or just below for the next 40 minutes. However, the tone of the jaw muscles was still clearly subnormal and it was considered unwise to extubate for a further two hours (about four hours after the last dose of curare). The patient made an uneventful recovery.

Case 2.—A man of 63, with moderate chronic bronchitis, had suffered from increasing abdominal pain and distension for one month. There had been no vomiting and there was no disturbance of serum electrolytes. Premedication was with atropine, mg. 0.9, and induction of anaesthesia with thiopentone, mg. 300, and succinylcholine, mg. 50. When respiration was re-established 75% N₂O/O₂ and a low concentration of ether were administered for three to four minutes. D-tubocurarine chloride was given in divided doses, mg. 10+10+10+5, and I.P.P.R. was established with N₂O/O₂. A further 5 mg. of curare was required 15 minutes later. The operation of colostomy lasted almost one hour and atropine, mg. 2.4, and neostigmine, mg. 5.0, were given in divided doses. Reversal of curarization was judged satisfactory by respiratory activity and strength of hand-grip. However, when seen in the ward a few minutes later, the patient's respiration was inadequate. He had a gross tracheal tug and cyanosis. He was reintubated and hyperventilated for about 40 minutes. Spontaneous respiration was then re-established but showed features of residual curarization. Edrophonium, mg. 10, caused a marked improvement and atropine, mg. 0.6, and neostigmine, mg. 2.5, were therefore given. Respiration became clinically satisfactory. Half an hour later a rebreathed gas sample showed a normal mixed venous Pco₂. Respiration and muscle tone were thereafter always adequate.

These two cases do not prove that oral neomycin can cause difficulty in reversal of curarization. But it is known that parenteral neomycin can affect neuromuscular transmission and that oral neomycin can, on occasion, be absorbed in appreciable quantities from the apparently normal intestinal tract. It may be that this absorption is increased in intestinal obstruction when it is likely that the concentration of neomycin in the gut is high due to stasis and the lack of normal food and fluid intake, and that the permeability of the wall is greater owing to distension. It is also likely that absorption may be rapid when the continuity of intestinal mucosa is lost, as at the site of a carcinoma or other ulceration. In this context, it is of interest that Dr. Ross's case was suffering from bleeding per rectum. It therefore seems that anaesthetists would be wise to regard oral neomycin as yet another potential hazard to safe practice.—We are, etc.,

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Antibiotic Cover for Dental Extractions

SIR.—The case of subacute bacterial endocarditis reported by Drs. C. G. Barnes and Rosalinde Hurley (November 9, p. 1205) is most interesting.

There are reports in the literature of endocarditis arising in spite of the provision of prophylactic penicillin during dental treatment on susceptible patients.¹⁻⁴ However, it is possible to explain the majority of these failures on two grounds: that the cover was inadequate in amount or timing, or that previous penicillin therapy had given rise to penicillin-resistant strains of oral streptococci—as Garrod and Waterworth⁴ showed can so easily happen.

Neither of these explanations fit this particular case, but before attributing the infection to a failure of an apparently adequate regime it is worth considering that the responsible bacteriaemia might have occurred either before or after the dental extractions. It was not clear whether total extractions were performed.

This would be coincidence, no doubt, but bacteriaemia may occur whenever dental sepsis is present.—I am, etc.,

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Nicotine and Colonic Muscle

SIR.—In the letter of Dr. K. C. Dave and Dr. K. S. Sachdev (November 9, p. 1205), which discussed our report of the action of nicotine on human colonic circular muscle *in vitro* (September 14, p. 666), the question of premedication was raised.

The pre-operative medication given to our patients has varied. The majority received a combination of atropine 0.6 mg. or scopolamine 0.5 mg. with "omnupon" 20 mg. or morphine 16 mg. However, several received atropine with pethidine 100 mg. and a few received atropine with an antihistamine. We have found no difference in the response of colonic muscle that can be related to the various combinations of premedication. The premedication has been given at least an hour before the start of the operation, and the muscle strips have been obtained two to three hours after the premedication.

We have reported that the colonic circular muscle strip responds to a dose of 10 µg./ml. (10⁻⁴) of acetylcholine. However, some of our strips have been more sensitive than this, responding to 1–10 µg./ml. (10⁻⁵, 10⁻⁶), and even these do not contract in response to nicotine.

The concentration of atropine in the tissues two to three hours after a dose of 0.6 mg. must be small; very much smaller, in fact, than the concentration found necessary by Gillespie and Mackenna¹ to block the parasympathetic effects of nicotine in the rabbit colon (10⁻⁴). The effects of atropine on the human colon *in vivo* have been studied by Kern *et al.*,² who showed that the

motility is depressed, but that even with doses of 1 mg. the effect was slight and of short duration, usually less than 30 minutes.

For these reasons we think that the pre-operative atropine is unlikely to affect the muscle in our *in vitro* experiments, but we must admit that we cannot be certain about this yet.—We are, etc.,

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Flavobacterium meningosepticum in Israel

SIR.—In 1957 Brody *et al.*¹ and in 1960 Cabrera and Davis² isolated a Gram-negative rod from the cerebrospinal fluid in nosocomial outbreaks of meningitis in newborn children, mostly prematures, which occurred in Greenville, South Carolina, Portsmouth, Virginia, and Columbus, Ohio, respectively. King³ examined these strains in detail and called the hitherto unnamed organism "Flavobacterium meningosepticum." As far as we are aware, the bacterium has only once been reported upon from outside the U.S.A.⁴

We have recently identified the same bacterium in Israel, where it was cultured from the cerebrospinal fluid in seven cases of meningitis occurring during the summer of 1962 among the newborn infants in the nursery of a hospital. As late as three months after the occurrence of the last meningitis case we isolated it also from the throat swabs of eight healthy babies in the same section. For the isolation from this material with mixed flora we used MacConkey agar with 5% blood from the blood bank, a medium which we found to be very suitable for this purpose.

Three of our strains were submitted to Miss King, who confirmed our diagnosis and determined them as belonging to serotype C, the one responsible for most of the cases in U.S.A. (personal communication).

Although no treatment of the healthy carriers was attempted, the bacteria were never found a second time on repeated examinations of nose and throat of the same babies, one or two weeks after the first isolation. Obviously, they remained only for a short time in the carriers as harmless commensals. No positive findings were obtained from the adult contacts with the infants.

Four of our healthy carriers were found amongst 15 premature infants present in the nursery at the time of our examination, the other four amongst 95 full-term babies—that is, in 26.6% of the first and in 4.2% of the second group. This percentage distribution in healthy infants seems to indicate that a host organism is the better suited as substrate