In their later papers, Truelove, Korelitz, and Palmer have consistently cited Palmer-Kirsner as the supreme authority but shut their eyes to the presteroid death rate in colitis of less than 2% per year.⁷⁻⁹ Patients seldom died from colitis; nearly the biggest worry was the possibility of cancer of the bowel.

From the introduction of steroid therapy physicians^{10, 11} and surgeons^{12, 13} have reported many deaths from perforation, anaesthetic shock, and infections; many patients with colitis became addicted to steroids. Later, surgeons were still more emphatic¹⁴⁻¹⁷; Brooke¹⁸ has written six condemnatory papers since 1956. Of Sauer's¹⁹ 52 patients after 10 years nine are addicted and four others went insane.

Lately Kellner²⁰ has reported three post-operative deaths in 21 patients treated with steroids compared with no deaths in 21 patients not treated in this way. Ross,²¹ a surgeon who is not in favour of steroid treatment, has had no deaths in 132 colitis patients; four developed peptic ulcers, "surely another argument against the indiscriminate use of steroids."

A recent semi-official pronouncement by Truelove²² says, "Corticosteroid therapy has been shown in a number of controlled clinical trials to increase the chance of obtaining a rapid clinical remission." A more relevant statistic for most doctors would be the addiction and death rates of patients who have had steroids.—I am, etc.,

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REFERENCES

- Edwards, F. C., and Truelove, S. C., *Gut*, 1963, 4, 299 and 309.
- Truelove, S. C. et al., *Brit. med. J.*, 1959, 1, 387.
- Banks, B. M., Korelitz, B. I., and Zetzel, L., *Gastroenterology*, 1957, 32, 983.
- Zetzel, L., and Atin, H. L., *Amer. J. Dig. Dis.*, 1958, 3, 916.
- Kirsner, J. B., Palmer, W. L., Spencer, J. A., Bicks, R. O., and Johnson, C. F., *Ann. intern. Med.*, 1959, 50, 891.
- Spencer, J. A., Kirsner, J. B., Mlynaryk, P., Reed, P. I., and Palmer, W. L., *Gastroenterology*, 1962, 42, 113.
- Sloan, W. P., Bergen, J. A., and Gage, R. P., *ibid.*, 1950, 16, 25.
- Morrison, L. M., *J. Amer. med. Ass.*, 1953, 151, 366.
- Svartz, N., *Gastroenterology*, 1954, 26, 26.
- Sloan, S., Briggs, J. D., and Halsted, J. A., *ibid.*, 1951, 18, 438.
- Sauer, W. G., Dearing, W. H., and Wollaefer, E. E., *Proc. Mayo Clin.*, 1953, 28, 641.
- Pessel, J. F., Beairisto, E. B., Edwards, M. H., Rathmell, T. K., and Wise, J. S., *Amer. J. Dig. Dis.*, 1953, 20, 318.
- Wilson, D. A. W., and Roth, D., *J. Amer. med. Ass.*, 1953, 152, 230.
- Hayes, M. A., *Surgery*, 1956, 40, 945.
- Gumpel, R. C., Lewis, E. C., and Thomson, R. V., *Arch. intern. Med.*, 1957, 99, 660.
- Goligher, J. C., *Medical Annual*, 1958, p. 61. Wright, Bristol.
- *Brit. med. J.*, 1961, 1, 151.
- Brooke, B. N., *Lancet*, 1956, 2, 1175.
- Sauer, W. G., and Maki, S. W., *Arch. intern. Med.*, 1961, 107, 88.
- Kellner, H., Hersch, R. A., and Barker, W., *Gastroenterology*, 1963, 45, 27.
- Ross, S. T., *Surgery*, 1964, 55, 782.
- Truelove, S. C., *Prescriber's J.*, 1964, 4, 24.

*. Most of the figures quoted by Dr. Kelly are extremely difficult to evaluate; the only analysis with significant meaning is that which relates to patients admitted or treated for their first attack of colitis and then followed up subsequently. This has been done in this country both by Truelove and Edwards,¹ and by Kellock² at Central Middlesex Hospital. Their findings convincingly demonstrate that ulcerative colitis in the pre-steroid era was far from being the

negligible threat that Dr. Kelly implies. He quotes a pre-steroid death rate of about 2% per year, yet this is not only several times the expected value but also overlooks a mortality rate greatly in excess of 2% in patients with a first attack within one year of referral to hospital.

