County, Denmark, blood samples were drawn in the fasting state at 8 a.m. for determination of serum levels of calcium, protein, cholesterol, and triglyceride. The serum calcium levels were corrected to a constant serum protein level.² All determinations were made in duplicate, and the coefficients of variation (C.V.) of duplicate measurements and the mean values and standard deviations (S.D.) are given in the table. A highly significant inverse correlation was found between serum calcium and serum triglyceride ($r = -0.36$, $P < 0.001$). No significant correlation was found between serum calcium and serum cholesterol ($r = 0.03$, $P > 0.05$).

Serum Levels	No. of Subjects	Mean	S.D.	C.V. (from duplicate measurements)
Calcium (mmol/l)	80	2.46	0.06	0.8%
Cholesterol (mmol/l)	80	7.04	1.48	1.5%
Triglyceride (mmol/l)	80	1.27	0.95	2.6%

Conversion: SI to Traditional Units—Calcium: 1 mmol/l $\approx$ 4 mg/100 ml. Cholesterol: 1 mmol/l $\approx$ 38.6 mg/100 ml. Triglyceride: 1 mmol/l $\approx$ 88.5 mg/100 ml.

Our results seem to be supported by the findings that serum cholesterol is lower than normal in patients with hyperparathyroidism and that the serum concentration of lipids increases after operation.³ They indicate that in the elderly serum calcium levels in the lower part of the normal range are not protective against raised serum lipid levels.—We are, etc.,

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¹ Hodkinson, H. M., et al., *Lancet*, 1973, 1, 910.

² Christiansen, C., et al., *Clinica Chimica Acta*, in press.

³ De Moor, P., et al., *Annales D'Endocrinologie*, 1973, 34, 616.

Postoperative Pseudomembranous Colitis

SIR,—We have recently completed a prospective randomized clinical trial to evaluate the effect of systemic prophylactic lincomycin on postoperative sepsis after bowel surgery¹; no other antibiotic was administered. No case of pseudomembranous colitis occurred among the 33 patients who received lincomycin for five days, though the complication was carefully sought. Prophylaxis reduced the incidence of anaerobic but not aerobic infection.

Since this trial we have been combining systemic lincomycin with another broad-spectrum antibiotic and had begun a prospective assessment on patients undergoing elective or emergency resection for large-bowel cancer. Of the 18 patients so far treated, six have developed postoperative pseudomembranous colitis and one has died as a result. In each case the amount of lincomycin received was small (between three and nine 600-mg doses at eight-hour intervals given by intramuscular injection). All six patients received additional broad-spectrum antibiotics (two received gentamicin, three tobramycin, and one co-trimoxazole). No

patient had concurrent disease or debility and hypotension was not observed at any stage during anaesthesia.

The association of lincomycin and pseudomembranous colitis is well recognized, but the pathogenesis of the condition is not understood and is probably multifactorial.^{2,3} Several points arise from our recent experience: firstly, small doses of lincomycin do not prevent the complication of colitis; secondly, the combination of lincomycin with a broad-spectrum antibiotic such as an aminoglycoside may increase the incidence of colitis; and finally, our present prospective trial of a combination of lincomycin and an aminoglycoside as prophylaxis against postoperative sepsis has been discontinued on ethical grounds.—We are, etc.,

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¹ Keighley, M. R. B., et al. In preparation.

² *British Medical Journal*, 1974, 4, 65.

³ Tedesco, F. J., Barton, R. W., and Alpers, D. H., *Annals of Internal Medicine*, 1974, 81, 429.

Surgery of Violence

SIR,—I have read with interest the articles in your series Surgery of Violence. Our recent experience in Cyprus underlines many of the excellent points made in these articles.

On 15 July 1974 a coup was staged in Nicosia and this was followed by an invasion by Turkish troops on 20 July and a subsequent "push" by these troops to Famagusta on 14 August. The British Military Hospital at Dhekelia received wounded of both Cypriot communities and of the contingents serving with the United Nations Force. In all, 59 patients were admitted with injuries associated with this violence. The sites of wounding were: upper limbs, 16 (three with fracture); lower limbs, 32 (six with fracture); head, four; neck, one; chest, seven; abdomen, eight; multiple fragments, two. (Some patients had more than one wound.)

