The complete analysis of the results consists of 27 tables, but obviously a selection had to be made for our paper.

JOHN GADDIE
C SKINNER
K N V PALMER

¹ Ciba Foundation Guest Symposium, *Thorax*, 1959, 14, 286.

² D'Souza, M F, *et al*, *Clinical Allergy*, 1973, 3, 177.

³ McAllen, M A, Assem, E S K, and Maunsell, K, *British Medical Journal*, 1970, 1, 501.

⁴ Skinner, C, and Palmer, K N V, *Thorax*, 1974, 29, 574.

⁵ Pride, N B, *British Journal of Diseases of the Chest*, 1971, 65, 135.

should distinguish vomiting due to thyrotoxic hypercalcaemia, vomiting due to thyrotoxicosis per se, and their coincidences. In the series of Gordon *et al*² vomiting was found with equal frequency in both normocalcaemic and hypercalcaemic groups (about 30% each). In fact, the only distinguishing symptoms of the hypercalcaemic group were apathy and constipation.

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¹ Rønnev-Jessen, V, and Kirkegaard, C, *British Medical Journal*, 1973, 1, 41.

² Gordon, D L, *et al*, *American Journal of the Medical Sciences*, 1974, 263, 31.

³ Baxter, J D, and Bondy, P K, *Annals of Internal Medicine*, 1966, 65, 429.

⁴ Rose, E, and Boles, R S, *Medical Clinics of North America*, 1953, 37, 1715.

⁵ Twycross, R G, and Marks, V, *British Medical Journal*, 1969, 2, 701.

⁶ Harper, P S, and Osborne, Hughes, R, *British Medical Journal*, 1970, 1, 213.

⁷ Guyer, P B, *British Medical Journal*, 1965, 1, 169.

⁸ Richards, A J, *Postgraduate Medical Journal*, 1970, 46, 440.

⁹ Adams, P H, *et al*, *Quarterly Journal of Medicine*, 1967, 36, 1.

Risk of venepuncture

SIR,—We have recently had a peculiar complication of antecubital venepuncture. A 67-year-old man was admitted because of suspected pulmonary emboli. He received intravenous heparin intermittently. On the sixth day of therapy, after an antecubital venepuncture for diagnostic purposes, a hematoma of 6×8 cm developed, and he began to complain of severe pain with median distribution. In two weeks he presented a clear-cut carpal-tunnel syndrome without any signs of bleeding in this region. After six weeks he was operated on and the nerve was found to be compressed by a tiny blood clot. His state improved but permanent damage remained. It must be assumed that this small amount of blood wandered along the nerve from the antecubital region to the carpal tunnel, where it coagulated and showed its adverse effect.

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ASIA BUSH
ISIDOR KESSLER

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Thyrotoxic vomiting

SIR,—I wish to dispel two myths regarding vomiting in thyrotoxicosis.

Firstly, it would appear from the paper by Dr F D Rosenthal and others (24 July, p 209) and from the subsequent correspondence that vomiting in patients with thyrotoxicosis is rare. This is not so. Rønnev-Jessen and Kirkegaard¹ reported vomiting in seven out of 28 patients over 60 years of age. Gordon *et al*² in a prospective study found thyrotoxic vomiting in 35 of 121 patients. The omission from textbooks of any mention of vomiting as a symptom of thyrotoxicosis is to be blamed for the general unawareness of this symptom, since diagnosis is based on mental processes trying to find the closest match between the patient and the textbook "disease."

