required a very much larger clinical trial. This is exactly the cause of the equivocal results obtained by Mr. G. E. Moloney and his colleagues (24 October, p. 244).—I am, etc.,

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1 Doran, F. S. A., Drury, M., and Sivyer, A., *British Journal of Surgery*, 1964, **51**, 486.
2 Doran, F. S. A., and White, H. M., *British Journal of Surgery*, 1967, **54**, 686.

Cholera Again

SIR,—I venture to disagree with some of the points in your leading article on "Cholera Again" (3 October, p. 2). The 5/4/1 Dacca solution can be used only for cholera or choleraic dysentery. It is absurd to stockpile specialized fluid of this kind when equally good results are obtainable by the use of lactated Ringer (Hartmann's solution), provided that the extra potassium and alkali needed for full replacement therapy are given orally, either as a four-hourly or six-hourly dose of KC7, 3 g, and $NaHCO_3$, 4·5 g. As lactated Ringer is used for the intravenous therapy of many conditions, it is widely available, and any stock of it can be sure to be turned over regularly as it is used and replaced, thus obviating deterioration as the result of lengthy storage.

It is unnecessary and often actually misleading to use the measurement of plasma specific gravity as an indication of the intravenous fluid requirements. So easy is it to see when the patient has been given enough fluid that we usually delegate the decision when to stop to the nursing staff—after they have had a little supervision over the first case or two—and they have no difficulty in deciding correctly. Surely what our nurses can do a physician in England can also do? I deprecate forsaking observing physical signs in exchange for reading laboratory reports.

My account[1] of the 1965 Brunei cholera epidemic may be of assistance to those who wish to know more of the practical aspects of cholera therapy. A second epidemic on a smaller scale has been dealt with subsequently in an identical manner, and we have yet to see one of our patients succumb to cholera. As a matter of practical experience, therefore, the simpler methods recommended in my article are difficult to improve on.—I am, etc.,

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State of Brunei, Borneo

1 Hart, P. L. de V., *Medical Journal of Malaya*, 1966, **20**, 281.

SIR,—In your excellent and most informative editorial on the topical subject of cholera (3 October, p. 2) you comment that "the usual vaccination at present is an injection of 0·5 ml of 4,000 × 10^6 each of killed cholera vibrios of Inaba and Ogawa serotypes per ml followed 7 to 28 days later by 1 ml of the same." Those practitioners who have used the Services' cholera vaccine may be interested to know that for many months now an El Tor component, in addition to the classical Inaba/Ogawa fractions, has been incorporated in the vaccine prepared at the armed Forces own vaccine laboratory—the vaccine contains not less than a total number of 8,000 × 10^6 bacilli per ml as recommended by the World Health Organization, and can be used both for subcutaneous injection as described in your editorial and also in 0·1 ml doses for intradermal inoculation, which may suit those who suffer undue reactions after subcutaneous vaccinations.

In the past few years the ever-expanding frontiers of the El Tor epidemic have given good cause for alarm, as indicated so well by Professor B. G. Maegraith's letter (10 October, p. 114), and recently reported experience has shown that El Tor vaccines in a particular locality may have a marginal advantage over a classical-strains vaccine. One must remember that, despite the strikingly different appellation, under the facade of the biotype El Tor there really lurks one or the other of the two classical serotypes Inaba or Ogawa, and provided a vaccine contains the full quota of antigens from both these serotypes then some protection will be afforded against cholera by such a vaccine whether the prevailing strain or strains are Inaba, Ogawa, or El Tor Inaba/Ogawa. Obviously, however, nothing is lost by inclusion of an El Tor component. Another interesting facet to this problem is the indication that antigenic stability is not to be expected in members of this group, and changes from one serotype to another can apparently occur in the human gut.

The Services' cholera vaccine thus appears to get the best of all possible worlds in the present state of knowledge, until a much needed better cholera vaccine (bacterial with toxoid vaccine, or, say, attenuated oral/parenteral vaccine) arrives to supplant it.—I am, etc.,

WALLY VELLA

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Cataract Spectacles

SIR,—As an aphakic motorist I am writing to thank you for your leading article (12 December, p. 634) describing methods of overcoming the difficulties that we have because of the limitation of our visual fields. As you say, the full-aperture lens with its comparatively wide visual field does have an inconvenient peripheral ring scotoma, but this scotoma is so far out to the side that the motorist wearing full-aperture spectacles has no trouble at road junctions or in reversing.

Unfortunately, aphakic motorists are commonly prescribed "pebble" lenses which are only 3·2 cm in diameter. With these not only is the visual field very limited but their peripheral ring scotoma is dangerous as it lies so far forward that cars coming from the side suddenly appear without warning in front of the motorist approaching an intersection. Reversing is both difficult and dangerous with these small lenses. In my experience, pebble lenses do not give clearer vision than full-aperture lenses.

Full-aperture lenses should be prescribed for all aphakic motorists so that they will have as wide a visual field as possible.—I am, etc.,

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Vision and Accidents

SIR,—In your editorial (12 December, p. 634) you state that the Medical Commission on Accident Prevention says that available evidence suggests that defective visual acuity is not important in the causation of accidents. As accidents occur more frequently after dark, more frequently in foggy weather, and people are required to have approximately a visual acuity of 6/12 before being allowed to drive, it seems that a prima facie case is made out that good sight is necessary for safe driving and that present standards should not be relaxed whatever the "available" evidence is.

I feel that the "evidence" is likely to be unsound for various reasons, one being that eye tests are not performed on cadavers. An illustrative case occurred when a lady, knocked down as she stepped on to a crossing, complained ten days later that the accident had caused defective vision in her right eye. She had had no head injury, her vision in the right eye was less than 6/60, with what was obviously a cataract of some long standing present. It was clear that her previously unnoticed poor vision in the right eye, far from being the result of the accident, was contributory to it.

Unless the eyesight of all people involved in accidents, including those at night whose dark adaptation is known at the moment of impact, is recorded, I do not see how reliable statistics can be obtained.—I am, etc.,

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Gold in Rheumatoid Arthritis

SIR,—In your recent Current Practice article on rheumatoid arthritis (5 December, p. 603) the place of gold therapy was briefly reviewed. Reference was made to the possible value of estimating gold levels in biological fluids[1] and it was claimed that toxic effects are associated with excessive plasma gold values.[2]

In this connexion two patients who developed blood dyscrasias having received 810 and 780 mg Myocrisin (sodium aurothiomalate) respectively have been studied at the London Hospital. Both patients were receiving Myocrisin 50 mg by intramuscular injection at monthly intervals when the toxicity was observed. Each patient had received weekly injections of 50 mg to a total dose of 730 and 580 mg respectively, during which time serial serum gold estimations had been performed. The mean serum gold levels (taken one week after the previous injection and after the patients had received a total of 330 mg Myocrisin) were 339 and 248 μg/100 ml, while in a group of nine patients who responded to gold without evidence of toxicity the mean level was 319 μg/100 ml.

Clearly, in these two patients there was no evidence of significantly raised serum gold values. The levels of gold found in the serum vary widely depending on the size of the dose administered, the frequency of the injections, the interval after the last injection, and the total amount of gold given. If it is felt that a serum gold determination might be helpful in a patient with a genuine or suspected toxic reaction to gold a random blood sample estimation could not be interpreted correctly unless all these factors