The wounds were caused by mine, shell or bomb fragments, high and low-velocity bullets, and (in one instance) a bayonet thrust. Treatment followed standard military lines which lay emphasis on the following points. (1) Adequate intravenous resuscitation with fluids and blood. (2) Exploration and rigorous debridement of all but the most superficial of wounds. (3) All wounds of muscle left open for delayed primary suture in five to six days. (4) Compound fractures subjected to manipulation at the time of debridement, held in padded plaster casts, and internally fixed only if required after sound soft-tissue healing (in none of our cases was this required). (5) Damaged colonic segments exteriorized and formed into a double-barrelled colostomy for later closure. We found the prophylactic use of dexamethasone and hyperventilation¹ extremely effective in the management of our head wounds.

Cases of interest included the following. (1) Temporal fracture and subdural haematoma due to a "side-swipe" by a high-velocity missile. (2) Tear of the internal carotid artery just below its entry into the base of the skull. (3) Bilateral division of the sciatic nerves by a high-velocity missile passing through both hamstring compartments. (4) Damage to diaphragm, liver, hepatic

flexure of colon, and right kidney due to tiny metallic fragment entering right chest. (5) Severely comminuted fracture of femur with tear of the femoral vein caused by an accidental discharge when a soldier dropped his machine-gun.

There was one death in hospital—a young woman with a severe high-velocity missile head injury—and one death of a man of 70 on return to his village following severe wounds of the pelvis and arm.

Violence, as your series suggests, is "coming home to roost" and it behoves all surgeons and casualty officers to be ready to deal with the multiple injuries that ensue, keeping in mind the basic principles which have saved, and will continue to save, many lives.—I am, etc.,

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¹ Crockard, H. A., *Annals of the Royal College of Surgeons of England*, 1974, 55, 111.

Lactase Activities in the Irritable Colon Syndrome

SIR,—In your leading article on lactose malabsorption (17 May, p. 351) the relevance of hypolactasia to the irritable colon syndrome is discussed. The evidence from Oxford¹ and Denmark² is mildly contradictory on this point, though in the latter study jejunal lactase activities were measured in only nine of the 78 patients under review.

We wish to add our own experience in 15 patients (four male, 11 female) diagnosed as suffering from the irritable colon syndrome by previously established criteria.³ The patients were all Caucasians living in this region. Mucosal lactase activities were measured⁴ using whole specimens taken with a hydraulic biopsy instrument. Our control range was established by reference to recent measurements in 32 subjects with histologically normal mucosa.

The mean jejunal lactase activity in the patients was 3.6 IU/g tissue (wet weight), the actual results ranging from 1.6 to 5.8 IU/g. This compared with the control range of 1.9–10.1 IU/g (mean $\pm$ 2 S.D. obtained by log transformation). In only one case was the value below the control range. Our findings support the conclusions of Pena and Truelove¹ that in most cases a cause other than hypolactasia is responsible for the symptoms of the irritable colon syndrome.—We are, etc.,

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¹ Pena, A. S., and Truelove, S. C., *Scandinavian Journal of Gastroenterology*, 1972, 7, 433.

² Gudmand-Hoyer, E., Riis, P., and Wulff, H. R., *Scandinavian Journal of Gastroenterology*, 1973, 8, 273.

³ Chaudhary, N. A., and Truelove, S. C., *Quarterly Journal of Medicine*, 1962, 31, 307.

⁴ Dahlqvist, A., *Analytical Biochemistry*, 1964, 7, 18.

Treatment of Asthmatic Children with Steroids

SIR,—I read with interest and some concern your leading article on this subject (22 February, p. 413). The article correctly

notes the substantial benefit many children with moderate to severe chronic asthma derive from the use of steroid aerosols. However, it fails to emphasize the important place that regular use of bronchodilators has in management.

