Mean Values (± S.D.) of Motor and Sensory Conduction Velocities (in m/sec.) in Hypertensive Patients and Controls

	Median Nerve		Ulnar Nerve		Posterior Tibial Nerve	Lateral Popliteal Nerve
	Motor	Sensory	Motor	Sensory	(Motor)	(Motor)
Hypertensive	58·89 ± 3·86	66·56 ± 4·03	57·37 ± 4·55	65·14 ± 4·24	49·56 ± 4·04	51·35 ± 5·28
Controls	59·19 ± 3·36	66·10 ± 3·95	57·37 ± 4·39	63·71 ± 4·51	51·44 ± 4·61	52·04 ± 4·64

rates in the lateral popliteal and posterior tibial nerves ($P < 0.001$).

The results of our investigation do not support the finding previously reported that hypertensive patients suffer from a subclinical peripheral neuropathy. Even in those patients with markedly elevated diastolic pressures (< 150 mm Hg) no significant slowing of conduction was discovered.

We are very grateful to Mr. Basil Hellman for his assistance with the statistics and to Dr. D. Taverner for his advice and encouragement with the study.

J. F. BRIDGMAN
L. BIDGOOD
R. HOOLE

General Infirmary,
Leeds

1 Viskoper, R. J., Chaco, J., and Aviram, A., *Archives of Internal Medicine*, 1971, **128**, 574.

Lomotil Intoxication in Children

SIR,—I am glad you have pointed out in your leading article (23 June, p. 678) the dangers of Lomotil. In the last two years I have seen several children who have been treated with Lomotil for diarrhoea. They have had a variety of puzzling clinical patterns, but the most disturbing has been the abdominal distension. When faced with a two-year-old with this situation my immediate suspicion is now Lomotil intoxication, not appendicitis. It is still a very disturbing situation to all concerned.

I support your contention that Lomotil should not be given to young children, but where it has to be prescribed the doctor should be aware that it is by no means a bland bowel sedative. Very few children who reach my department with diarrhoea have escaped either powerful antibiotics or Lomotil, or both. Our routine (and usually effective) treatment is to stop all medicines. Many children in this country are being seriously overtreated for diarrhoea and vomiting.—I am, etc.,

R. M. FORRESTER

Paediatric Department,
Billinge Hospital,
near Wigan

Fibre Optics in Medicine and Surgery

SIR,—Your recent report describing the work of our department (9 December 1972, p. 604) has provided us with a considerable amount of feedback from the profession at large, an example being Dr. J. de Swiet's letter (21 July, p. 169) in which he has suggested a novel use for our fine-calibre endoscope and also sympathized with our financial problems. With regard to the latter, we are pleased to say that the Rank Trust has generously agreed to support our work in this field (applying fibre optics to medicine and surgery) for a period of two years. Secondly, as a novel application for a fine-calibre probe, Dr. de Swiet has suggested that the diagnosis of bacterial (infective) endocarditis may be achieved using our probe for the visualization of both mitral and aortic valves and that a Valsalva manoeuvre may adequately clear blood from the visual field.

The problem of blood in the visual field is indeed significant and difficult to overcome, and we think that a simple modification of our instrument's distal tip will provide the easiest solution to the problem. This would be accomplished by fitting a transparent inflatable latex balloon around the endoscope's distal tip, which could then be inflated when the instrument is in situ and hence displace sufficient blood from the visual field to enable the inspection to be completed. The addition of a Valsalva manoeuvre may further enhance the visualization.—We are, etc.,

P. J. BRAND
L. T. COTTON

Department of Biochemical Engineering,
King's College Hospital Medical School,
London S.E.5

Smoking Hazards to the Fetus

SIR,—The assertion in your leading article (17 February, p. 369) that "no reasonable doubt now remains that smoking in pregnancy has adverse effects on the developing fetus" overlooks several reports[1-6] that disagree. That the birth weights of children of cigarette-smoking women are lower, on the average, than those of children of non-smoking women is not in question. However, the inference from statistics[7] that smoking *causes* low birth weight (< 2,500 g) is invalid.[6 8] Causation cannot be inferred from statistical association; unfortunately this error has occurred in a number of studies.[7 9 10] For example, a 1964 report[10] states that "the causal significance of an association is a *matter of judgement* which goes beyond any statement of statistical probability" (our italics). Scientific method requires objectivity; but this "method" fails to satisfy such a criterion.