The second point, touched upon by Dr P H Sanderson (4 September, p 584), is the association of thyrotoxic hypercalcaemia with vomiting. Although hypercalcaemia in thyrotoxicosis is as common as in sarcoidosis (about 17%),^{2,3} it has had the reputation of a rare complication for the same reasons as for vomiting. Rose and Boles⁴ and Twycross and Marks⁵ each reported three patients with thyrotoxic hypercalcaemia, and all six patients also had vomiting. Single cases of combined hypercalcaemia and vomiting in thyrotoxicosis were described by Harper and Hughes,⁶ Guyer,⁷ and Richards.⁸ While both hypercalcaemia and vomiting in thyrotoxicosis are unrelated to the severity of the disease,^{2,3,9} vomiting may also be a symptom of hypercalcaemia. Thus we

SIR,—I wonder if the vomiting described by Dr F D Rosenthal and others (24 July, p 209) might be due to too rapid ingestion of large amounts of food. I have observed this mechanism in one patient with gross thyrotoxicosis. Treatment with carbimazole resulted in more leisurely eating *pari passu* with cessation of vomiting.

B I CHAZAN

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Coronary artery disease and coalworkers' pneumoconiosis

SIR,—I write to support Dr Dewi Davies (16 October, p 925). It seems that the DHSS have drawn sweeping conclusions from the articles quoted, which are by no means proved. Even the authors themselves only postulate certain possible explanations. Unfortunately the DHSS loftily expresses its views as based on "authoritative opinions," suggesting that nobody else has any authority or is worth considering, and by incorporating these in "case law" wrongly turns a theory into a proved fact, failing to realise that one of the conclusions which could be drawn from the articles quoted is that death from coronary thrombosis can be avoided by tying a ligature around one's neck to obstruct the airway and then arranging to die from something else at the earliest possible moment.

We do not know all about the cause of chronic bronchitis or emphysema or why some cases of pneumoconiosis progress to progressive massive fibrosis. We do not know the cause of coronary atheroma or why some cases develop thrombosis which may or may not lead to myocardial infarction. How, in the midst of all this ignorance, can we produce "authoritative statements" on the interaction of the two conditions?

In practice these statistical surveys mean little. One is faced with the problem of a patient who has in fact developed both myocardial infarction and pneumoconiosis and died. I do not think that anybody can say that pneumoconiosis did not play some part in the cause of death or that it did not have any effect on the heart because I do not believe that anybody can say whether a patient with myocardial

infarction will recover or not. I do a large number of post-mortem examinations and it is quite obvious that many, many people, often dying from unrelated conditions, have had previous extensive myocardial infarction which has completely healed without the patient having made any serious complaints or requiring treatment.

It seems to me, therefore, that in the light of our present proved knowledge Dr Davies's opinion is the only reasonable one and, indeed, the only humane one. There is something rather objectionable about a government department using the whole of its weight to sweep aside the claim of a widow whose husband probably died as the result of working and at the same time hastening to distribute money to drug addicts and the like.

G S ANDREWS

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Professional standards

SIR,—As a practising surgeon in a district hospital I find myself becoming increasingly concerned about the professional standards of my recently qualified colleagues. I get the impression that their undergraduate training has concentrated on the "scientific" rather than the clinical and practical aspects of medicine. Many do not appear to have learnt how to take a proper history, how to perform a proper clinical examination, and most have virtually no experience of practical procedures.

Perhaps even more serious is their lack of "professionalism." I do not refer solely to the matter of dress and address of colleagues (and other staff) in front of patients, but mainly to their attitude towards their job and patients. Too often they seem to lack interest, inquisitiveness, and a sense of personal responsibility to patients under their care, even if their contact with the patient is of brief duration. Many do not understand that every doctor has a legal responsibility to take "due care" within the limit of his knowledge and competence: they do not appear to have been taught what constitutes "due care."

The practice of medicine is based on compassion and caring; all possible and foreseeable risk to the patient—not the doctor—should be eliminated so far as is practical. In other words, as is implied in the word professionalism, we must strive for, even if we cannot achieve, perfection. We cannot condone any fall in the standard of care given to our patients. There is only one yardstick by which such care should be measured. Would we like to be on the receiving end?

