Cyclopropane Anæsthesia

By JOHN BOYD, M.D., D.A.

THIS paper is based on my experience of one thousand cases of cyclopropane anæsthesia personally conducted by me since October, 1938, both in hospital and in private. But before discussing these it might be convenient for me to mention here something about the drug itself.

HISTORY.

Cyclopropane was first isolated in Germany in 1882 by Freund, who also demonstrated its chemical structure, C_3H_6. He did not, however, describe its anæsthetic properties. Following its discovery it seems to have been forgotten until 1928, when Henderson and Lucas of Toronto, in investigating contaminants of propylene, another anæsthetic with undesirable side-effects, and itself an isomer of cyclopropane, found that the supposed cause of the cardiac disturbances was in reality a better and less toxic anæsthetic. They demonstrated its anæsthetic properties first on animals, and then, before releasing it to the medical profession for clinical trial, they anæsthetised each other, and determined the quantities necessary for administration to man.

In 1933 the first clinical trials of cyclopropane were made by Waters and his associates of the University of Wisconsin. In October of that year Waters presented a preliminary report on its anæsthetic properties in man,[1] confirming the findings of Henderson and Lucas.

Rowbotham introduced it to England first in 1935, and since then its use has spread rapidly throughout the country.

PREPARATION.

Cyclopropane is prepared commercially by the reduction of trimethylene bromide in the presence of metallic zinc in ethyl alcohol. It is also made commercially from propane in natural gas by progressive thermal chlorination.

PROPERTIES.

At room temperature it is a colourless gas with a characteristic naphtha-like odour. Its boiling-point is—32°C, and its density one and a half times that of air. It is relatively inert chemically, and is insoluble in water, but soluble in lipoids and in concentrated sulphuric acid. It liquefies at a pressure of 75 lb. per sq. in., and as such is stored in cylinders. Storage at this pressure obviates the need for a reducing valve. This is of importance, as cyclopropane would produce deterioration in the rubber used for reducing valves. It is very explosive, especially when mixed with oxygen, the range of explosibility being from 2½ to 63 per cent.[2] As the anæsthetic range varies between 3 and 40 per cent., it will be appreciated that the anæsthetic mixture is at all times within these limits. It costs approximately three shillings per gallon, but as it is always used in closed circuit machines, one or two gallons suffice for an hour's operation.

PHARMACOLOGY.

The pharmacology of cyclopropane has been extensively studied in America

since 1934, when Waters and Schmidt[3] presented their first report following its administration to over two thousand unselected patients for a great variety of operations. They summed up their findings in these words: "The ability to produce deep anæsthesia without respiratory stimulation, irritation, or the possible necessity of producing oxygen-want, gives the anæsthetist a feeling of safety and assurance not experienced with any other agent." Another investigator, H. R. Griffith, of Montreal,[4] expresses his opinion of cyclopropane as follows: "My conception of anæsthesia with the older gases is that we administer the gas, plus enough oxygen to keep the patient alive and in good condition. With cyclopropane, on the other hand, we administer oxygen with just enough of the anæsthetic gas to keep the patient asleep."

Cyclopropane is non-irritant to the mucosa of the respiratory tract. In this respect it stands out in marked contrast to the more commonly used chloroform and ether. For this reason the anæsthetist must be very careful in its administration, for he lacks the warning signs given, for example, in an ether induction. With ether the patient draws the anæsthetist's attention to a too rapid increase in concentration by holding his breath. This is absent in the cyclopropane induction. There is also much less salivation with it than with the other inhalation anæsthetics. The most marked effect of cyclopropane on respiration is the depression of the respiratory centre. Both the rate and depth of respiration are reduced as the anæsthesia deepens in the third stage. Unlike ether, the rate of breathing does not increase just prior to respiratory arrest. Some observers are of the opinion that much of the respiratory depression observed with cyclopropane is due to the high percentage of oxygen with which it is administered. Despite this depression of respiration, the patient remains quite well oxygenated, because of the high oxygen content of the anæsthetic mixture. Fortunately, respiratory arrest takes place with cyclopropane long before cardiac arrest. Following a long cyclopropane anæsthesia, a partial or even a total collapse of one lung has been observed, because during the deeper levels of anæsthesia the distal alveoli are not functioning. These alveoli contain only oxygen and cyclopropane without any inert gas, e.g., nitrogen. As these two gases are rapidly absorbed at the end of the anæsthetic, the alveoli collapse.

Cardiac irregularities are sometimes observed in cyclopropane anæsthesia, the more usual being bradycardia, tachycardia, and ventricular extra-systoles, the last-mentioned predominating. It is thought that these are produced by too sudden an increase in the cyclopropane content during induction. They are easily brought under control by adding some more oxygen. These arrythmias are not so frequently observed if the patient is premedicated with a barbiturate rather than morphine. In those cases where arrythmias occur and disappear when oxygen is added there is no permanent damage to the heart. Animal experiments by Robbins[5] show that there are no electro-cardiographic changes until near the stage of respiratory arrest, also that dogs anæsthetised with cyclopropane after premedication with barbiturates do not show electro-cardiographic changes until long after respiratory arrest. Even after respiration has ceased, by carrying out

artificial respiration the blood cyclopropane content can be increased by thirty per cent. over that needed to produce respiratory arrest without disturbing the electrical mechanism of the heart.

The pulse rate and volume remained within normal limits in a series of experiments on dogs.

Opinions vary as to the effect that cyclopropane has on blood pressure. Waters[6] states that there is no change; Rovenstine[7] says there is a slight fall when it is used for thoracic surgery; while Rowbotham[8] thinks that there is always a definite rise. Robbins,[5] experimenting on dogs, finds a rise when no premedication is used, a fall after morphine, and no change after barbiturates. Robbins also established the fact of an increased cardiac output of forty-five per cent. during moderate surgical anæsthesia, but noted a decrease in the deeper planes of anæsthesia.

There is little, if any, change in the blood under cyclopropane anæsthesia, except for an increase in the white cell count, as is found with ether and other inhalation anæsthetics. The blood sugar seems to rise slightly in the normal patient, but remains level in the controlled diabetic.[3] The clotting time of blood is not affected, the slight increase in capillary bleeding being due to a capillary vaso-dilatation.

Cyclopropane seems to have no effect on the liver or kidney function. Contractions of the gravid uterus are either not affected, or may be slightly increased. Less post-delivery bleeding is noticed in patients under cyclopropane than in patients under other agents.[9] Griffith[10] states that after considerable experience he was convinced that "there is better uterine contraction under cyclopropane. This is true with normal and forceps delivery, as well as cæsarean section, and we do not now see the alarming post-partum hæmorrhages which occasionally used to worry us."

Premedication.