It is inferred that if adequate control of asthma is not achieved with regular prophylactic disodium cromoglycate and bronchodilator therapy given early in the course of any acute wheezing attack steroid therapy in one form or another should be used. As about 0.5% of children have chronic airways obstruction as a result of asthma^{1,2} and this is often inadequately controlled with regular disodium cromoglycate and intermittent bronchodilators, steroids appear to be recommended for a large number of children. This is despite the well-known complications of oral corticosteroids and the lack of information of the long-term effects of topical steroids on the human lung.

In fact many children with moderate chronic asthma whose airways obstruction persists despite regular disodium cromoglycate and intermittent bronchodilator therapy will be adequately controlled with regular bronchodilator therapy. The effectiveness of regular oral theophylline in full therapeutic dosage is now well documented.³ While the value of combining one of the newer β -adrenergic stimulants either orally or by inhalation with regular theophylline is not proved, we have used this combination for many years and have achieved very adequate control in a substantial number of children with moderate to severe chronic asthma without any apparent side effects.⁴ Orciprenaline or salbutamol by inhalation using a hand nebulizer driven by small air compressor pump as described in detail elsewhere⁵ is particularly valuable both for the control of persisting airways obstruction and for the rapid relief of acute exacerbations.

Certainly no child should be started on corticosteroids, either by inhalation or orally, until it has been clearly demonstrated that the combination of regular disodium cromoglycate, oral theophylline, and orciprenaline or salbutamol by inhalation has failed to give adequate control. The criteria for control outlined in your article are adequate, but in older children objective evidence from pulmonary function testing is also of considerable importance. Some parents of children with chronic asthma fail to appreciate the severity of the condition and may report that their child is normal whereas he has substantial persisting airways obstruction. Conversely there are a small number of children, usually from disturbed backgrounds, who on reporting by parents seem to be having a great deal of trouble with their asthma, but this is not supported by objective data. There is a substantial risk of undertreating the former group and overtreating the latter if history alone is the guide to therapy.—I am, etc.,

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Motor Insurance and Ischaemic Heart Disease

SIR,—Some interesting points have been raised by Dr. G. J. Rockley (22 March, p. 679) concerning the underwriting of motor insurance where the medical condition of the policy-holder is relevant.

Motor insurance contracts are governed by the principle of utmost good faith—that is to say that there is a requirement on the part of the policy-holder to notify his insurer of any fact which would materially alter the risk. This would include any change in health affecting the ability to drive advised to the patient by his doctor. Insurers would always expect a doctor to advise a patient on the extent and nature of his future activities relative to his medical condition, whether his advice is sought specifically or not. It remains as a matter for the patient's own conscience and common sense as to whether he accepts or rejects the doctor's advice. In the case of illness or some form of disability insurers must defer to the opinion of a registered medical practitioner on the question of the policy-holder's ability and suitability to drive.

In these circumstances it is vital for a patient to understand and appreciate the fact that if he fails to advise his insurers of a change in his medical condition then he would certainly run the risk of having a claim for damage to property (including damage to his own car) repudiated. In so far as personal injury claims are concerned the Road Traffic Act deals with this situation—particularly section 148.

When an insurer requires a report from a doctor concerning fitness to drive, it is normally expected that the report will come from the patient's general practitioner, and it would be assumed that the G.P. would be in possession of all facts concerning the case. In the case of ischaemic heart disease, where fitness to drive may improve over the months after clinical recovery, a certificate of fitness to drive would be required by the insurers from the patient's doctor stating that the condition had improved to the extent where driving a vehicle would not be abnormally hazardous. Equally, the insurers would be expected to be notified of any decline in the patient's condition which would affect fitness to drive. Insurers require only an affirmative opinion from a medical practitioner regarding the patient's ability to drive. The extent to which a doctor feels it necessary to examine a patient and to consult with specialists, etc., is entirely a matter of opinion for the doctor in individual cases. It follows from this that the fee which a doctor charges a patient for such a professional opinion would reflect the amount of work, time, and consultation necessary for him to produce such an opinion.

