for ruptured peptic ulcer. In our series the operative and postoperative course has been satisfactory. In all cases the drug has been used in conjunction with light anesthesia with cyclopropane. It is our custom to inject 3 cc. of the product intravenously as the fascia is opened. If muscular relaxation is not satisfactory following injection of the initial 3 cc., we inject an additional 1 or 2 cc. of the drug. Special attention is paid to the respiration at all times. If too large an initial dose is used, complete intercostal paralysis and apnea will result. Fortunately this complication is usually transient, and if the patient is ventilated by pressing on the bag of the gas machine he usually will start breathing after one or two minutes. Prostagmine has been described as an antidote but fortunately we have not been forced to use it.

"The apnea may be prevented by carefully injecting the preparation of curare and watching its effect on the respiration rather than injecting too large a dose at any one time. If more relaxation is needed as the operation progresses, supplementary injections are made as needed. Usually two or three injections are adequate for most operations. Another use we have found for the product under consideration is for the patient who is resistant to the anesthetic agent. This type of patient usually is heavy and muscular and frequently is a heavy smoker and drinker. In this type of case, frequently more or less severe laryngospasm develops; the patient becomes cyanotic and his anesthetic course is highly unsatisfactory. We have found that the preparation of curare will cause this type of patient to relax and then the anesthetist can produce and maintain a satisfactory plane of anesthesia. Some means of giving artificial respiration always should be available because, if apnea results and no means of ventilating the patient is at hand, death from anoxia may result." 2 references.

J. C. M. C.


"During four months of last summer thirteen patients developed cranial nerve palsies following a variety of general anaesthetics. The degree of severity varied from subjective trigeminal anaesthesia only to a complete picture of encephalitis in one of the patients who died. The nerves involved were Vth with VIIth in many, and in some IIIrd, VIth, VIIth, Xth, or XIIth. Herpes of the febrile type developed about the third day in eleven of the patients. The only abnormalities in the cerebrospinal fluid were in two cases a small increase in polymorph cells and in five an increase in protein. The outstanding post-mortem finding in the patient who developed a picture of encephalitis was oedema of the brain and brain-stem although there were occasional localized perivascular collections of lymphocytes. The earliest approach to the problem of investigation was an analysis of anaesthetic drugs but as there were no impurities apart from a trace of chlorine in the cyclopropane, and this within the specifications of the U.S.P., the possibility of a virus infection was considered. . . . The virus theory was neither proved nor disproved but all the evidence points to a toxic drug effect following reactions between trilene and soda-lime. It was realized that trilene could have persisted in the machine or the soda-lime which may have been 'rested' for as long as three days. The use of the contaminated soda-lime at a later date might explain the intervals between the trilene administrations and the anaesthetics which were followed by pal-
Abstracts


"The changes in cardiac rhythm which occur during trilene anaesthesia are regarded by some with alarm whilst others consider them to be of no importance. There seem to be two reasons for this divergence of opinion. In the first place the exact nature, and therefore the significance, of these arrhythmias cannot properly be determined unless frequent electrocardiograms (E.C.G.) are recorded throughout the anaesthetic, and up to the present this has been done only in six cases. Secondly, in the reported cases in which marked arrhythmias occurred there have been other factors—such as intubation—which might themselves be held responsible for the change in rhythm. . . . Forty patients have been investigated, 30 males and 10 females. All of these were examined clinically by one of us (C. G. B.) on the day before the operation and a routine 3-lead E.C.G. was taken. Each subject selected for this inquiry was normal clinically and showed a physiological E.C.G. Their ages varied from 10 to 74 years. . . . Cardiac disturbances during the anaesthetic were noticed clinically in 12 cases; bradycardia and occasional premature contractions being the most common changes. Pulsus bigeminus was found in 1 case and 2 others each developed for a short period a rapid and completely irregular pulse. These few arrhythmias were in striking contrast to the large number observed cardiographically, for only 7 of our 40 patients showed no change at all in the E.C.G. during the operation. Very many varieties of arrhythmia were observed in the E.C.G.s and it was common for a patient to exhibit several of these successively as the anaesthetic continued. In general, however, these alterations in rhythm fell naturally into two groups. In the first there was a series of changes due probably to increase in vagal tone, and these tended to occur in the first ten or twelve minutes—often in the induction phase of the anaesthetic. We noticed that these changes were more likely to occur in those patients who showed signs suggestive of high vagal tone before operation, in the form of bradycardia and sinus arrhythmia. . . . They were all transient and disappeared as the anaesthetic continued.

"The second group of arrhythmias tended to occur later, during the lower first or upper second plane, and took the form of ectopic foci initiating premature contractions sometimes in the auricles (in 5 cases), but more often in the ventricles (in 16 cases). At first the ectopic beats occurred quite haphazardly, and from a single focus, but as the anaesthetic proceeded they gave way in 11 patients to alternating ventricular premature contractions causing pulsus bigeminus. Shortly after this, in 6 cases, they were followed by multiple ventricular contractions arising from several different foci, and in 4 patients these abnormal beats occurred at a great rate—between 130 and 200 a minute—to give extremely bizarre tracings in the E.C.G. This last arrhythmia is, we believe, important, and we shall refer to it as 'multifocal ventricular tachycardia.' It must be emphasized that with this arrhythmia the pulse becomes completely irregular and extremely rapid, in fact indistinguishable clinically from au-