Premedication follows much the same lines as with other inhalation anæsthetics. Atropine gr. 1/100, or scopolamine gr. 1/150 is given to reduce the secretion of the glands of the oral and respiratory mucosa. With regard to sedation, it is generally agreed that too much premedication with drugs that depress respiration is inadvisable. Waters[3] prefers to decrease the dose of the opium derivative generally used before anæsthetic agents by at least one-half, while retaining the full customary dose of scopolamine, giving both one and a half hours pre-operatively. Some give preference to barbiturates rather than opiates, considering that the former abolish cardiac irregularities.[11] Rowbotham[8] prefers omnopon gr. 1/3 and scopolamine gr. 1/150. Wood[12] favours avertin, .9 gm. per kgm. body weight, for premedication, combining it with atropine.

Technique of Administration.

Owing to the high cost of cyclopropane as compared with other gaseous anæsthetics it is generally administered by means of some sort of closed circuit apparatus, with total rebreathing and carbon-dioxide absorption. This also obviates to a large extent the danger of explosion associated with a cyclopropane-oxygen mixture. The principle underlying carbon-dioxide absorption is the

supplying of sufficient oxygen necessary for the patient's metabolic requirements, and the removal of the resultant carbon-dioxide, which would tend to dilute the mixture, and cause hyperpnœa. The percentage of gases in the mixture remains constant, as the patient uses up the oxygen only, cyclopropane not being broken down in the body. Thus the anæsthesia remains stable. The net result of all this is less respiratory effort on the part of the patient, who stands a prolonged operation better, less anæsthetic is needed to maintain anæsthesia, and less anæsthetic is inhaled by the surgeon and anæsthetist.

Four essentials are required in an anæsthetic machine for the administration of cyclopropane : (1) Some form of flow-meter capable of measuring and delivering as small a volume of gas as 50 cc. per minute; (2) A rebreathing bag of one to two gallons capacity; (3) A leakproof facepiece; (4) A canister containing soda-lime, to absorb the carbon-dioxide. Carbon-dioxide absorbers are of two types : (a) the to-and-fro absorber, where the rebreathing bag and absorber are attached to the facepiece, the anæsthetic gases being led to the facepiece by a narrow-bore tube; (b) the circle absorber. In this type the absorber and rebreathing bag are attached to the machine, and wide-bore tubes carry the anæsthetic mixture to and from the facepiece, uni-directional valves maintaining a one-way gas flow. The to-and-fro absorber gives more efficient absorption, but is more cumbersome to use. Soda-lime is a mixture of 65 to 95 per cent. calcium hydroxide and 5 per cent. caustic soda. One pound of this will absorb carbon dioxide efficiently from the gas mixture for about six hours.

There are several methods of inducing anæsthesia with cyclopropane. Some anæsthetists partly fill the bag with oxygen, and then run in cyclopropane at a rate of 300 to 700 cc. per min. with 250 cc. of oxygen. After a period of from one to five minutes, varying with the rate of flow of cyclopropane, the anæsthetic gas is stopped. By this time the patient is in the third stage of anæsthesia. Another method of induction is to give 1,000 cc. oxygen plus 400 cc. cyclopropane for one minute. Then the oxygen is reduced to the basal flow of 250 cc. per minute, and 200 cc. of cyclopropane per minute is run in for four minutes. After a further four minutes, during which the cyclopropane is given at the rate of 100 cc. per minute, the patient is usually in the third stage.

The maintenance of anæsthesia is carried out as follows : The patient continues to receive 250 cc. to 300 cc. of oxygen, sufficient for his metabolic requirements, and either a constant trickle of 50 cc. of cyclopropane per minute is added, or 100 cc. per minute is given for two or three minutes at intervals during the operation, when the anæsthesia tends to get lighter. At no time subsequent to the induction should the patient be given cyclopropane at a faster rate than 100 cc. per minute, on account of the risk of respiratory depression due to too sudden an increase in its concentration. Theoretically there should be no need to add cyclopropane once anæsthesia is stabilised, but despite close-fitting facepieces, etc., there seems to be a slight constant leak of gases. In addition to the mechanical loss through the imperfections of the machine, some cyclopropane is lost through the skin wound due to hæmorrhage, etc. At the end of the operation

a little air should be added gradually to the system, to ensure that the change over be not too sudden from the high oxygen content of the anæsthetic mixture to the normal oxygen content of air.

Constant and undivided attention must be paid to the details of the administration of cyclopropane because of its potency in low concentrations, the absence of respiratory stimulation, and the fact that dangerous concentrations might be reached without receiving the timely warning that cyanosis or laryngospasm affords. A gradual induction and a gradual deepening of anæsthesia when necessary, combined with a close supervision of the patient, are essential with an anæsthetic which is lacking in the usual physical signs associated with the various stages of anæsthesia.

Signs of Anæsthesia.

The physical signs of anæsthesia with cyclopropane follow much the same pattern as with ether, with one or two slight differences. These differences are due to the fact that two properties associated with ether are not observed with cyclopropane, namely, respiratory irritation and stimulation.[3] The respiratory irritation of ether is largely responsible for the excitement of second stage anæsthesia with that drug. With cyclopropane the second stage is seldom well defined, the patient passing quietly and rapidly into the third stage. As it has no irritant effect on the respiratory tract it does not produce the laryngospasm that is a protective mechanism against the inhalation of too high concentrations of ether. The stimulation of ether produces an increase in the respiratory rate up to the point at which the depression of an overdose of the drug, or of oxygen-want, supervenes. Cyclopropane administered with carbon-dioxide absorption does not increase the respiratory rate even when the depressive doses associated with respiratory paralysis are reached.

The pupils are of little value in estimating the depth of cyclopropane anæsthesia. Usually the first sign that the patient has entered the third stage is the fact that the breathing has become automatic, with the loss of the pause at the end of expiration. The pupil is still roving about. This occurs when the cyclopropane content is about 7 per cent.[5] As the patient passes through the first plane of the third stage the pupil becomes fixed and central. About 13 per cent. cyclopropane is necessary for this level. The demarcation of the third and fourth planes of the third stage is very vague, and the patient with little warning enters the fourth stage, the stage of respiratory arrest. This occurs with a 43 per cent. cyclopropane concentration. A valuable guide is found in the blood pressure and pulse rate, as I hope to mention later. Fortunately the respiratory arrest of the fourth stage can be quickly overcome by the addition of some oxygen, coupled with rhythmic compressions of the rebreathing bag. The respiratory arrest that can be easily produced with cyclopropane is often used clinically under the name of controlled respiration. By means of ample premedication with sodium pentothal and omnopon the respiratory centre is depressed. Efficient carbon-dioxide absorption removes another stimulus to respiration, with the result that respiratory arrest takes place with relatively low concentrations of cyclopropane. The anæsthetist then carries on

respiration for the patient by squeezing the rebreathing bag at the rate of fifteen to twenty times per minute. This can be kept up for long periods. It is employed largely in thoracic surgery, where it produces adequate oxygenation by preventing paradoxical respiration when one side of the chest only is opened. This is not true fourth-stage anæsthesia, for, despite the fact that the patient is making no voluntary respiratory effort, he may be kept in a light plane of anæsthesia. Controlled respiration is sometimes employed to procure adequate relaxation for upper abdominal surgery.[13]

The muscular relaxation produced by cyclopropane compares favourably with that during ether anæsthesia, with the possible exception of upper abdominal surgery, when it is suggested that ether be added to secure adequate relaxation

Sequelæ of Cyclopropane Anæsthesia.

