sprouts, which arise from surviving subterminal nerve fibres and eventually make contact with the denervated muscle fibres, upon which they form new end-plates. mechanism is far easier to envisage as an explanation of gradual recovery of some months' duration than that postulated by Dr. Jackson et al., who would suggest that, if oedema of the roots is unduly severe, recovery can only take place by growth of sprouts (presumably from the intrathecal part of the anterior roots), past the scarred area where the roots have been compressed at the intervertebral foramina, and on for 2-3 ft. (60-90 cm.) down the nerves to the muscles. Accepting the figure given by Seddon et al.5 for the rate of advance of the regenerating tip of a peripheral nerve after suture (1.5 mm. per day), it will be seen that even under the best conditions, which will certainly not obtain, it would take well over a year for regenerating sprouts to travel down the nerves from the lumbar roots to the distal muscles of the leg. Add to this time the period which elapses between onset of paralysis and the subsiding of oedema in the spinal roots and it will be obvious that the sprouts, should they ever reach the muscle, would find the latter almost completely atrophied and fibrosed. The formation of an end-plate on any surviving muscle fibre would be impossible. Of course, the clinical history of these cases would be quite incompatible with such a method of reinnervation. Recovery is gradual and continuous from within a few weeks of the paralysis reaching its maximum. If the explanation just rejected were correct, recovery would suddenly set in after many months. This mechanism of axonal growth down peripheral nerves, while so acceptable in the case of complete nerve section a short distance proximal to a muscle,6 must therefore be considered as improbable in the extreme as an explanation of the usually complete functional recovery which characterizes the Guillain-Barré syndrome. These observations do not in any way lessen the danger of severe damage to the nerve fibres in the spinal roots as emphasized by Graveson and Dr. Jackson and his collaborators. On the contrary, the significance of the damage is increased, since once the nerve fibres are totally destroyed at this level their regeneration is impossible, and if all are destroyed reinnervation even at the intramuscular level will also be impossible, and not, as Dr. Jackson et al. suggest, merely delayed. However, the good functional results usually obtained, even in untreated cases, suggest that total destruction of nerve fibres is rare, and it is usually well within the capacity of the surviving neurones to reinnervate such muscle fibres as have been denervated.

May I suggest, in conclusion, that before cortisone can be accepted as having a rational basis in the therapy of the Guillain-Barré syndrome much more information is required concerning the pathological changes, and that, in the absence of necropsy material, muscle biopsy using Coërs's techniques may not be entirely without value. I am, etc.,

Smethwick.

A. L. Woolf.

## REFERENCES

Graveson, G. S., Lancet. 1957, 1, 340.
 Guillain, G., Ann. Méd., 1953, 54, 81.
 Walton, J. N., Lancet, 1956, 1, 1023.
 Coërs, C., Acta neurol. psychiat. belg., 1955, 55, 741.
 Seddon, H. J., Medawar, P. W., and Smith, H., J. Physiol. (Lond.), 1943, 102, 191.
 — Brit. J. Surg., 1948, War Surg. Suppl., No. 2, p. 325.

# Police Evidence Preferred

SIR,-May I comment on the report (Journal, February 23, p. 468) under the title "Police Evidence Preferred"? An experienced police officer who sees a motorist at the time he is driving, or attempting to drive, is in a far better position to assess his ability to drive than a doctor remotely, by tests, in a police station.

I quote another case to illustrate this. One evening a motorist, whose speech was slurred and who was excited, brought to my house a cyclist he had knocked over. I told him he had had too much to drink and advised him to leave his car and walk home. He was very incensed at this and

demanded that I should call a policeman, as I had insulted him. I rang up the police station, and asked the constable who arrived to persuade him to go home quietly without his car. The constable got him outside on the pavement, and, after testing his walking, told him that he would be wise to follow my advice but that he was at liberty to make his own choice.

The driver thereupon got into his car, started off at a fast speed down a hill, mounted the pavement on the opposite side of the road, returned to his own side, crossed the pavement there, and collided with the wall bordering the churchyard. He had no explanation to offer for this driving. He was arrested, brought back to my house, and I was asked to certify him. I refused to do this, and he was examined later at the station by a colleague who told me the rest of the story. After submitting the defendant to various inconclusive tests and fortified by the history, he told him hé was drunk. He was then asked by the motorist: "Doctor, could a drunk man stand up in the middle of this room, jump into the air, turn a complete somersault, and land down on his feet ? " My colleague was injudicious enough to say, "Certainly not "-and was then and there proved wrong.

