The note also states that "during 1970, 101 cases of malaria were reported by [Public Health] laboratories in the United Kingdom." This figure is a serious underestimate of the total number of cases of malaria notified in Britain.

Information provided by the Department of Health and Social Security indicates that the quarterly returns of the Registrar General for 1970 quote 137 confirmed notifications of malaria in England and Wales. In addition to that the Scottish Home and Health Department received in 1970 not less than 24 notifications of malaria of which 19 were confirmed. This brings the known number of malaria cases in England, Wales, and Scotland to at least 156. Carswell and Stevenson² reported recently that over the period 1965-9 the number of cases of malaria seen in Scotland averaged 20 per annum. Malaria is not notified in Northern Ireland but the cases confirmed by the Public Health Laboratories in 1970 were included in the figures reported in your note.

I wish to acknowledge with thanks the assistance obtained from the Department of Health and Social Security in providing me with the relevant data. I should also like to draw attention to the excellent *Notice to Travellers* of 1970 prepared by the D.H.S.S. and available on demand.

—I am, etc.,

L. J. BRUCE-CHWATT

London School of Hygiene
and Tropical Medicine,
London W.C.1

¹ Bruce-Chwatt, L. J., *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 1970, **64**, 201.

² Carswell, J., and Stevenson, D. J. D., *Scottish Medical Journal*, 1971, **16**, 250.

Drugs and the Fetal Heart Rate

SIR,—We have read with interest the letter by Dr. J. C. Grimwade and others concerning the influence of morphine on the fetal heart rate pattern (7 August, p. 373). We have observed another effect on the fetal heart rate pattern of analgesics or central depressor drugs administered to the mother.

The normal fetal heart rate pattern shows both short term and long term irregularity.¹ These two types of irregularity can be distinguished and measured by automatic processing methods.² We have found that the short term irregularity decreases or even disappears, producing a "silent" fetal heart rate pattern, after administration to the mother of analgesics or central depressor drugs such as diazepam (10 mg i.v.), meperidine (75 mg i.m.), phenobarbitone (75 mg i.m.), mephobarbitone (75 mg i.m.), or the combination of meperidine (50 mg i.m.) with promazine hydrochloride (25 mg i.m.).^{3,4} We have seen cases of anencephaly with short term irregularity of the fetal heart, though others have had a "silent" fetal heart rate pattern.^{3,4}

Evidently short term irregularity can occur in the absence of the cerebral cortex, and this suggests that the central depressor drugs must be acting at a lower level of the central nervous system (possibly hypothalamus or medulla oblongata) when they abolish the short term irregularity. The short term irregularity appears to result from oscillating vagal activity.⁴—We are, etc.,

J. DE HAAN
L. A. M. STOLTE

Department of Obstetrics and Gynaecology,
Academisch Ziekenhuis der Vrije Universiteit,
Amsterdam, Holland

¹ Caldeyro-Barcia, R., in *The Heart and Circulation in the Newborn and Infant*. Ed. Donald E. Cassels. New York, Grune and Stratton, 1966, p. 7.

² Haan, J. de, et al., *European Journal of Obstetrics and Gynecology*, 1971, **1**, 95.

³ Haan, J. de, et al., *European Journal of Obstetrics and Gynecology*, 1971, **1**, 103.

⁴ Haan, J. de, *Short Term Irregularity in the Fetal Heart Rate Pattern*, M.D. thesis, Amsterdam, 1971.

Doctor in the Lab

SIR,—Your leading article (25 September, p. 719) draws an inference that pathologists who spend part of their time practising another specialty than their main one appear to tolerate a lower standard than is accepted in the bedside specialties, where you infer this does not happen. We all know it does happen, and often much to the benefit of patient and of doctor. A physician keeps his eyes, ears, and mind open to symptoms and signs outside his immediate specialized reference, and has the courage and modesty to accept the contribution of all the signs and symptoms he and others observe when forming his final opinion. The pathologist who neglects to consider every aspect of pathology he can turn his mind to when assessing the relevance of his laboratory's findings provides a lower standard of service than the one who keeps his mind open and refuses to dogmatize on the inadequate evidence of his own specialty.