Since 1952, when cortisone was first introduced into treatment at Oxford, Truelove has found a notable improvement in the fatality rate and several controlled trials have confirmed the beneficial effects of steroids. Such treatment certainly carries its own risks, not the least of which is the steroid servitude mentioned by Dr. Kelly, but the application of these methods appears to have provided a much more hopeful prognosis than formerly existed.—ED., *B.M.J.*

REFERENCES

- Truelove, S. C., and Edwards, F. C., *Gut*, 1963, 4, 299.
- Kellock, T. D., and White, B., *Gastroenterologia*, 1957, 88, 13.

Local Analgesics

SIR,—In your interesting article in "To-day's Drugs" on local analgesics (28 November, p. 1380) your contributor says that lignocaine "falls short of the ideal because . . . serious, even fatal, side-effects can result from overdosage." Surely all local analgesics have a toxic effect, especially on nerve tissue, and it is difficult to imagine any such drug without side-effects when used in excess dosage.

In this connexion, it is remarkable that local analgesics such as lignocaine can produce convulsions in overdosage, and that the same drugs given as an intravenous infusion can terminate existing convulsions such as those occurring in status epilepticus. The same effect has been shown in cats and monkeys when the exposed cerebral cortex was stimulated electrically to cause convulsions. This apparent paradox has been known for many years, and it would be interesting to hear a reasonable explanation.—I am, etc.,

C. LANGTON HEWER.

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London E.C.1.

SIR,—In your expert's review of local anaesthetics, under "To-day's Drugs" (28 November, p. 1380), it is stated that lignocaine is the nearest available approach to an ideal local anaesthetic drug and that there is little reason to use the newer local analgesics.

To my mind, amethocaine has many advantages over it. One of these is that a large volume of effective fluid can be injected, even feeble subjects tolerating over 400 ml. of 1/2,000 solution with adrenaline. This is a distinct advantage if an abdominal operation such as gastrectomy is to be carried out—or a radical mastectomy, or a thoracotomy—and there are occasional indications for local anaesthesia for these procedures. The latent period before anaesthesia develops is short, for if the surgeon, having finished the injection, prepares and towels the part, the operation can be started at once. The duration of anaesthesia is such that I have on one occasion injected the amethocaine solution, then gone away for lunch, and returned to

carry out a radical gastrectomy for cancer without further anaesthesia of any sort being required.

With regard to toxicity, we have used the drug as a 2% solution or as a tablet for every gastroscopy for over 25 years, certainly more than 12,000 times, without any reaction. In addition we have used it in a 1/2,000 solution for abdominal-wall anaesthesia and for thoracotomy well over 4,000 times without any reaction. The only reaction I have seen from the drug was when a non-sterile 2% solution was injected into the tissues by a new house-officer unfamiliar with the drug, but of course most local anaesthetics are dangerous if given in 40 times the correct strength.

Its only disadvantage is its instability in solution. This is, in fact, an advantage, for we keep it as a dry powder, 100 mg. per ampoule, and make up each solution fresh in normal saline. The ampoules are extremely convenient for use abroad. They are much more portable than flasks of lignocaine.

I regret very much that the English drug firms are giving up the manufacture of this safe and useful drug, and that we are compelled to explore abroad for it.—I am, etc.,

London W.1.

NORMAN C. TANNER.

Cephaloridine in Typhoid

SIR,—By courtesy of Professor J. W. S. Blacklock and Dr. J. P. Currie of Glaxo Research Limited, five patients with typhoid fever in this year's epidemic in Aberdeen were treated with the new broad-spectrum antibiotic cephaloridine (Dr. P. W. Muggleton and his colleagues, 14 November, p. 1234; Dr. J. McC. Murdoch and his co-workers, 14 November, p. 1238). The minimal inhibitory concentration *in vitro* for the organism (phage-type 34) causing the epidemic lay between 5 and 6 µg./ml., at which level there was virtually 100% kill (Dr. Currie, personal communication).

Three patients with moderate or severe typhoid were started on cephaloridine in the recommended dosage within an hour or two of admission, one was treated with it for a second relapse, and one at the end of a seven-weeks illness which had shown no lasting response to either chloramphenicol or ampicillin.

The first patient, a woman aged 26 admitted in her third week of illness, was one of the relatively few patients in the epidemic who were dangerously ill. On admission her temperature was 104° F. (40° C.), her abdomen distended and tender, and toxæmia severe. Cephaloridine 0.5 g. intramuscularly was given twice on the first day, and on the second day increased to 0.5 g. six-hourly. Fluids were given intravenously. On the afternoon of the third day, when 4 g. of cephaloridine had been given, her temperature was 103° F. (39.9° C.) and her condition had worsened; blood culture, reported later, was positive. Persistence with an untried drug was obviously not justified and she was started on chloramphenicol 3 g. daily with an intravenous loading dose, and intravenous hydrocortisone six-hourly for two days. She improved steadily and was afebrile on the sixth day of chloramphenicol. Convalescence was uneventful.