Your correspondent's report induces further thoughts. The borderline case is mentioned. It can be proved that a single drink will invariably lengthen the reaction time, and must therefore impair the control of a vehicle. With further alcohol other impairments follow—lack of concentration, faulty visual accommodation, muscular incoordination, and so on—until the patient ultimately becomes paralytic. At this stage he is quite safe to be "in charge" of his car, as he can no longer start it. Where is the borderline? There is a perfectly good measure of alcoholism if it is really wanted, and that is the urine alcohol concentration. figure can be fixed and it can be proved which side of the borderline any motorist may be. It has been argued that this is unfair to the man who can carry his drink, but it is no more unreasonable than the 30 m.p.h. limit is to the first-class driver. The limit is set for the average man and all must conform.

Finally, your correspondent asks why accused persons should be asked to submit to examination by a doctor. The purpose of the medical examination should be to exclude illness, accident, or infirmity which might be mistaken for alcoholism or contribute to its effects. The medical profession should be allowed to confine its evidence to such matters; for on these it is qualified to speak. On other matters, police evidence is to be preferred.—I am, etc.,

Warwick.

CHARLES L. WORTHINGTON.

### Fatal Reactions to Local Anaesthetics

SIR,—Your annotation on fatal reactions to local anaesthetics (Journal, February 2, p. 276) is most timely, particularly in view of the tendency to look upon very ill cases as those best suited to surgical interference under this form of anaesthesia. Unfortunately, it is this particular type of case in whom untoward reactions are most likely to occur. It should be stressed that in such cases, as indeed in all cases, the least amount and the lowest concentration of the least toxic agent should be the rule in the application of local anaesthetic agents. Your annotation notes that adrenaline is often included in local anaesthetics in order to retard absorption and prolong effect. While this is true, and indeed desirable, I would underline the danger of too high concentrations of adrenaline being employed. A concentration of 1 in 300,000 is quite sufficient for all practical purposes.

Adequate sedative premedication with either a barbiturate or an opiate is not only humane but undoubtedly does raise the individual's threshold to the toxic effect of local anaesthetic agents. It should rarely, therefore, be omitted. 30 ml. of 2% lignocaine is mentioned in your annotation as constituting an overdose, and with this few would argue, although it was not sufficiently stressed that such an amount should be combined with adrenaline. But why use 2% lignocaine in such quantities? Lower concentrations provide adequate sensory loss. Indeed, the only justification for using 2% lignocaine is where motor loss is required as When used in low concentrations with appropriate amounts of adrenaline in patients suitably premedicated, relatively large amounts of lignocaine can be used with safety. Scurr and Wilson and Gordon reported the routine use of as much as 1,000-1,200 mg. of lignocaine in cases of thoracoplasty, and they emphasize that the safety factors lie in premedication, low concentration of lignocaine, and low concentration of adrenaline. Perhaps as important as all these precautions is a more than passing knowledge of the techniques of performing local anaesthesia.—I am, etc.,

HOWARD BRUCE WILSON.

#### REFERENCES

Scurr, C. F., Curr. Res. Anesth., 1952, 31, 225.
 Wilson, H. B., and Gordon, H. E., Anaesthesia, 1952, 7, 157.

SIR,—May I congratulate you on your excellent and timely annotation on fatal reactions to local anaesthetics (Journal, February 2, p. 276)? There is, however, one small point in it which may confuse the inexperienced. It is stated that "more than 30 ml. of this strength [2%] would constitute an overdose in most patients." I would suggest that more than 25 ml. of 2% lignocaine should be considered an overdose, whereas the maximum dose of procaine is definitely higher than this, and is usually considered to be 1 g. Goldberg1-3 and Hunter,4 after careful experiments with lignocaine, recommend that the safe maximum dose should not exceed 500 mg. (25 ml. of 2% solution). Since Goldberg considered lignocaine 2% to be twice as toxic as 2% procaine, it behoves the anaesthetist to be particularly careful when injecting the former into a particularly vascular area.

At St. Mark's Hospital, Mr. W. B. Gabriel has been using 2% procaine with 1 in 80,000 adrenaline into the perianal space and ischiorectal space, 20 ml. on each side, without any untoward result for many years. This amount of adrenaline is necessary to overcome the strong vasodilator action of procaine, and certainly delays absorption, the analgesia sometimes lasting for as much as five hours. I have never seen any reaction to this concentration of adrenaline other than a transient rise of pulse rate in some patients. Lignocaine 2% with 1 in 80.000 adrenaline is also used in the same manner at St. Mark's, but the dose injected is 10 ml, each side with equally good results. It has been my practice lately to give thiopentone 300 mg., followed by chlorpromazine 12 mg., promethazine 25 mg., and pethidine 25 mg, prior to the local injection in nervous patients, with excellent results. The patient does not rouse when the lignocaine is injected and sleeps for about two hours post-operatively. "Omnopon" <sup>1</sup>/<sub>3</sub> gr. (22 mg.) and scopolamine 1/150 gr. (0.43 mg.) are given as premedication one hour before operation.—I am, etc.,

London, W.1.

