General Attack Rate in "Hong Kong" H ₂ N ₂ Influenza Epidemic	Previous "Asian" H ₂ N ₂ Virus				Previous B-type Influenza Virus			
	No. in Group	Infected with H ₂ N ₂ Virus		No. in Group	Infected with H ₂ N ₂ Virus		No. in Group	Infected with H ₂ N ₂ Virus
		Expected	Observed		Expected	Observed		
Epidemic I 5% ..	118	5.9	0	73	3.6	1	73	3.6
Epidemic II 15% ..	118	17.7	5	72	10.8	9	72	10.8
Epidemic III 4% ..	113	4.5	0	63	2.5	2	63	2.5
Total		28.1	5		16.9	12		

given in the accompanying Table, where it is shown that a group of persons known to have been infected with "Asian" H₂N₂ virus had a significantly lower attack rate with "Hong Kong" influenza virus (H₂N₂) than a group known to have been infected with influenza B during the same period. The difference was four-fold. The figures are derived from a combined field and laboratory study of respiratory disease in a general practice population of 3,600. The investigation began in October 1961 and is continuing.—I am, etc.,

R. EDGAR HOPE-SIMPSON

Public Health Laboratory Service,
Epidemiological Research Unit,
Cirencester, Glos

1 Gill, P. W., *British Medical Journal*, 1971, 3, 308.

2 Hope-Simpson, R. E., *British Medical Journal*, 1971, 3, 531.

Warfarin and Hydroxyethylrutosides in Deep Vein Thrombosis

SIR,—Although the exact pharmacological mechanisms involved are not yet fully understood, it is known that hydroxyethylrutosides have a beneficial effect on capillary function, reducing oedema and augmenting venous return in venous stasis.^{1,2,3} In an attempt to determine whether long-term oral anticoagulant treatment of patients with deep vein thrombosis could be improved by the addition of rutosides 12 such patients admitted consecutively were treated with a standard regimen of 4 to 5 days of continuous heparin infusion followed by oral warfarin plus troxerutin (Paroven; a preparation of hydroxyethylrutosides) given orally at the rate of 250 mg every six hours. After discharge the patients were examined regularly as outpatients, when blood samples were tested for haemoglobin concentration, haematocrit, white blood cell count, total platelet count, in vivo adhesive platelet count,⁴ plasma fibrinogen, plasma viscosity, and prothrombin ratio (P.R.). Warfarin dosage was adjusted according to the plasma activated partial thromboplastin time (A.P.T.T.)⁵ and, in addition, the heparin-retarded activated partial thromboplastin clotting time was estimated routinely.⁶

It had previously been found that the A.P.T.T. is reduced below the normal range after thrombosis, trauma, or haemorrhage,⁷ and a comparison of the A.P.T.T. with its

By the t test there is no significant difference between the mean values for any two of these three clinical groups. ($P > 0.05$.)

Clinical Condition (Deep Vein Thrombosis)	No. of Patients	No. of Tests	Mean A.P.T.T.:P.R. ratio	S.D.
Post-operation warfarin only ..	52	372	32.09	5.59
"Spontaneous," warfarin only ..	39	883	30.93	5.19
Warfarin plus troxerutin ..	12	129	31.98	4.84

Logarithm of heparin-retarded A.P.T.T. results plotted against the corresponding A.P.T.T. results in (a) patients treated with warfarin only, and (b) patients treated with warfarin plus troxerutin, after deep vein thrombosis.

corresponding P.R. during long-term oral anticoagulant therapy appeared to show this same tendency in patients after deep vein thrombosis when compared with patients after myocardial infarction.⁸ When the present series of patients treated with

warfarin and rutosides was compared with similar patients treated with warfarin only no significant difference in the A.P.T.T.: P.R. ratios was found (see Table), nor was there any significant difference in the adhesive platelet counts.

There is an exponential relationship between the A.P.T.T. and the concentration of added heparin, and it has been reported that after thrombosis or trauma heparin has less effect in prolonging the A.P.T.T. than normal,⁹ this apparent resistance to heparin decreasing with recovery. In the present series no difference in response to heparin was found between the two clinical groups (see figure).

Haemorrhage is a well-known complication of oral anticoagulant therapy. During 1967-71 inclusive 301 patients were treated with long-term oral anticoagulants in this laboratory outpatient department for a total of 3,445 man/months, and 103 haemorrhagic episodes occurred.¹⁰ During 100 man/months of treatment of 12 patients with oral warfarin plus troxerutin four haemorrhagic episodes occurred, two of which were known to have been caused by the unwitting prescription of drugs known to potentiate the action of warfarin (imipramine hydrochloride and phenylbutazone respectively).