A woman aged 49 was admitted at the end of her first week, moderately ill with a temperature of 104.6° F. (40.3° C.). Cephaloridine was given in a dose of 0.5 g. six-hourly. On the fourth day of this treatment, when 6 g. of cephaloridine had been given, her temperature was 104° F. (40° C.), and her condition if any-

thing worse. Treatment was changed to chloramphenicol 3 g. daily following an intravenous dose of 1 g. She was afebrile after four days of chloramphenicol. Blood culture taken on the day after cessation of cephaloridine was positive.

A woman aged 47 was admitted in the first week of illness with a temperature of 104° F. (40° C.). Blood culture, positive two days before, was negative on the day of admission. Cephaloridine 0.5 g. six-hourly was given to a total of 5 g. over three days without effect on her pyrexia or general condition, though blood culture remained negative. She responded in four days to chloramphenicol 2 g. daily.

A girl aged 16 had a second clinical relapse lasting three weeks, with fever reaching 102° F. (38.9° C.), but only mild discomfort. Blood culture, positive earlier, was negative throughout this relapse. In the course of it she received 10 g. of cephaloridine over five days without apparent effect, though the pyrexia began to subside two days after its cessation. In this patient serum levels of cephaloridine, estimated in the City Hospital laboratory, ranged from 7 to 15 µg./ml. at one to two hours after an injection of 0.5 µg. to less than 1 µg./ml. at six hours.

A woman aged 30 had a severe illness lasting seven weeks and showing little if any response to chloramphenicol or ampicillin other than an allergic rash and monilial infection. A 10-day course of cephaloridine totalling 20 g. coincided with defervescence and general improvement, but these had probably begun before this drug was started. After discharge her stool was positive for *Salmonella typhi*.

My thanks are due to Dr. C. D. Needham, Dr. D. S. Short, and Dr. J. M. Stowers for their ready co-operation in the trial of cephaloridine in four of these patients.

—I am, etc.,

City General Hospital,
Aberdeen.

W. WALKER.

Cephaloridine

SIR,—A point in your leader (14 November, p. 1211) seems to invite comment. As regards broad-spectrum activity cephaloridine appears to show a relatively narrow spectrum *in vitro* against the common urinary pathogens. The figures published as Table I by Dr. P. W. Muggleton and his colleagues in the same issue (p. 1234) show that of these only *Proteus mirabilis* and *Escherichia coli* have a minimum inhibitory concentration (M.I.C.) less than 100 µg./ml.

It is interesting to note that of 26 strains of proteus reported in this Table 18 were all one species, *P. mirabilis* (M.I.C. 25 µg./ml. or less). Of the other three species, only four *vulgaris*, three *morgani*, and one *rettgeri* were tested, all of which showed an M.I.C. of more than 100 µg./ml. (How much more is not stated.) This is loading the dice, and a better picture of the *in vitro* sensitivity of proteus would have been given by reporting the M.I.C. for 18 strains of each species.

Similarly, only one strain of *Streptococcus faecalis*, and four of *Pseudomonas pyocyanea* were tested (all with M.I.C. more than 100 µg./ml.) Why, then, were 25 strains of *E. coli* reported (all with an M.I.C. of 25 µg./ml. or less)?

Can this loading be based on relative frequency of isolation from clinical material? Or is the spectrum of cephaloridine *in vitro* less broad than your leader suggests?—I am, etc.,

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P. W. KIPPAX.

** Dr. Kippax has ignored the remark in the leader that Barber and Waterworth (8 August) give fuller information about activity

against coliforms. These authors tested roughly equal numbers of strains of *Proteus* species, etc., and show that cephaloridine is active against penicillinase-forming *P. mirabilis* and two species of *Klebsiella*, all of which are resistant to ampicillin. If ampicillin is a broad-spectrum antibiotic, and it has always been so described, then cephaloridine is rather more so.

What he calls "loading the dice" can rightly be said to be based on "relative frequency of isolation." After all about 80% of clinical isolates of *Proteus* are *P. mirabilis*, and this organism and *E. coli* are the commonest causes of urinary tract infection.—Ed., B.M.J.