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Traumatic rupture of ear drum

SIR,—Recently I saw an overseas student who had just sustained a traumatic rupture of his ear drum from a cause I have not met before. His girl friend had inserted her tongue into his auditory meatus for his delight and when she removed it he experienced a sharp pain which had persisted. Examination showed a traumatic perforation of the drum with haemorrhage. Presumably the tongue had

been slowly inserted, expelling the air gradually. When the time came for removal it was fitting the meatus closely and removal exerted considerable negative pressure. Perhaps "the champagne cork syndrome" would be a suitable name for the injury.

D McCracken

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Direct translation

SIR,—In your leading article entitled "Beyond Calais" (11 September, p 606) you state that direct translation is often impossible and different nations often claim the same syndrome for their own compatriots. You give as an example hyperthyroidism, which is known in German as "Glotzaugenkrankheit." I have not met with this designation in my long years of medical practice. On the contrary the designation "Basedow-Erkrankung" is used by us, because C A von Basedow was a German doctor and had worked in Merseburg (1799-1854). Therefore we speak also of the "Merseburg triad"—namely, exophthalmos, struma, tachycardia.

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Oculo-mucocutaneous reactions to β -adrenoceptor antagonists

SIR,—Dr R G Finch (16 October, p 946) questions my statement (31 July, p 289) that "a validated case of oculo-mucocutaneous syndrome associated with practolol treatment has not yet been reported with any other beta-blocker," and quotes two supporting references.

No one denies that skin problems or eye symptoms may develop in patients receiving treatment with beta-blockers other than practolol.¹ However, proof of a "true-bill" oculo-mucocutaneous syndrome² similar to that associated with practolol has not been validated, so far as I am aware, in any patient on oxprenolol, or propranolol, at the time of writing.

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¹ Petrie, J C, *et al*, *Postgraduate Medical Journal*, 1976, 52 (suppl 4), 63.

² Wright, P, *British Medical Journal*, 1975, 1, 595.

Misuse of tubular elasticated bandages

SIR,—I read with interest Mr C V Ruckley's condemnation of the increasing use of untapered tubular elasticated bandages for everything from dressings to fractures (16 October, p 940).

This summer I injured my left little finger playing cricket, and subsequent radiography showed a fracture of the terminal phalanx. I was immediately expertly cared for by a colleague who applied a tubular elasticated bandage to hold a dressing in place. In spite of keeping my hand up to help reduce swelling an intense throbbing pain developed over the next three hours, and I could feel the dressing becoming tighter and tighter on my little

finger. Great relief was obtained by removing the tubular elasticated bandage and re-bandaging using the left ring finger as a splint.

Perhaps the use of tubular elasticated bandages with their inherent property of radial compression in the immediate management of finger injuries should be questioned and investigated further.

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In vitro lymphocyte reactivity during pregnancy

SIR,—Many in vitro studies on lymphocyte reactivity during pregnancy have been reported in your journal.¹⁻⁴ There is still some controversy about the part played by maternal serum blocking factors, because conflicting results have been obtained when lymphocytes were cultured in the presence or absence of autologous sera. We would like to report that even thoroughly washed lymphocytes have demonstrable serum proteins on their surfaces. It is possible that these adsorbed surface proteins, if they contain blocking factors, could modify in vitro lymphocyte reactivity in the absence of additional autologous sera.

Lymphocytes were obtained from defibrinated venous blood of pregnant and non-pregnant women as previously described.⁵ Crossed immunoelectrophoresis⁶ using an LKB Multiphor system was performed on: (1) freshly isolated lymphocytes; (2) lymphocytes which had been kept overnight in autologous serum at 37°C, room temperature, and 4°C; and (3) lymphocytes which had been kept in liquid nitrogen in a DMSO/autologous serum mixture. The cells were washed three times and six times before immunoelectrophoresis. The first dimension was performed with 20 microlitres of a 10⁷/ml concentration of lymphocytes for 35 minutes at 8-10 V/cm. Following this, viability of the cells was checked by removing the cells from the wells and staining with trypan blue. The second dimension was run overnight at 2 V/cm against 1% anti-whole human serum incorporated in the gel.