Unpleasant post-operative sequelæ are, generally speaking, less frequent than with other anæsthetics of equal potency. Waters and Schmidt,[14] comparing a series of 2,200 cyclopropane anæsthesias with a similar number of nitrous oxide, ethylene, and ether anæsthesias, came to the conclusion that as far as circulatory complications were concerned cyclopropane was better than ethylene or ether but inferior to nitrous oxide. Respiratory complications and vomiting were much less frequent after the cyclopropane than after the control series. Nausea and vomiting were less in major surgery, but possibly greater in minor surgery. They concluded their report as follows : "The impression gained by surgeons, anæsthetists, nurses, and patients who have had experience of cyclopropane is distinctly favourable."

Indications and Contra-Indications.

Owing to the fact that cylcopropane is non-irritant to the respiratory tract. it is the ideal anæsthetic in thoracic surgery. The control that the anæsthetist has over the respiration enables the surgeon to get short periods for delicate work, when the lung is absolutely quiet. With cyclopropane the presence of active pulmonary tuberculosis is no longer a contra-indication to general anæsthesia. It is eminently suitable for thoracoplasty in pulmonary tuberculosis. It is ideal also in patients suffering from bronchitis or asthma, who need an operation for some intercurrent malady.

The adequate oxygen supply with which cyclopropane is administered makes it the ideal anæsthetic in cases of cardiac decompensation, in anæmias, in goitres, and in severely shocked or toxic patients. Generally speaking, most of the bad operative risks met with in surgical practice are quite easily anæsthetised with cyclopropane. It is of equal toxicity with nitrous oxide, and yet it supplies that essential component of good anæsthesia, relaxation, which is sadly lacking in nitrous oxide-oxygen anæsthesia. To obtain anæsthesia, nitrous oxide must be administered with a percentage of oxygen insufficient for the requirements of a bad risk case. Thus, instead of nitrous oxide being a good anæsthetic in bad risks, it is a highly dangerous one. Cyclopropane, on account of the high oxygen percentage with which it is administered, provides sufficient relaxation with ample oxygenation, and is a god-send to the bad surgical risk.

Cyclopropane is the ideal anæsthetic in midwifery, especially in those cases where there is an associated toxæmia of pregnancy. Here the need for an anæsthetic which causes no further damage to the liver is amply met. Wesley Bourne[15] of Montreal gives his impressions of the value of cyclopropane in obstetrics as follows: "I may say that cyclopropane seems to be very suitable for the relief of pain in obstetrics, for the following reasons—(1) An abundance of oxygen is given with the cyclopropane. (2) Circulation and respiration are not depressed. (3) Anæsthesia is produced without appreciable metabolic disturbance. (4) Liver function is not impaired. (5) Anæsthesia is quickly and easily induced, satisfactorily maintained at any desired depth with ready flexibility, and with minimal danger to the mother and child, and recovered from easily and uneventfully." On account of the fact that with cyclopropane there are better uterine contractions, it causes less post-partum hæmorrhage,[6] and fewer cases of retained placenta than had been experienced when chloroform was given for the delivery.

Griffith[10] states that a marked improvement in the post-operative course of cæsarean section after cyclopropane was noticed, particularly with regard to vomiting and abdominal distension. Paralytic ileus, which hitherto he had found a fairly common complication, was absent after cyclopropane.

Rosenfeld and Snyder [16 17] carried out an interesting series of experiments on pregnant rabbits. They demonstrated that the normal fœtus, both in the experimental animal and in woman, has active respiratory movements. They investigated the effects of ether, nitrous oxide, and cyclopropane upon the fœtal respiration. With ether this was abolished during the period when the mother was anæsthetised. In nitrous oxide anæsthesia 90 per cent. nitrous oxide and 10 per cent. oxygen abolished fœtal respiration, even though the mother was not fully anæsthetised. A mixture of 85 per cent. nitrous oxide and 15 per cent. oxygen did not abolish fœtal respiratory activity, but it did not anæsthetise the mother. With cyclopropane alone, with its high percentage of oxygen, were they able to produce anæsthesia in the mother without depressing or abolishing fœtal respiration. They concluded, "Most anæsthetics of both non-volatile and volatile types suppress intra-uterine respiration long before surgical anæsthesia is reached in the mother. The result with cyclopropane illustrates the attainment of one important objective in obstetric anæsthesia, namely, the production of full surgical anæsthesia in the mother without interruption of fœtal respiration." These observations are interesting in view of the clinical experience that babies suffer less from asphyxia neonatorum after cyclopropane than after other anæsthetics administered to the mother.

There are three well-defined contra-indications to the employment of cyclopropane for anæsthesia. (1) When the operation necessitates the use of the diathermy current, either for cutting or for coagulation. This is because of its liability to explode when mixed with oxygen. This applies particularly to its use about the head or chest, but it may be used in the vagina when a completely closed circuit is employed in the administration of the gas. No form of apparatus, where there is an electric spark, should be used in close proximity to an apparatus for the

administration of cyclopropane. It should not be used near an X-ray machine. (2) In operations where adrenaline is being employed, for example, where a local is added to the general anæsthesia, or where adrenaline is being used for its vaso-constrictor effect on mucous surfaces. It is apt to cause ventricular fibrillation, and adds greatly to the anæsthetic risk. (3) Where the closed circuit system cannot be employed, e.g., for tonsillectomy. This is on account of the relatively high cost of the gas. Apart from these contra-indications, cyclopropane may be employed in the vast majority of surgical operations.

I now wish to present to you some conclusions which I have formed following an extensive personal use of cyclopropane. I have used it almost exclusively since 1938, apart from the contra-indications just mentioned. The great majority of patients I anæsthetised privately received cyclopropane, but its use in hospital was confined to bad-risk cases only, owing to the difficulty in procuring supplies in the early days of the war.