Because they have a duty placed upon them to provide insurance cover for the motoring public insurers feel that they must satisfy themselves at all times regarding the level of fitness of a driver, and it is to this end that it is necessary for them to require their policy-holders to seek medical advice. In many cases, while insurers are prepared to provide insurance cover they may wish—and indeed they have the right—to impose special terms and to charge higher than normal premiums.

With regard to the certificate of ability to drive mentioned in Dr. Rockley's letter, may I say that this certificate, which is couched

in the affirmative form, has been the subject of discussion and agreement between insurers and the B.M.A.—I am, etc.,

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British Insurance Association

London E.C.4

Prophylaxis of Postoperative Deep Vein Thrombosis

SIR,—We are obliged to Mr. F. S. A. Doran (24 May, p. 442). He has done as much as anyone else to popularize electrical calf muscle stimulation for the prevention of postoperative deep vein thrombosis. We also thank Mr. J. A. Lewis (5 July, p. 41) for pointing out the superiority of random number tables (which we now use). The method of randomization in the published trial (22 March, p. 649) based on the month of birth of the patient produced an unfortunate excess of patients in the control group. This made the figures clumsily uneven but it can surely not be claimed that it was anything but strictly random or that the method of prophylaxis used was determined by our preference and not purely by chance.

Mr. Doran's labour in calculating χ^2 of the stimulator group against the heparin group has been largely wasted. He has added together laparotomies for benign disease, laparotomies for malignant disease, and operations on the bladder and prostate and has produced a χ^2 of 4.924—favouring heparin with a probability of over 95%. If he had taken laparotomies for benign disease alone and compared the two groups he would have found a χ^2 of 0.875 (no significant advantage of heparin over the stimulator). In the laparotomies for malignant disease on the other hand χ^2 is 13.298 and the chance that heparin is no better than electrical stimulation is less than one in 1000.

He makes another statistical mistake twice. First, in comparing major deep vein thrombosis (D.V.T.) between the two groups. The total number of cases of major D.V.T. in the stimulator group was five and in the heparin group one. Add these together and divide by two and the total expected number is three. No statistician is prepared to apply the χ^2 test (or indeed any other test) when the expected number is less than four. Similarly, four haemorrhages in the heparin group against none in the stimulator group gives an expected number of two and, again, cannot be analysed statistically. To put it another way, if you toss a penny four times there is a strong possibility that it will come down heads on each occasion.

Twenty-two patients were withdrawn from the trial. There were four deaths, two in the stimulator group, one in the control group, and one in the heparin group. Their details are as follows: (1) Stimulator. A man of 80 died two days after laparotomy, suture of a caecal perforation, and transverse colectomy for an obstructing carcinoma of sigmoid colon. Overwhelming sepsis. No necropsy. (2) Stimulator. A man of 76 died two days after gastrectomy, splenectomy, and transverse colectomy for advanced carcinoma of the stomach. Superior mesenteric artery occlusion. No necropsy. (3) Control. A woman of 71 died three days after gastrectomy and transverse colectomy for gastric reticulosis. Cardiac and respiratory failure. No necropsy. (4) Heparin. A man of 74 died four days after right hemicolectomy for a perforated carcinoma of the caecum. Uraemia and bronchopneumonia. Necropsy showed no pul-

¹ McNicol, K. N., and Williams, H. E., *British Medical Journal*, 1973, 4, 7.

² Hill, D. J., et al., *Archives of Disease in Childhood*, 1972, 47, 874.

³ Weinberger, M. M., and Bronsky, E. A., *Journal of Pediatrics*, 1974, 84, 421.

⁴ Williams, H. E., and Phelan, P. D., *Respiratory Illness in Children*, p. 116. Oxford, Blackwell Scientific, 1975.

⁵ Phelan, P. D., and Stocks, J. G., *Archives of Disease in Childhood*, 1974, 49, 143.