Precipitin arcs could be seen in all cases but were particularly numerous with lymphocytes which had been kept in autologous sera at 37°C, room temperature, 4°C, and in liquid nitrogen. Serum proteins were present in substantial quantities after three washes, which is the usual number employed in most experimental procedures, and traces were still demonstrable after six washes. This occurred with pregnant and nonpregnant lymphocytes. Most lymphocytes (average of 76%) were alive after the first dimension electrophoresis, as judged by trypan blue exclusion. This high viability count, together with the gradual disappearance of serum proteins with increasing number of washes, indicate that these proteins were from the lymphocyte surface rather than liberated from disrupted cells.

Inhibitory factors in pregnancy serum appear to be effective in very low concentrations⁷ so a small amount bound to the lymphocyte surface could be sufficient to alter its in vitro reactivity. It has also been suggested that the immunosuppressive activity of pregnancy serum globulins acts upon the earliest events of lymphocyte response at the time of antigen or mitogen recognition, possibly by competition for receptor sites,^{8,9} so the presence of these globulins on the

lymphocyte surface at the beginning of culture could bring about a depressed reactivity. Thus it would seem that serum blocking factors cannot entirely be excluded even in those experiments where lymphocytes were cultured without autologous sera, and the conflicting results reported could be influenced by the amount of serum proteins left on the lymphocyte surface.

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G E ALLEN

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¹ Curzon, P, *et al*, *British Medical Journal*, 1972, 4, 49.

² Finn, R, *et al*, *British Medical Journal*, 1972, 3, 150.

³ Walker, J S, *et al*, *British Medical Journal*, 1972, 3, 469.

⁴ St Hill, C A, *et al*, *British Medical Journal*, 1973, 3, 513.

⁵ Loke, Y W, *et al*, *American Journal of Obstetrics and Gynecology*, 1975, 122, 561.

⁶ Weeke, B, *Scandinavian Journal of Immunology*, 1973, 2, suppl 1, 47.

⁷ Gatti, R A, *et al*, *Clinical and Experimental Immunology*, 1973, 13, 427.

⁸ Cooperband, S R, *et al*, *Transplantation Proceedings*, 1969, 1, 516.

⁹ Yachnin, S, *Journal of Immunology*, 1972, 108, 845.

Intravenous and aerosol salbutamol

SIR,—Drs M R Hetzel and T J H Clark (16 October, p 919), comparing intravenous and aerosol salbutamol, showed that aerosol salbutamol had a longer bronchodilatory effect. They suggest that this discrepancy may be due to the slow removal of salbutamol from the bronchial mucosa or its inhibition of mediator release. This difference may also be due to the differing metabolism of salbutamol when administered via these routes.

Studies by Evans *et al*¹ on the metabolism of salbutamol showed that salbutamol is conjugated in the liver and that this metabolite had negligible beta-adrenoreceptor stimulant activity. Following intravenous salbutamol 27% of the drug excreted in the urine over 24 h had been metabolised. However, when salbutamol was introduced directly into the lungs through a bronchoscope most of the dose in the plasma and urine was present as free salbutamol, suggesting that it is not metabolised in the lung. Their study also showed a more rapid urinary excretion rate for intravenous salbutamol, with 50% dose excretion by four h compared with only 25% dose excretion by four h for aerosol salbutamol. The longer bronchodilatory effect of aerosol salbutamol therefore reflects the higher proportion of active drug in the lungs and its slow removal from the lungs.

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¹ Evans, M E, *et al*, *Xenobiotica*, 1973, 3, 113.

Can hepatitis B be transmitted sexually?

SIR,—If your expert seriously considers sexual contact to be an example of "non-parenteral" infection (28 August, p 517) he must surely be preoccupied with "unnatural practices." The opposite of "parenteral" is more concisely expressed as "enteral" than by the clumsy and ambiguous term "non-parenteral."^{1,2} Perhaps your expert was really thinking of inapparent parenteral transmission, for which, of course,