The one thousand anæsthesias consumed 1,178 gallons of cyclopropane, that is, about 1.2 gallons per anæsthetic. I found that an average of 1.4 gallons were sufficient for an hour's operating. Some of the operative procedures for which it was used were longer than others. The average duration was fifty-two minutes, with the longest just over three hours.

It was employed in most of the operations done by the general surgeon. The list includes 29 prostatectomies, 120 lower abdominal operations, and 109 upper abdominals; 322 of the cases were gynæcological, including 84 hysterectomies. I have used it 105 times in midwifery, 55 being for normal labour, and the rest for cæsarean sections; 125 were orthopædic operations, including 14 for the insertion of a Smith-Petersen pin for fractured neck of femur, generally speaking a very bad anæsthetic risk. In thoracic surgery it was employed 17 times, and, finally, to complete a very varied list, once for the removal of adenoids, and once for the extraction of teeth.

For lower abdominal surgery cyclopropane produces ideal anæsthesia. The patient goes to sleep quietly, and the relaxation is quite adequate. Soon after he is put back to bed the patient is awake, with very little post-operative nausea. Relaxation is a little more difficult to obtain for upper abdominal work, especially when the sub-diaphragmatic region is being explored. At first I was inclined to add a little ether to produce the required relaxation. This I found necessary in eight cases. With further experience I found that I was able to get the patient sufficiently slack by pushing the anæsthetic in anticipation of the need for relaxation, or by passing an endotracheal tube. This latter procedure enables one to keep the patient well relaxed without having to deepen the anæsthesia unduly, and is the technique I now employ when the upper abdomen is the site of operation. Another very satisfactory method of securing good upper abdominal relaxation is the use of one per cent. novocain solution, without adrenaline, as a field block of the abdominal wall. This is a method that I feel ought to be employed more often than it is: the muscles are beautifully slack, although the general anæsthesia may be quite light. On four occasions I used a low spinal, heavy percaine, in association

with the cyclopropane, for excision of the rectum, and for hæmorrhoids, where the spasm of the sphincter was difficult to overcome.

Almost one-third of the anæsthesias were for gynæcology, a branch of surgery eminently suitable for cyclopropane, both for abdominal and for vaginal work. Even the use of the diathermy machine is not contra-indicated, because of the distance of the spark from any great concentration of the gas. Hysterectomies do very well with cyclopropane; most patients are sitting up and feeling fairly well the next day.

But it is in midwifery that we best see the superiority of cyclopropane over the other anæsthetics in common use. Labour pains are not interrupted, except when the patient is anæsthetised deeply into the third stage, a condition only necessitated by version. I feel that this is the one condition when one would prefer to use chloroform in midwifery, as cyclopropane does not relax the patient's voluntary muscles nearly so well. In every other respect chloroform must take second place. The patient's metabolism, so often strained in pregnancy to the verge of pathology, is unaffected. Her need of extra oxygen at term is readily supplied. The toxæmic patient, dehydrated after a prolonged labour, with a grossly damaged liver, does not run the risk of delayed chloroform poisoning, an ever-present menace of the past. There is a marked reduction in the incidence of post-partum hæmorrhage, the uterus contracting down quickly and well. There is a much shorter third stage. Generally speaking, the placenta is separated within five minutes of delivery. There are fewer retained placentæ; in fact, I have yet to see one with cyclopropane. The employment of sedatives for twilight sleep is enhanced with cyclopropane. One of the great disadvantages of twilight sleep was the difficulty in getting the baby to breathe soon after delivery. This was usually blamed on the sedatives used. But it seems to me that the fault really lies with the anæsthetic used for the delivery. With cyclopropane, no matter what sedatives were given beforehand, during labour, the child is a good pink colour, and breathes soon after birth. This may be due to the high percentage of oxygen with which it is administered. Even when the mother has received three grains of seconal and four or five doses of 1/150 gr. of scopolamine, the babies showed no signs of prolonged apnœa, as is often seen when chloroform is given after any of the normal sedatives employed in twilight sleep. Thus the patient can have perfect amnesia, and yet give birth to a healthy baby.

As I have mentioned already, cyclopropane is the ideal anæsthetic for thoracic surgery. I have used it some seventeen times; fourteen times for thoracoplasties, twice for lobectomies, and once for the exploration of the thorax in a patient found to have an inoperable carcinoma of the lung. Five of the thoracoplasties were in one patient, over a period of eight months. All of these operations he stood perfectly well, the last one being the most severe test of all, in that it lasted over three hours. On each occasion he was sitting up and feeling fairly well the next day. Most of these thoracoplasty patients did quite well, but two of them call for comment. (1) A woman of 32, who was slightly cyanosed throughout most of the operation. This was found to be due to muco-pus partially blocking the trachea,

and did not clear up until the trachea was aspirated at the end of the operation. (2) A very ill, toxic patient, who came to the table with a pulse rate of 160 and a blood pressure which was barely perceptible. Despite his condition it was decided to go on with the operation. This lasted for one hour, and the patient seemed to stand it fairly well, his condition at the end being no worse than it was at the start. Next day he recovered a bit, but died on the fifth day. His death was not in any way due to the cyclopropane. Rather, this case shows how a seriously ill patient may be anæsthetised for a big operation, and be little the worse for it.

Two of the thoracic cases were for lobectomies, because of bronchiectasis. Both were children. The first case was a small girl aged five years, who had a bad bronchiectasis. She had cyclopropane for only twenty minutes, as the facepiece did not fit well. Anæsthesia was then continued with oxygen and ether. The second was a boy, aged 13 years. He was given omnopon-scopolamine and sodium pentothal for pre-medication, and an endotracheal tube inserted through his mouth. The idea in this was to abolish his own voluntary respiration and to carry on with controlled respiration. This was found impracticable, as the facepiece was not a good fit. His condition remained quite good throughout an operation lasting over two hours, apart from a fast pulse rate, running between 100 and 120. As his systolic blood pressure kept up between 120 and 130 it was thought that carbon-dioxide accumulation due to paradoxical respiration was the cause. This condition occurs when one side of the chest is opened. At inspiration, instead of the exposed lung expanding, it contracts, and expands on expiration. The result of this is that the contents of the lungs are being shuttled from one to the other. Carbon dioxide then accumulates, as most of it never reaches the absorber, and the patient becomes anoxæmic. This is best overcome by abolishing the patient's voluntary respiratory efforts, and by carrying on controlled respiration by rhythmically squeezing the rebreathing bag, thus ensuring a proper entry and exit of gases to both lungs. Both lungs are inflated and deflated at will.