At least two hypotheses may explain the smoking birth weight statistics: (1) cigarette-smoking causes low birth weight, and (2) "the smoking behaviour of women and the birth weights of their children are influenced by a common cause—the individual genotype"[6] or constitution. In a study reported by Yerushalmy[2] the percentages of low-birth-weight infants for women smokers were 6·4 (white) and 12·3 (black) and for non-smokers 3·2 and 5·8 respectively. Thus the presumed "harm" of low birth weight was absent for over 87% of all infants. Hypothesis[2] asserts that infants born to women *before* they become smokers will generally be smaller than those of non-smokers. This was confirmed ($P < 0.01$) for white and weakly confirmed ($0.05 < P < 0.1$) for black women. Further, hypnosis[2] predicts a lower incidence of low birth weight among the infants of ex-smokers, while smoking than for those of habitual smokers. This was confirmed for both black and white women ($P < 0.02$). These findings are incompatible with hypothesis.[1]

The restricted bioenergetic potential of high altitude,[11] and possibly of smokers,[6 12-14] could lead to low birth weight. But since infants of both high and low birth weights are at elevated perinatal mortality risk,[15] the problem is clearly complex.

Endocrinological anomalies influence ethological homoeostatic mechanisms[12 13 16] affecting dietary self-selection[16] and probably self-selected use of coffee[13] and tobacco.[12 14] The biological basis of the coffee and tobacco use association probably involves biogenic amine physiology and bioenergetics.[12-14] Smoking behavioural subgroups (smokers, non-smokers, ex-smokers) are self-selected rather than selected at random, creating special problems for hypothesis testing.[6] If smoking is, for some, a symptom of deficient bioenergetics, such a deficiency may cause a limitation of the metabolism or detoxification of chemicals that nicotine can alleviate.[6 12-14] To the extent that environmental mutagenic hazards are involved, such as HSO_3 and HNO_2 from atmospheric SO_2 and NO_2,[6 13 17] the degree of non-alleviation should be proportional to the risk of accumulation of mutations of germinal DNA.[6] Such mutations can affect development and therefore birth weight.[18]

In summary, it appears that the question of causal relationships between smoking and low birth weight is far from settled.—We are, etc.,

R. J. HICKEY
E. B. HARNER
R. C. CLELLAND

Management and Behavioral
Science Center,

D. E. BOYCE

Regional Science Department,
University of Pennsylvania,
Philadelphia, Pennsylvania

1 Yerushalmy, J., *American Journal of Epidemiology*, 1971, **93**, 443, 1972, **95**, 1.
2 Yerushalmy, J., *American Journal of Obstetrics and Gynecology*, 1972, **112**, 277; **114**, 571.
3 Yerushalmy, J., in *Proceedings of the Sixth Berkeley Symposium on Mathematical Statistics and Probability*, Vol. IV *Biology and Health*, eds., L. M. LeCam, J. Neyman, and E. L. Scott, p. 329. Berkeley, University of California Press, 1972.
4 Yerushalmy, J., *American Journal of Obstetrics and Gynecology*, 1964, **88**, 505.
5 Jansson, I., *Acta Obstetricaet Gynecologia Scandinavica*, 1966, **45**, 279.
6 Hickey, R. J., Clelland, R. C., and Harner, E. B., *Lancet*, 1973, **1**, 270.
7 Butler, N. R., Goldstein, H., and Ross, E. M., *British Medical Journal*, 1972, **2**, 127.
8 Fisher, R. A., *Nature*, 1935, **136**, 474.
9 Fedrick, J., Alberman, E. D., and Goldstein, H., *Nature*, 1971, **231**, 529, 1973, **242**, 263.
10 U.S. Public Health Service, *Smoking and Health*, Publ. No. 1103, p. 182. Washington, D.C., U.S. Government Printing Office, 1964.
11 Lichty, J. A., Ting, R. Y., Bruns, P. D., and Dyar, E., *American Journal of Diseases of Children*, 1957, **93**, 666.
12 Hickey, R. J., Harner, E. B., Clelland, R. C., and Boyce, D. E., *Naturwissenschaften*, 1973, **60**, 206.
13 Hickey, R. J., Clelland, R. C., Harner, E. B., and Boyce, D. E., *Lancet* 1973, **1**, 1003.
14 Hickey, R. J., and Harner, E. B., in *Smoking Behaviour: Motives and Incentives*, ed. W. L., Dunn, jun., p. 267. Washington, D. C., Winston and Sons, 1973, and references cited.
15 Ellis, W. S., *American Journal of Physical Anthropology*, 1973, **38**, 145.
16 Richter, C. P., *Acta Neurovegativa*, 1954, **9**, 247.
17 Hickey, R. J., Harner, E. B., and Clelland, R. C., *Lancet*, 1972, **2**, 1362.
18 Casarett, A. P., *Radiation Biology*, Englewood Cliffs, N. J., Prentice-Hall, 1968.