The present attempt to appoint at least one specialist in each of the four current (and already outdated and technically illogical) subdivisions of pathology leads to quite ludicrous situations when these specialists have been trained in centres where the responsibility for whole groups of tests can be quite differently allocated. The same test may even be performed, with variations according to technical taste, in three or even four different divisions of the same laboratory.

The function of the clinical pathologist is to collaborate and consult with his clinical colleagues; to go back in fact to the beginnings of modern clinical pathology again.

F.D.P. in Glomerulonephritis

SIR,—We read with interest the paper by Dr. A. R. Clarkson and his colleagues (21 August, p. 447) and would like to add our own observations and comments.

We measured serum and urinary F.D.P. content on specimens taken from 20 consecutive patients attending two renal outpatient clinics. The tanned red cell haemagglutination inhibition immunoassay was used for all determinations. In our laboratory the normal range for serum F.D.P. is 1.25-20 µg/ml. Of 40 normal subjects 38 had no detectable F.D.P. in their urine, while the

His training must provide a wide clinical experience and a practised scepticism. The latter is most readily acquired in a laboratory with correlation and follow-up systems; and in the postmortem room. He gives his time freely to discussion with the clinician in assessing the relevance of his laboratory findings to any suspected diagnostic entity, and, more important still, to consideration of what next is to be done—to treat or test again. He must be on terms of complete and honest appreciation of his own and of his colleagues' limitations if his opinion is to have full value and if a mutual confidence is to develop for the benefit of future patients. The more factual information his data-storage and computer-analysis systems provide in the future, the more important (and just possibly the easier) this will become.

A transition to Zuckerman¹ (slow for reasons of pride and humanity) will help the clinical pathologist to concentrate on his re-found role. There will be plenty of scope for the ultra-specialist in the large central non-urgent laboratories of the future, and the universities will supply (according to funds, development of methods, and the availability of men and enthusiasm) the laboratory space, equipment, and stimulus for him. In the teaching hospitals, however, there must be a department of clinical pathology with one general pathologist at its head. He will have the functions I describe above, and there will be no need for him to compete for divisional chairmanship (or whatever) with his fellow pathologists on any other than equal terms, deriving his satisfactions from the knowledge that his functions are unique and not competitive with those of his fellow pathologists at one or more removes in their specialized laboratories.—I am, etc.,

G. A. C. SUMMERS

Group Pathology Laboratory,
County Hospital,
York

¹ *Hospital, Scientific, and Technical Services*. London, H.M.S.O., 1968.

remaining two had concentrations of 1.25 µg/ml.

Without recourse to the F.D.P. results, the patients were divided into three groups: (1) those with normal renal function; (2) those with impaired but static renal function; and (3) those with progressive impairment of renal function.

The criteria for the continued activity of the disease process were essentially the same as those offered by Dr. Clarkson and his colleagues.

Normal Renal Function		Static Impairment of Renal Function		Progressive Impairment of Renal Function	
Serum F.D.P. (µg/ml)	Urine F.D.P. (µg/ml)	Serum F.D.P. (µg/ml)	Urine F.D.P. (µg/ml)	Serum F.D.P. (µg/ml)	Urine F.D.P. (µg/ml)
10	0	10	5.0	20	20 (a)
5	0	10	1.25	10	10
5	0	20	2.5	20	10
10	1.25	20	2.5	80	>20 (b)
		20	1.25	5	5
		5	1.25	10	5
				< 1.25	5
				20	>20 (c)
				40	20 (d)
				20	40 (e)

(a) Lupus Nephritis; (b) Hydronephrosis; (c) Polycystic Kidneys with Renal Failure; (d) Post-streptococcal Nephritis; (e) Proliferative Glomerulonephritis.