The advantages of controlled respiration are well exhibited in another case where it was employed. The patient, a man of forty-seven, had a carcinoma of the bronchus. It was thought that he would be a suitable case for pneumonectomy. For premedication he received 1½ gr. of luminal and one ampoule of omnopon-scopolamine. His throat was sprayed with ten per cent. cocaine. Then he was given .75 gm. of sodium pentothal, and an endotracheal tube passed through the mouth under direct vision laryngoscopy. When cyclopropane was added, his respiratory centre ceased to function, and its activity was taken over by the anæsthetist for the duration of the operation, voluntary breathing ceasing for almost two hours. This kept him a nice pink colour throughout. His systolic pressure kept between 110 and 120 throughout the operation except for one period when it dropped to 90, due to extensive intra-thoracic manipulations. His pulse rate did not rise beyond 80 during the whole operation. In this case, in addition to employing controlled respiration, the tracheo-bronchial tree was aspirated at definite intervals by means of a gum-elastic catheter passed down the endotracheal tube.

In the bad-risk patient cyclopropane is seen to advantage. Despite the fact that

in high concentrations it causes cardiac arrythmias, it is by far the best anæsthetic for cases of grave cardiac decompensation. In my series twelve patients had a history of myocardial involvement prior to operation, four of whom had gross decompensation. Notwithstanding this defect, all stood the operation well. It would seem that the high oxygen content of the anæsthetic mixture and efficient absorption of carbon dioxide help towards this end. One case in particular stands out. A patient, aged 33, with a badly decompensated heart, needed an anæsthetic to have her pregnancy terminated. She had many moist sounds at her bases, and was so distressed that she could not lie down on the table, nor could she tolerate a mask on her face owing to dyspnœa. She was given .35 gm. sodium pentothal in the sitting-up position. As soon as she was asleep she was put down flat on the table, and the anæsthetic continued with cyclopropane and oxygen. Even when she was put up into the lithotomy position for operation, her heart condition gave no anxiety. That evening she felt very well, was not sick, and had much less dyspnœa.

The importance of cardiac irregularities during cyclopropane anæsthesia is difficult to assess. They are easily abolished by adding some oxygen to the mixture, and lightening the anæsthesia. They also disappear if the anæsthesia is deepened. Some authorities[18] consider that there is a band in the planes of narcosis where arrythmias occur, and above and below which they disappear. I found that cardiac irregularities were present in twenty-eight patients. Of these, twenty-two (78 per cent.) occurred in the first five hundred cases, whereas only six were seen in the second half of the series. It may be that the slower induction and deepening employed later were responsible for the disappearance of this disturbing feature. At any rate I notice it much less frequently now than at the start. The usual irregularities I met were extra systoles, tachycardia, and bradycardia. Several times one found a patient coming to the table with a pulse rate of 72, and after the cyclopropane had been going for five minutes it would drop to 50, and remain at this rate for about ten minutes, to settle down again at 72 per minute.

Three of the cases were diabetics, two of them having rather lengthy operations. All stood the operation well. Only one had a little post-operative nausea, a factor of paramount importance in stabilising diabetics after operation.

Jaundiced patients have always presented a difficult problem to the anæsthetist. The patient's metabolism is seriously impaired, and yet the nature of his operation necessitates good relaxation, only provided by deep anæsthesia. In the days of chloroform and ether this was almost impossible without endangering the patient's life. With cyclopropane, however, good relaxation can be obtained with practically no metabolic upset, as it has no effect on the liver. My series included six patients with jaundice. In anæsthetising these I preferred to pass an endotracheal tube. This obviated the need for very deep anæsthesia, and yet gave good relaxation. I have only notes of three of these after operation, and none of these three had any post-operative vomiting.

Six patients needed operation despite a co-existent chest condition, but only

one of these had any exacerbation after the operation, and even this was of little consequence.

In 757 of the cases I kept ten-minute records of the patient's blood pressure and pulse rate. These follow a fairly regular pattern with cyclopropane. Most patients coming to the operating theatre have a fastish pulse rate, from 80 to 160 per minute. After anæsthesia is induced with cyclopropane it almost invariably falls to 72 per minute. This would suggest that the tachycardia is due to excitement. At the same time the systolic blood pressure rises about 10 to 20 mm. Sometimes the blood pressure comes down as well as the pulse rate at the start, if it had been raised due to nervousness. The subsequent course of the blood pressure and pulse rate graphs depends on a variety of factors. In the great majority of operations the pulse rate will remain constant at 72, and the blood pressure will also remain constant, round about 10 mm. above the level when the patient came to the theatre. In this respect cyclopropane differs from ether, with which there seems to be a great tendency for the blood pressure and pulse rate to vary a lot during the operation. With cyclopropane these both keep fairly constant for the first two hours' operating. After that the blood pressure usually drops a little. The best way to overcome this is to set up a glucose-saline drip, preferably beforehand, when one expects a longish operation. If necessary, blood can also be given to the patient through the drip. By varying these and the rate at which they are given, one can control the blood pressure during the operation. In my experience the usual analeptics, coramine, methedrine, etc., are not of much value in a falling blood pressure during operation. Any rise that may occur is but temporary. A better plan is to find the cause, and remedy it, for example, the anæsthesia becoming too light, or severe hæmorrhage, or rough handling of viscera. In prolonged operations one often notices that the blood pressure tends to rise soon after the surgeon commences to close the wound.

If the carbon dioxide is not being efficiently absorbed, due either to an obstructed airway, or to the soda-lime becoming exhausted, the presence of a rising carbon dioxide content in the anæsthetic mixture is indicated by a rise in blood pressure accompanied by a rise in the pulse rate. When this state of affairs is remedied both settle down quickly to their former levels. If the anæsthesia is becoming light, possibly the first indication one receives with cyclopropane is the fall in blood pressure of 10 to 20 mm. with an increase in the pulse rate of 10 per min. This is what one would expect, a slight degree of shock, as the patient is not being protected fully by the anæsthetic from the baneful effects of surgical stimuli. With avertin as a premedication before cyclopropane there is the usual fall in blood pressure and fall in the pulse rate associated with this basal anæsthetic. This continues until the patient comes to the table, when the administration of cyclopropane raises both pulse rate and blood pressure. This latter then keeps round the normal range, while the pulse rate remains about 100 throughout the operation. A similar fall is seen when pentothal is employed before cyclopropane. Here, however, the pulse rate varies little, but the blood pressure drops, about 20 to 30 mm., and remains down until the pentothal has been all eliminated from the system.

Contrary to what one might expect, patients with an initially high blood pressure do very well with cyclopropane. Six patients in my series had a systolic pressure of over 200 mm. These seemed to fall into two groups as far as the subsequent pressure records were concerned.

(1) Patients with an accompanying fast pulse rate. In these the blood pressure drops about 60 to 70 mm. simultaneously with the usual fall in the pulse rate at the start of the anæsthesia. In these the hyperpiesis was possibly caused by extreme nervousness.