Accidental Hypothermia in the Elderly

SIR,—The excellent B.M.A. report on accidental hypothermia in the elderly and the B.M.J.'s leading article (14 November, p. 1255) query the incidence of body temperatures between 90–95° F. (32.2–35° C.). My experience during the last two winters shows that body temperatures of all old people, when there is no other illness, fall below 95° F., and some below 90° F., if the air temperatures of their bedrooms and kitchens are freezing, or below. And this happens to very many of them in winter. These low body temperatures rise to near normal by the evening, if the air temperatures of their living-rooms are 60° F. (15.6° C.) or over. If their day living-rooms are cold, as they often are in severe cold weather, their body temperatures remain low while cold conditions last. With low body temperatures their pulse rate is slow, returning to normal with normal temperatures.

Many newly built old people's bungalows have badly fitting doors and windows, are not insulated, and have grossly inadequate heating arrangements. In contrast, some old country and miners' cottages are warm, their walls are thick and windows small; the bedroom is warm from the living-room below.

The B.M.A. report refers to the National Assistance Board's extra fuel winter allowance. This was 5s. weekly, less than half a cwt. (0.025 metric tonnes) of coal, and only 500,000 of the nearly three million living at the national assistance level apply for it.

I think the B.M.A. report underestimates the extent of protein, vitamin C, and B group of vitamin deficiencies. Angular stomatitis, glossitis, myoedema, and anaemia are common in the elderly at national assistance levels. They cannot afford sufficient meat, fish, fruit, and vegetables in the winter. Advertising tends to make them buy biscuits and over-refined carbohydrates in glossy packs.

The B.M.A. suggest that "clothing needs to be light in weight, closely woven, and not restricting." In fact many of the poorer old-age pensioners rarely buy new clothes. They cannot afford them. Their clothing is often old, matted, shrunken, and constricting. Their bedclothes have often lost their warmth through age. Some have difficulties in drying their bedclothes in winter. Some are incontinent.

The B.M.A. recommends a thermometer for general use recording from 75 to 105° F. (23.9 to 40.6° C.). A scale from 75 to 110° F. (23.9 to 43.3° C.) will be better. For example, in the N.W. Frontier Province and the Punjab (where I worked for many

years) temperatures above 105° F. are not uncommon. Looking back, hypothermia was often seen there but not recognized.

To solve the problems of hypothermia we need national surveys of old people's houses and the extent of their malnutrition. A standard of warmth is needed similar to that for offices and factories of a minimum of 61° F. (16.1° C.). The Ministry of Housing suggests 70° F. (21.1° C.) for old people's rooms. This is a striking contrast to the existing freezing conditions of many old people's bedrooms and kitchens. The lowest minimum temperature I have recorded was 7° F. (–13.9° C.)—25° F. (14° C.) of frost on a newly built bungalow in South Somerset. Establishing a standard of warmth will revolutionize our attitude to existing and future houses.

Is it beyond the power and will of the British people to ensure that our old people are warm and adequately fed this winter? Must we continue to ignore freezing conditions and allow millions of old people to be cold, from which some die each winter?—I am, etc.,

Ilminster, Somerset.

GEOFFREY TAYLOR.

Surgery for Peptic Ulcer

SIR,—In discussing tests for completeness of vagotomy (24 October, p. 1070) J. P. Lythgoe quotes my findings in respect of the variations of the coeliac branch of the posterior vagus nerve. I believe that the surgical conclusions he draws from these findings are unjustified.

The relevant variations were, firstly, that there may be two coeliac divisions, one of which may deviate to the right, coming to lie on the right crus of the diaphragm, while the other adheres to the left gastric artery, and, secondly, that there may be a high bifurcation of the posterior trunk.

I do not presume to instruct Mr. Lythgoe in the technique of vagotomy, but I would point out that when the lower oesophagus is properly mobilized for selective or total vagotomy it should be possible both to draw into the abdomen a little of the lower thoracic oesophagus with its adjacent nerves, and also to pass a finger two or three inches up through the hiatus into the chest. In practice, the simplest way of finding the coeliac branch is to slide the tip of a finger down the posterior aspect of a posterior vagal trunk. The finger is automatically deviated to the coeliac axis by the coeliac branch.

I am sorry if Mr. Lythgoe has been misled by the description "high bifurcation." Neither in my own series nor, to my knowledge, in any other published account has a coeliac branch been observed to arise above the oesophageal hiatus. It follows that a finger, electrode, or any other appropriate device can be passed around the oesophagus and all abdominal vagal fibres in every case, and that neither of the variations mentioned by Mr. Lythgoe alters this fact.

I was interested to learn from Mr. Hopton's letter of the same issue that vagal fibres destined for the alimentary tract may run "amongst the fibres of the right crus." I should be interested to learn of the evidence for this statement.—I am, etc.,

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C. V. RUCKLEY.