royal Children's Hospital, Melbourne, Australia.
HELEN R. NOBLETT
P. E. CAMPBELL.

GASTROENTERITIS

SIR,—Last week (p. 843), under the heading Public Health, you published a description of gastroenteritis at Booth Hall Children's Hospital, Manchester, in which it is stated that "*E. coli*, type 0114—a type normally regarded as non-enteropathogenic" was believed to be the cause. *Escherichia coli* 0114 was isolated from sporadic cases of enteritis in babies in the London area and from an outbreak of infantile diarrhœa in Birmingham towards the end of 1951, and strains were also identified from cases of calf scours,[1 2] so this type is known as a cause of enteritis in babies. Between 1951 and late 1968 very few cases have been identified in this laboratory, which receives strains from the U.K., so suggesting that this infection has not been an important cause of infantile diarrhœa during this period. Towards the end of 1968 cases arose in the London area and subsequently the outbreak in Booth Hall Children's Hospital was recognised. The flagella antigen of the recent London and Manchester cases is H2—the same as that encountered in 1951 in the Birmingham outbreak. The early London cases and the calf strains were of different flagella types, not H2. An outbreak due to *E. coli* 0114.H2 in 1958 was described in Germany.[3]

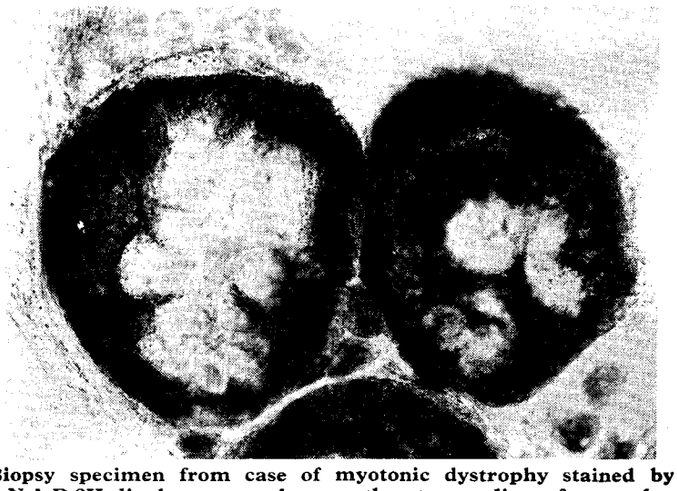
Salmonella Reference Laboratory, Central Public Health Laboratory, London N.W.9.
JOAN TAYLOR.

HISTOCHEMICAL ANOMALY IN MYOTONIC DYSTROPHY

SIR,—Enzymohistochemical techniques in the study of neuromuscular diseases, with the enzymatic recognition of type I and II fibres, have contributed to the demonstration of characteristic patterns of atrophy, preferential involvement of fibre types, and, in some cases, the existence of structural anomalies of the enzymatic texture of the muscular fibres (e.g., target and targetoid changes, central core, mitochondrial aggregates), and are of value in the differential diagnosis of the myopathies.

In two cases of myotonic dystrophy, in brothers aged 38 and 39 years, we have observed an identical anomaly affecting many muscle fibres. With the nicotinamide-adenosine-dinucleotide (reduced form) (N.A.D.2H)-diaphorase and succinate-dehydrogenase techniques, many of the muscle-fibre cross-sections show focal loss of enzymatic activity, resulting in large unstained areas with irregular boundaries, giving a coarse, moth-eaten picture (see accompanying figure). As a rule, these areas of enzymatic inactivity have a border with irregular discoloration and clumping of formazan granules, and the intermyofibrillary frame is bizarre in structure. With hæmatoxilin-eosin staining, and adenosine-triphosphate (A.T.P.)-ase and edetic-acid-activated A.T.P.-ase techniques, the texture of these fibres is normal.

Because of the reproductibility of the results, the similarity



Biopsy specimen from case of myotonic dystrophy stained by N.A.D.2H-diaphorase to show moth-eaten outline of muscular fibres.

Reduced to three-fifths, from × 600.

of the features in both cases, and the enzyme-dependence of the lesions described, we do not believe that these related changes are artefacts caused by the technique used. We do not know how far the anomaly we have observed corresponds to an actual structural alteration of the muscle fibre, or to disturbance of myofibrillary disposition by myotonic changes during the surgical operation of biopsy, but we do not think it has been described before—it was not reported in the histochemical study of cases of myotonic dystrophy by Engel and Brooke.[1]

A more detailed report of the present study will be published elsewhere.

Department of Pathology, Rehabilitation Centre, Ciudad Sanitaria de la Seguridad Social, Barcelona 16, Spain.
AUGUSTO MORAGAS
R. SALES-VAZQUEZ.

MACROCYTOSIS IN DOWN'S SYNDROME

SIR,—A survey of routine blood-samples from non-anæmic mentally retarded patients showed a significantly increased mean red-cell volume in epileptic patients treated with anticonvulsant drugs. We also found macrocytosis in non-anæmic patients, aged 21–65 years, with Down's syndrome (D.S.) not treated with anticonvulsants (confirming the findings of Naiman et al.[1] in 12 patients with D.S. and 12 controls), as follows:

Patients	*Hb (g. per 100 ml.)*	*No.*	*Mean cell volume (cμ.)*	*(±1S.D.)*
Women ..	11·5–16·4	47	102·7	(±7·22)
Men	13·5–18·0	45	102·2	(±8·67)
Normal range[2]	..	..	76–96	

Red-cell counts were performed on a Coulter counter after dilutions of whole blood had been made using Hook & Tucker 'Autodiluters'.

Full details are being published elsewhere.

Frenchay Hospital and Stoke Park Hospital, Bristol.
R. D. EASTHAM
J. JANCAR.

FOUNDATION OF THANATOLOGY

Dr. AUSTIN H. KUTSCHER (president, Foundation of Thanatology, Columbia-Presbyterian Medical Center, 630 West 168th Street, New York 10032) writes: "The Foundation of Thanatology, a non-profit-making organisation, devoted to scientific and humanistic inquiries into death, loss, grief, and recovery from bereavement, has been set up to offer an educational, research, and publication programme to workers in the health professions, theology, psychology, and the social sciences. Associate membership is available by writing to me. The chairmen of the executive committee, multidisciplinary professional advisory board, and research committee are Dr. Bernard B. Schoenberg, Dr. Arthur C. Carr, and Dr. David Peretz, respectively."

1. Rogers, K. B., Cracknell, V. M. *J. Path. Bact.* 1956, **72**, 27.
2. Charter, R. E. *ibid.* p. 33.
3. Linzenmeier, G. *Zentbl. Bakt. ParasitKde*, Abt I, Originale, 1960, **177**, 435.

1. Engel, W. K., Brooke, M. H. *in* Progressive Muskeldystrophie, Myotonie, Myasthenie (edited by E. Kuhn); p. 203. Heidelberg, 1966.
2. Naiman, J. L., Oski, F. A., Mellman, W. J. *Lancet*, 1965, i, 821.
3. Dacie, J. V., Lewis, S. M. Practical Hæmatology. London, 1968.