(2) Patients with a normal pulse rate. In these the blood pressure chart corresponds to the normal picture, that is, a rise of 10 mm. keeping at much the same level throughout the operation. None of these patients had any post-operative cardio-vascular upset, their convalescence varying little from normal patients.

The patients who had a sudden excessive hæmorrhage during the operation present a fairly typical blood-pressure and pulse-rate chart. Such a picture is frequently seen in operations for the radical removal of the breast for carcinoma. In these cases there is a fairly smart hæmorrhage for a time, but it is quickly brought under control. When this occurs, the systolic pressure drops, often by about fifty per cent. After the bleeding is stopped the blood pressure rises steadily almost to the initial level, especially when the skin is being sutured over the large raw area. During this short period of lowered pressure the pulse rate often remains the same, round about 72. I have found that in these cases it is not necessary to give coramine or other analeptic, as the blood pressure rises quite well without it. It would seem that the blood-pressure recordings give a much earlier warning of serious hæmorrhage than the pulse rate, for it is only with persistent loss of blood that the pulse rate rises. Then it is more difficult to improve matters. Sometimes shock is present during an operation, apart from hæmorrhage. Shock is said to be present when there is more than a twenty-five per cent. fall in the systolic blood pressure accompanied by a rise in the pulse rate of over 25 per cent. One of my cases illustrates this well. A young lady of 19 was having an amputation through her hip joint under cyclopropane. All went well until the surgeon cut the sciatic nerve. Then her systolic blood pressure fell from 110 mm. to 60 mm., and the pulse rate rose from 84 to 120. Unlike the fall in blood pressure due to hæmorrhage, the patient did not recover from her shock until back in bed.

Another common cause for a fall in blood pressure during an operation is the unduly rough handling of viscera, especially those of the upper abdomen. One cannot stress too much the need for slow, gentle manipulations inside the abdomen. Much handling of the viscera calls for a considerable depth of anæsthesia. This, and the surgeon's rough handling, rapidly produce shock. The raising of the kidney rest on the table either for a nephrectomy or a cholecystectomy often shocks the patient. The condition, however, improves immediately the rest is lowered. Changing the patient's position on the table, say from the Trendelenburg to the lithotomy positions, may cause a fall in blood pressure, if not done gently.

One patient reacted adversely to the addition of adrenalin to the novocain used for

local infiltration. This was a very nervous and excitable woman of 34 with an exophthalmic goitre. Her pulse rate remained at 96 per minute for the first fifteen minutes, but when her neck was being infiltrated with one per cent. novocain to which a little 1 in 1000 adrenalin was added, her pulse rate rose suddenly to 180. For the next fifteen minutes, despite the administration of 500 cc. oxygen per minute, the patient was cyanosed. At the end of this period the cyanosis suddenly cleared up, the pulse rate came down to 80, and remained so till the end of the operation. Apparently the trouble here was that the adrenalin, being injected into a very vascular area, had entered a vein. When novocain is being used for a field block now I prefer that adrenalin be not added to it if cyclopropane is to follow.

It is quite a common experience with cyclopropane to find that a patient's blood pressure and pulse rate are perfectly satisfactory on table at the end of the operation, but when he is back in bed his condition has sadly deteriorated. Two factors may be responsible for this. (1) The too sudden change from the high percentage of oxygen which he has been breathing for the previous hour or more to the relatively low oxygen content of atmospheric air. This may be overcome by adding some air gradually to the anæsthetic mixture at the end of the operation, instead of removing the facepiece suddenly. It is a good plan, too, to allow the patient's carbon dioxide to accumulate while the air is being added. This ventilates the lungs, which have not been fully expanding during the operation, due to the quiet breathing associated with the adminstration of cyclopropane. (2) A second factor in the causation of post-operative collapse is rough handling in transferring patients to bed. Unfortunately, both in hospital and in private nursing homes, the methods employed in returning patients to bed after operation can only be characterised as crude and awkward, and detrimental to the best interests of the patients. They are either dumped at arm's length from the table to the trolley, and from the trolley to the bed, or else put on a flexible stretcher, and twisted and hauled round narrow corridors, up steep stairs, to be finally flung by exhausted carriers in a heap on the bed. Both of these methods produce shock to the patient, and unnecessary fatigue to those who make the transfer. This may be remedied by having a stretcher under the patient during the operation. This stretcher should have two channels in the edges running the full length to hold poles, which are then lifted gently and easily by two persons, preferably porters, one at either end. Failing this the patient can quite easily be lifted by two people only. These both stand at the same side of the patient, and between him and the trolley. They both put their arms underneath the patient, and while still on the table he is rolled gently over on to his side, facing the lifters. Then the patient is carried, not at arm's length, but on the lifters' chests, and carefully laid on the trolley or bed. The leverage on the arms is small, and the patient can be carried in comfort, and with gentleness. It is surprising how, after a little experience of this method, two persons can lift quite easily a man of fifteen stones weight. It seems to me a great pity that more attention is not paid to the transport of the unconscious patient. It is all wrong if, after the surgeon and anæsthetist have been concentrating their attention for one and a half hours to avoid producing shock, a five-minute transfer to his bed gravely shocks him.

I have used various combinations of drugs for premedication with cyclopropane. Over seventy per cent. of my patients received omnopon-scopolamine, chiefly because of the ease with which it is administered, usually by a nurse one hour before the operation is due to commence. Unfortunately it has several drawbacks. (1) The dose cannot be easily adjusted to meet the varying needs of patients. (2) In adults it is almost always inadequate as an amnesic, even in full doses. (3) Many patients have an idiosyncrasy for omnopon or morphine, and suffer much from post-operative vomiting. (4) Omnopon often so depresses the respiratory centre that the subsequent use of cyclopropane readily produces apnœa. These drawbacks can be overcome by using other drugs prior to cyclopropane.

The most accurate premedication, as far as dosage is concerned, is avertin. This I have used in eighty cases. The dose is measured according to the patient's weight, and administered per rectum. Avertin produces quiet basal narcosis, perfect abdominal relaxation, absolute amnesia, and an almost complete absence of post-operative vomiting. There is one serious drawback to avertin premedication. It must be prepared immediately before administration, and its use entails the presence of the anæsthetist at the nursing home at least three-quarters of an hour before the operation, an impossibility for the busy anæsthetist, who finds great difficulty in getting his day's work all fitted in. I have frequently used the barbiturates with cyclopropane. Here more accurate dosages are possible than with omnopon-scopolamine. Sodium pentothal is most useful. It is administered intravenously, when just sufficient is given to produce the desired result. Pentothal may produce some apnœa in the early stages of the subsequent anæsthesia, but it passes off quickly. The blood pressure is inclined to keep low until the pentothal is all detoxicated and excreted. Seconal I often employ in children, increasing the dose according to age. My experience with seconal is that, when the dose exceeds $1\frac{1}{2}$ gr., the patient needs 1/80 gr. atropine if the salivary secretions are to be adequately controlled. Sometimes one is asked to give cyclopropane in an emergency to an unpremedicated patient. Then I prefer to give the patient 1/100 gr. atropine, followed in five minutes' time with sodium pentothal, just prior to the commencement of the inhalation anæsthesia. This makes for a smooth, quiet induction, and often the patient has no sickness afterwards. Atropine alone makes for continual difficulty in the subsequent anæsthesia. Some narcotic drug is essential before cyclopropane.