FRANKIS EVANS.

- REFERENCES
- Goldberg, L., Svensk tandläk, T., 1947, 40, 819.
   Svenska LäkT., 1948, 45, 107.
   Acta physiol. scand., 1949, 18, 1.
  Hunter, A. R., Lancet, 1956, 1, 158.

## Paediatric Anaesthesia

SIR,—I read with interest Dr. W. N. Rollason's reply (Journal, September 29, 1956, p. 769) to Dr. John Bullough's letter (Journal, August 11, 1956, p. 360), in reference to premedication and anaesthesia for guillotine tonsillectomy in children. I agree with Dr. Rollason's criticism, and add with sincerity of purpose that in this era of prolific manufacture of drugs some of the older innocuous tranquillizers, in particular chloral hydrate, are not given the publicity they deserve. The crux of the matter is to achieve the tranquillity of the patient with safety before, during, and after the operation. I have used syrup of chloral in 4 gr. per stone (0.3 g. per 6.4 kg.) body weight, with excellent results, and would suggest that its overall safety should certainly justify its continued use as a sedative pre-operatively and post-operatively if necessary.—I am, etc.,

R. M. B. PENHEAROW.

### Tonsils and Adenoids

SIR,—I have just read the article by Dr. J. Fry (Journal, January 19, p. 124). My comment is, I could not agree more. Tonsillectomy is recommended too often by some practitioners.

As far as my work as a school medical officer is concerned, practically speaking I never recommend tonsil and adenoid removal in school entrants (5- or 6-year-olds). I think a child has some tonsillar infection I arrange to see him again the following year, and often a third time another year or more later, before I refer him to an otolaryngologist. All indications mentioned by Dr. Fry are taken into account, including the history as given by the parents. One thing is certain: we do see patients at school inspections who never attend their general practitioners and who eventually do require tonsillectomy. Even in these days of an apparently free Health Service some parents do not take their children to the doctor or call him when they ought to do so. These children have a history of frequent colds (the catarrhal child), which I am convinced are undiagnosed (because not seen by a doctor) tonsillitis.

Dr. Fry says quite truthfully that children grow out of their tonsillitis tendency. In one's "critical assessment" one must take into account the amount of continuous or repeated discomfort and misery that the child may have to go through before growing out of it. I can remember vividly one poor boy whose parents would not give consent that he should see an otolaryngologist with a view to tonsillectomy. He was persistently deaf from his hypertrophied adenoids. Eventually at about the age of 10 years (not 6) this passed off and the tonsils shrank. He was well behind the other children in his education, as, even when present at school, he could not hear properly. I could say more about this case and why tonsillectomy was not done, but I will not, as I should probably get a come-back.—I am, etc.,

Kettering.

MURIEL C. GOODCHILD.

# Antiseptics in Midwifery

SIR,—We have read with interest the paper "Antiseptics in Midwifery," by Dr. R. M. Calman and Dr. J. Murray (Journal, July 28, 1956, p. 200), and we note particularly the comparison of the quaternary ammonium (O.A.) compounds, such as cetrimide and benzalkonium chloride, with chlorhexidine. These results are of particular interest in the light of similar investigations now being carried out in our laboratories with benzalkonium chloride and chlorhexidine.

The general intention of the authors is that the field of investigation should cover the value of the compounds not only in vitro but also in the field of the sterilization of the skin in midwifery practice and in the treatment of wounds. While we would agree that the in vitro tests show chlorhexidine to possess an advantage in activity over benzalkonium chloride against Ps. pyocyanea, we would submit that in vitro conditions are not those appertaining when attempting to measure sterilization of the skin. The authors have not attempted to measure the activity of chlorhexidine on the skin in the same manner as Colebrook1 did in his investigations on chloroxylenol. It is now well known that the Q.A. compounds are highly adsorbed on certain surfaces, and when so adsorbed retain their bactericidal action over a long period. Thus Steingold et al.2 showed that benzalkonium chloride is adsorbed on to wool, such as blankets. and that the adsorption is so pronounced and so firm that the blanket yielded three successive water extractions, all of which inhibited the growth of Staphylococcus aureus and streptococci in nutrient broth.

Blankets on which Q.A. compounds are adsorbed, even after rinsing with water and drying, have a surface which is highly bactericidal to droplet-infection of these organisms. We have now evidence that Q.A. compounds are similarly adsorbed on to the skin, and it would be interesting to know if chlorhexidine is adsorbed in the same manner.