These findings are disappointing in that we have no clinical or laboratory evidence for more rapid improvement in recovery following deep vein thrombosis when troxerutin was given with warfarin. On the other hand these findings show that there is no potentiation of warfarin activity when troxerutin is given, and it appears that this drug can be given orally with safety during warfarin therapy.

We are grateful to our clinical colleagues at Frenchay Hospital for their encouragement, and we thank Zyma S.A. for generous supplies of Paroven.—We are, etc.,

R. D. EASTHAM
T. G. M. PERHAM
P. V. POCOCK

Department of Pathology,
Frenchay Hospital,
Bristol

- McEwan, A. J., and McArdle, C. S., *British Medical Journal*, 1971, 2, 138.
- Sorensen, A. H., and Hansen, H., *Angiologica*, 1970, 7, 187.
- Pfister, R., and Lindner, U., *Therapiewoche*, 1967, 17, 674.
- Borchgrevink, C. F., *Acta Medica Scandinavica*, 1960, 168, 157.
- Eastham, R. D., *British Medical Journal*, 1968, 2, 337.
- Eastham, R. D., *Journal of Clinical Pathology*, 1962, 15, 86.
- Eastham, R. D., and Morgan, E. H., *Lancet*, 1964, 2, 543.
- Eastham, R. D., in *International Society on Thrombosis and Haemostasis. II Congress (abstracts)* p. 14 Oslo, Norway, 1971.
- Poller, L., *Postgraduate Medical Journal*, 1960, 36, 564.
- Eastham, R. D., *Bristol Medico-Chirurgical Journal*, 1972, 87, 5.

Pulmonary Aspiration after Fibre-endoscopy

SIR,—Drs. B. J. Prout and C. Metreweli (4 November, p. 269) stress the contribution made by local pharyngeal anaesthesia to the hazard of pulmonary aspiration during gastrointestinal endoscopy. They conclude that the effect of local anaesthesia is unavoidable, but their results may well have been influenced by the remarkably heavy dosage used.

Pharyngeal anaesthesia is a traditional preparation for endoscopy, but it is omitted in a number of foreign centres. Controlled studies of tripelethamine (Pyrizemine)¹

and diphenhydramine² have shown no advantage over placebo from the point of view of patient or doctor. In a brief experience so far our patients appear to have tolerated examinations satisfactorily without any pharyngeal anaesthesia. It may thus be unnecessary as well as potentially dangerous.—I am, etc.,

P. B. COTTON

St. Thomas's Hospital,
London S.E.1

¹ Buchanan, D. P., *American Journal of Digestive Diseases*, 1960, 5, 121.
² Sparberg, M., and Knudsen, K. B., *American Journal of Digestive Diseases*, 1967, 12, 1131.

Harry's Angels

SIR,—How refreshing to read the story from South Africa under the title "Harry's Angels" (4 November, p. 288). It lifts one's spirit to hear of a team of highly-skilled specialists from Johannesburg who sacrifice their leisure weekends to travel by air to and from Swaziland (that small country with one doctor to 8,000 patients) in order to create, at their own expense, a first-class free medical and surgical service there, until such time as the country can supply its own needs.—I am, etc.,

OLIVE K. BURNETT

Hove, Sussex

Foley-catheter Induction of Labour

SIR,—The method described by Mr. J. R. Saunders (28 October, p. 237) deserves to be widely known. It has the great advantage of not compromising the patient's physical or mental condition. Further, when delivery is not urgent and there is time for its leisurely employment, it is, in cases when the cervix is unfavourable, a most effective insurance against failure of induction.

The patient is hardly aware of the catheter after its insertion. The membranes have not been ruptured; there is no risk of sepsis. There is no prolonged period of draining liquor and the patient is not attached to an oxytocin infusion or sucking Pitocin tablets by day and unable to take her normal meals. A course common with induction with the unripe cervix, which is encouraged by anxiety and starvation and which leads to acidosis and incoordinate labour, is never begun. The Foley catheter does not compromise the fetal condition. Oxytocin infusion, buccal Pitocin, and amniocentesis can all adversely affect it sooner or later.

The method has great value in cases where the patient has previously been subjected to caesarean section for inco-ordinated uterine action. I have concluded that there have been many cases when this method of induction might have avoided the original operation.—I am, etc.,

W. G. DAWSON

Shaw, Near Swindon,
Wilts

The "Dysarthria Address"

SIR,—Throughout various medical centres in the world and particularly in hospitals in the United Kingdom the patient is asked to say "37 West Register Street, Edinburgh," one of the common phrases used in testing for dysarthria. During a recent visit to Edinburgh I decided to determine what is located at that famous address.