The endotracheal technique is frequently of value in the administration of cyclopropane. I have used it in about ten per cent. of my cases. Besides being of advantage in operations about the head and neck, it produces smoother anæsthesia in upper abdominal surgery. Then the operation can be carried out at a lower level of anæsthesia, with the result that the patient is in better condition leaving the table. An endotracheal tube in situ makes the anæsthetist's task lighter if apnœa supervenes, and he is called upon to do artificial respiration by squeezing the rebreathing bag. Endotracheal anæsthesia is a *sine qua non* in thoracic surgery when controlled respiration is being employed. With cyclopropane alone the introduction of the tube through the glottis is a matter of some difficulty, due to

the shallow respiration. I have found it useful to add a little ether to the anæsthetic mixture for a few minutes at the start, as this stimulates the respiration, opens the glottis wider, and makes for easier intubation, which in most cases is performed blindly.

One of the great advantages of cyclopropane over chloroform and ether is the relative absence of post-operative nausea and vomiting. In my series I have records of the incidence of vomiting or of its absence in 606 cases. I have divided these into three classes: (1) No vomiting; (2) when the patient vomited a little during the first twenty-four hours after operation; (3) gross vomiting, when it was continued for several days. In these 606 cases 438 (seventy-two per cent.) had no vomiting whatever, 154 (twenty-six per cent.) vomited only two or three times the same day, while in 14 (two per cent.) the nausea and vomiting persisted for more than twenty-four hours. Only one of the patients had a severe bout of vomiting, for she continued to feel sick, and kept retching for almost a week. This I feel was largely neurosis, as she said that she had been sick for a week after a previous chloroform-ether anæsthesia, and was sure to be sick again. Her retching seemed to occur mainly when she had someone in the room with her. These figures, I believe, could be improved with a better choice of premedication, and a little psychological persuasion before the operation.

As I have mentioned already, in many patients the use of omnopon-scopolamine predisposes to nausea and vomiting. I feel that the use of the barbiturates rather than opium and its derivatives would reduce this tendency. In this connection it is interesting to notice that all the midwifery patients, with one exception, had no post-anæsthetic vomiting. This applies to normal midwifery cases, to those who had a forceps delivery, and to those submitted to cæsarean section. None of these patients had any morphia or omnopon. Most had seconal and scopolamine, a combination which seems to suit midwifery cases very well. The one midwifery patient who vomited had so quick a second stage that she had no scopalomine, and had been given some chloroform before my arrival.

Another factor in the causation of post-operative nausea is the long periods before and after operation during which the patient is deprived of food. This applies particularly to patients operated upon in the late afternoon, when the patient, after being without food the previous night, has had very little during the day, whilst awaiting the operation. I have found that when patients give a history of previous post-operative vomiting of any degree, it is a good plan to give them 100 cc. of concentrated glucose solution intravenously immediately after the operation. Frequently one noticed that the patient vomited a little either before leaving the table, or just after being put back to bed. This happened before the patient was fully conscious, and often he had no memory of the vomiting. One very satisfactory feature of cyclopropane anæsthesia was the fact that none of the diabetics vomited. This is of great importance, and it is desirable to get these patients' diets stablised as soon as possible after operation. The diabetics included in the series all did very well with cyclopropane. Ten units of insulin were given

with the premedication. One of these patients had a hysterectomy done, the operation lasting for two hours. She was in excellent condition afterwards.

I have not found that the incidence of vomiting is materially affected by the duration of the anæsthesia with cyclopropane. It seems to be much more common in women than in men. Of the 166 patients who were sick after operation, 140 were women and 26 were men, that is, five times as many women as men. This would point to a psychological basis for the vomiting. Another thing I noticed is that most patients who have had the cervix dilated vomited after operation. Also, the application of radium to the uterus invariably produced nausea and vomiting until the radium was removed. Thus, post-operative nausea and vomiting cannot always be blamed on the anæsthetic used.

One or two disadvantages have been noticed in the use of cyclopropane. A general capillary oozing from the skin wound is observed, particularly when the wound is being closed at the end of the operation. This, I believe, is due to three factors : (1) The patient's blood pressure remains fairly high throughout the operation; (2) the use of antiseptic solutions containing spirit applied to the skin edges; (3) the capillary dilatation produced by the cyclopropane itself. These all produce a dilitation of the cut capillaries, and the wound starts to ooze. This is somewhat annoying, but it is never of such serious proportions as to produce a hæmatoma of the wound afterwards. Cyclopropane does not alter the clotting time of the blood.[3]

Another disadvantage is the sudden alteration in the depth of anæsthesia that is sometimes noticed with cyclopropane. The patient may quickly become light and incommode the surgeon. When the anæsthesia is being deepened, if it be done too rapidly, he may as suddenly become anoxæmic, to the annoyance of the anæsthetist. Again, the sudden failure of the oxygen supply, if unnoticed for a few minutes, can produce an alarming greyish cyanosis. This means that a very constant, close supervision must be given to the patient throughout the operation, as, even with experience, these variations can occur so quickly. Cyclopropane is not an anæsthetic for the novice to handle. There is a much smaller margin of safety than with ether, and the anæsthetist must be quick to recognise the smallest alteration in the patient's respiratory rhythm, pulse rate, or colour. As I have mentioned when dealing with blood pressure, a slight increase in the pulse rate with a reduction in the systolic pressure would indicate that the patient needs more anæsthetic. But sometimes the surgeon is the first to notice that the patient is getting light, when the abdominal muscles tighten a little.

As far as post-operative sequelæ are concerned, cyclopropane compares favourably with the other inhalation anæsthetics. In this series only six patients showed any sign of post-operative respiratory trouble. Of these one had what seemed to be a pneumonia which did not resolve well. An X-ray was taken two months afterwards, when dulness was reported at one base without any fluid. This seems to have been a partial collapse of the lung. Another patient, on the third day after operation, developed a frank lobar pneumonia, which quickly responded to suphapyridine. This patient had been cyanosed during the greater part of the operation, despite the fact that an endotracheal tube had been passed. The cause of the

anoxia was never determined. The other four patients had varying degrees of post-operative cough, without much in the way of physical signs. All had abdominal operations, and all had slight colds before operation. One felt that the cause of the persistent post-operative cough in the majority of these cases was a reluctance on the part of the patient to clear the air passages of secretion on account of the pain caused in the wound. A few doses of a stimulating expectorant work wonders for these people.

Two patients developed auricular fibrillation after operation, but this cleared up with rest in bed.

The question of post-operative complications leads us to the consideration of deaths following cyclopropane anæsthesia. These I have divided into three categories. (1) Those who died on the table. (2) Those who died inside twenty-four hours after operation. (3) Those who died after twenty-four hours, before leaving the hospital or nursing-home.

Two patients died on the table. One of these was a woman of 50, who was brought for examination under anæsthesia. She had a complete paralysis of her left vocal cord, with a paresis of her right cord. She had a very poor myocardium, and had been given intensive courses of anti-syphilitic treatment. During induction, and before any operative interference, the patient suddenly collapsed, with cardiac and respiratory failure. A post-mortem examination revealed gross pathology in the liver, heart, kidneys, and spleen. This was apparently a case of primary cardiac failure due to cyclopropane, in a very bad-risk patient. The second death on the table was in an elderly woman of 62, who had a fractured neck of femur, for which a Smith-Petersen pin was being inserted. Shortly after the reduction of the fracture this patient became an ashen-grey colour, and remained so throughout the operation, which lasted forty minutes. Her condition did not improve despite the fact that her respirations were ample, and the oxygen content of the mixture was kept high. Just as the operation was being finished she died. It seems as if death in this case was due to an embolic infarct in the pulmonary artery, a not unusual complication of a fracture of the neck of the femur. The manipulations necessary for the reduction caused a clot to be dislodged, with fatal results, which could not in any way be blamed on the anæsthetic used.

Three patients died inside the first twenty-four hours after operation. The first of these was a young woman with a bad albuminuria of pregnancy. When she was brought to the table for a caæsarean section she was comatose, and never regained consciousness before she died, twelve hours later. The second was a healthy old man of 78, having a prostatectomy. His condition during operation was very good except for excessive hæmorrhage. A blood transfusion given shortly after operation improved him, but a further severe hæmorrhage late that evening caused his death. The last patient in this class was a man who was a very bad risk, and for whom a gastrectomy was done. He had been greatly dehydrated before operation, and his condition was always poor, even though an intravenous blood drip was started before the operation. The gastrectomy took three hours, and,

while he seemed fairly well later in the evening, he died twelve hours afterwards. In none of these three cases could the anæsthetic be blamed for the death.

Sixteen patients died before leaving the nursing home or hospital, at periods varying from two days to six weeks after operation. Half of these deaths were due to the fact that the condition for which the operation was performed was unrelieved. As might be expected, most of these were malignant conditions. One was a child of one year with an intussusception, where an extensive resection of gut was necessary. After seeming well the day following operation, he died on the second day. I have included in this group the young man mentioned above as having a thoracoplasty done even though he was very ill. He died on the fifth day. Three patients died due to cardiac trouble supervening after operation. One woman after a cholecystectomy took a sudden heart attack two days later, and died immediately. Another patient had a third dose of radium applied to her cervix, and died suddenly on the fifth day from coronary thrombosis. She had taken the two previous anæsthetics perfectly. The third patient did well for ten days before her heart suddenly gave out. In three of the cases nothing definite could be ascertained as to the cause of death. Two of these had extensive operations for cancer. One died on the third and the other on the fifth day after operation. The third was an elderly patient who had a cholecystectomy, but she died on the tenth day. Two other deaths remain to be described. One was in a patient of 45 who had a hysterectomy, and after doing quite well for seven days without any respiratory trouble, suddenly developed a broncho-pneumonia, and died on the tenth day. The other patient was also a hysterectomy, who developed an entero-coccal infection of her kidneys, and, after weathering a thrombo-phlebitis in both legs, died of a pulmonary infarct on the forty-second day. It will be seen that none of these late deaths could be attributed, either directly or indirectly, to the cyclo-propane, as in all cases the patients had recovered from the anæsthesia, and were in fairly good condition the day following the operation.

Little remains for me except to sum up our findings. It would seem that in cyclopropane we have an anæsthetic of almost equal potency with chloroform and ether, yet without many of their inherent disadvantages. Induction is quiet and pleasant for the patient, and, while relaxation may not be as good as that obtained with other inhalation or spinal anæsthetics, a sufficient degree can be obtained for most operative procedures, especially if the patient is intubated. The chief advantage of cyclopropane seems to lie in the relative absence of adverse post-operative complications. The distressing nausea and vomiting, the frequent respiratory troubles, and the slow excretion of the drug associated with ether anæsthesia, are all conspicuous by their absence. The administration of cyclo-propane demands constant, close supervision of the patient, and, especially in longer cases, continuous records of blood pressure and pulse rate. In the bad-risk patient, the patient with a poor myocardium, the bronchitic, the frail elderly woman, cyclopropane is seen to its greatest advantage. It is surprising to see how well these patients are the day following operation. In midwifery, cyclopropane has no rival; the imperfections of gas and air, the dangers of chloroform, and the

difficulty of administering ether, are all abolished by its use. In cyclopropane, then, we have a valuable anæsthetic agent, of high potency and low toxicity, of ready flexibility in its administration, and of relative freedom from unpleasant post-operative sequelæ.

In conclusion, I should like to pay a tribute to those surgeons whose patients I have anæsthetised with cyclopropane during the past seven years. I would like to thank them very sincerely for their patient longsuffering with me in those days before one became thoroughly acquainted with the new drug, when anæsthesia was far from perfect, and also for their valued help in my investigations, and for their kindly encouragement at all times.

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REVIEW

MEDICAL ASPECTS OF GROWING OLD. A. T. Todd, M.B.Edin., M.R.C.P.Lond. Bristol : John Wright & Sons. 162 pages. 15s.

IN his preface the author declares his threefold objective : "to assist the medical practitioner . . . . to help the elderly subject to make the most of his later life . . . . and for those not yet in later life for the correction of those many errors which may be made by them before extensive ravages have occurred." It may be said at once that he has succeeded as well as anyone may hope to succeed in the impossible task of writing for doctor and layman in the same text. A critical medical reader will find a good deal to criticise, as, for example, when he is told, "The full or irritated stomach appears to have some priority over activities, and it will not let us breathe freely, and unless we take things very quietly we find we are short of breath, and if we make ourselves work in spite of this shortness of breath, we soon experience pain about the heart which will soon radiate pangs down the arms—which is the beginning of angina pectoris or so-called effort syndrome."