Following West Register Street from Saint Andrew's Square the numbers ceased in the 40s and continued from 19 somewhat further down the street. Occupying most of this numberless gap was an office building with a temporary sign pasted on its door. The inhabitants of the building did not know its street number, and therefore the query was referred to the supervisor. With a puzzled look, he admitted that they only recently had transferred their office to the present location and ruefully noted that he did not know the number of the building. His curiosity aroused, we walked up and down West Register Street together trying to decide where "37" would be. We concluded that it was one of the many numbers encompassed by the building housing his office.

If so, 37 West Register Street is one of the addresses of the Public Relations Office of the Royal Bank of Scotland.—I am, etc.,

GEORGE J. DOHRMANN

The National Hospital, Queen Square,
London W.C. 1

Association between Anaemia and Hypolipidaemia

SIR,—The paper by Professors L. E. Böttiger and L. A. Carlson (23 September, p. 731) is of interest since it suggests a hypothesis which may explain, at least in part, the long-known but hitherto unexplained association between anaemia and hypolipidaemia. The hypothesis assumes a constant total blood volume and a constant plasma cholesterol pool size, so that an increase in packed cell volume (P.C.V.) produces a rise in plasma cholesterol concentration secondary to the contraction in plasma volume.

Professors Böttiger and Carlson studied subjects with haemoglobin values in the normal range, and it is not known if their conclusions can be extrapolated to the anaemic situation. Data collected for a different purpose from five subjects with pernicious anaemia, at the time of diagnosis and during response to treatment, are given in the Table below together with the change in serum cholesterol which would have been predicted by the Böttiger-Carlson hypothesis.

In every case the observed increase in plasma cholesterol exceeds that predicted by the Böttiger-Carlson hypothesis, and overall the observed increase is of the order of twice the predicted increase. Professors Böttiger and Carlson were careful to state that their hypothesis "may in part account for the positive correlation between haemoglobin and plasma lipids" [my italics], and the data given here suggest that it may indeed be necessary to postulate an alteration in either or both of total blood volume and plasma cholesterol

pool size to explain the rise in plasma cholesterol concentration which occurs when anaemia is treated. Huber, Lewis, and Szur¹ found little evidence of any change in total blood volume over the P.C.V. range 20-50%, so it seems likely that there is an increase in plasma cholesterol pool size when anaemia is treated, but clearly more direct evidence is needed before this can be stated unequivocally.—I am, etc.,

J. P. MILLER

Royal Postgraduate Medical School
London, W. 12

¹ Huber, H., Lewis, S. M., and Szur, L., *British Journal of Haematology*, 1964, 10, 567.

Infertility after the Pill

SIR,—Your leading article on infertility after the pill (14 October, p. 59) reminds me that a few years ago you published a letter from me,¹ the gist of which was to point out the possibility of this in certain cases.

My view² has always been that there is an appreciable number, if a small percentage, of young and apparently normal women in whom some degree of hormonal dysfunction exists resulting in endometrial change and menstrual disturbance unrecognizable by the individual. This group is at risk in coping with the interference with the function of the hypothalamus consequent upon taking the pill over a lengthy period of time. In such cases the prolonged and somewhat unpredictable treatment by clomiphene and pituitary gonadotrophin cannot be altogether satisfactory.

In order to minimize the possibility of lifelong disappointment in these women I suggest that a pelvic examination, and a diagnostic curettage if necessary, be carried out in all cases exhibiting the slightest menstrual irregularity before prescribing the pill. If such a procedure is impracticable I think these cases will increase in number in these days in which oral contraceptives are regarded as a universal panacea.—I am, etc.,

K. VERNON BAILEY

Altrincham, Cheshire

¹ Bailey, K. V., *British Medical Journal*, 1969, 1, 183.
² Bailey, K. V., *Journal of Obstetrics and Gynaecology of the British Empire*, 1959, 66, 556.

Tumours of the Salivary Glands

SIR,—Salivary tumours occur predominantly in the parotid gland, and the advances in parotid surgery in the past quarter-century have largely solved the problem of treatment of all except the uncommon highly malignant tumours of this organ. These advances have depended on the adoption of the

Patient	P.C.V. %	(100-P.C.V.) %	Plasma Cholesterol mg/100 ml	Change in Plasma Cholesterol mg/100 ml	Predicted Change in Plasma Cholesterol mg/100 ml
H.C.	15	85	115	0	0
	28	72	157	+42	+21
	41	59	205	+90	+51
J.D.	29	71	178	0	0
	46	54	264	+86	+56
G.R.	36	64	199	0	0
	47	53	281	+82	+41
J.B.	23	77	158	0	0
	40	60	293	+135	+45
I.Q.	28	72	120	0	0
	33	67	139	+19	+9
	37	63	157	+37	+17
	40	60	211	+91	+24