A Post-Graduate Lecture

ON

CIRCULATORY CHANGES IN ANAESTHESIA
AND THE USE OF OXYGEN.*

BY

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In considering the choice of a general anaesthetic we are guided by the fact that ether is far less liable to cause dangerous symptoms than chloroform and ought, therefore, to be always used in preference, excepting only cases where very special circumstances indicate the use of the latter. Chloroform is naturally preferred by those ignorant of its danger because it is less irritant, and, being potent in less concentration, excites less feeling of suffocation. Chloroform may have to be used in cases where excitement is to be avoided and very deep anaesthesia is to be obtained with complete muscular relaxation, in cases of bronchitis where the irritating effect of ether must be avoided, in the case of drunkards in whom ether sometimes fails to produce anaesthesia, and in cases where naked lights are necessary on the actual cautery used (that is, when the inflammability of ether forbids its use), and in very hot climates where, owing to rapid evaporation of ether and want of a proper closed apparatus for inhalation, it is difficult to produce anaesthesia. But in many of such cases the safe anaesthetic nitrous oxide and oxygen, if available, should be chosen, or the new anaesthetics recently introduced: 82 per cent. ethylene and 18 per cent. oxygen, which mixture seems safe apart from its inflammability (4 per cent. with 96 per cent. air forms an explosive mixture), or acetylene and oxygen, which, it is claimed, give fuller relaxation of the muscles and form a safe anaesthetic. Propylene is also said to be a safe anaesthetic. A mixture of 70 per cent. propylene, 25 per cent. oxygen, and 5 per cent. nitrogen induced analgesia in one minute, anaesthesia in two minutes, and respiratory failure only after sixteen to twenty minutes. The animals were easily revived after respiration had ceased for half a minute. Nitrous oxide and oxygen form an ideal anaesthetic, safe during, and leading to no bad effects following, anaesthesia, while in the case of chloroform, even after anaesthesia has been successfully conducted, necrosis of the liver follows in a not inconsiderable percentage of cases. We know that chloroform is a general cell poison, but as the cause of susceptibility of the liver in certain cases is unknown, no precautions can be taken against this accident other than shortening the period of anaesthesia, avoiding want of oxygen, and using a concentration no higher than necessary to secure anaesthesia. Such mixtures as A.C.E. are dangerous on account of the chloroform they contain, and their use should therefore be avoided just as much as that of chloroform. The conclusion reached by the American committee on anaesthesia was that the use of chloroform as the anaesthetic for major operations is no longer justifiable. For minor operations also the use of chloroform should cease. As a means of avoiding the ill effects of a prolonged period of ether excitement—in alcoholic and other difficult subjects—the temporary employment of chloroform is, perhaps, sometimes the lesser of two evils. It is justifiable only when nitrous oxide is not available. If a change to chloroform has to be made, it should be made early, as soon as the difficulty of anaesthetizing with ether becomes evident. I wish to emphasize the point that chloroform should never, in any circumstances, be administered after a prolonged period (ten to fifteen minutes or more) of ether excitement. Even a small administration of chloroform is then peculiarly liable to induce respiratory or cardiac death. As

soon as full anaesthesia is obtained ether should be substituted.

"Physiological experiment," said Waller, "shows that ether is five or six times safer than chloroform—that is to say, it takes a concentration of ether five or six times greater to produce poisoning of living tissues." The margin of safety in administration of ether is obviously, then, very much greater. Cushman says that no less than thirty-six times more ether than chloroform must be added to the blood in order to bring the frog's heart into a condition of diastolic standstill.

"The moderately healthy patient under ether," says Hewlett, "may be raised into the sitting posture; he may be subjected to an operation before he is very deeply anaesthetized. His air supply may be so restricted that a considerable degree of cyanosis is occasioned; and yet his circulation will not show that liability to comparatively sudden fluctuations and depressions which undoubtedly occur under chloroform." In addition, under ether "respiration usually is so obvious and audible that any alteration in the function of breathing may at once be detected"; the sleep-like and inaudible respiration not infrequently met with under chloroform is almost unknown under ether. Under ether the pulse is full, bounding, and regular, the blood pressure about normal, the face is abnormally flushed, and incised parts, more especially in the neighbourhood of the neck, are very vascular. "With an overdose of ether, the circulation at the moment when breathing ceases is sufficiently satisfactory for remedial measures to be almost invariably successful, early syncope as under chloroform does not occur." It is only in bad subjects with dilated, fatty, or diseased hearts that syncope may occur under the asphyxial strain of inducing ether anaesthesia. But under chloroform the healthiest subjects may die from heart failure.

Ether anaesthesia should be induced quickly and kept as uniform as possible to prevent excessive breathing and consequent washing out of carbon dioxide from the body, which may result in a period of apnoea, the concentration of carbon dioxide in the blood being the natural stimulant of the respiratory centre in the brain. During apnoea want of oxygen ensues until the centre becomes excited by this; but to produce such want of oxygen is unwise. The poisoned centre may fail to be excited.

Want of oxygen by itself, if sufficient, results in anaesthesia. Consciousness is suddenly lost on breathing nitrogen for some fifty seconds, and speedy minor operations, such as extraction of teeth, have been done under such anaesthesia. But we know that prolonged want of oxygen, as in those rendered insensible by breathing carbon monoxide, leads to degeneration of tissues, such as the heart and brain, from which recovery is difficult or impossible.

Want of oxygen has an injurious effect on living cells such as chloroform or ether has, and adds its ill effect to theirs. Diabetics suffer especially from want of oxygen, some degree of which always accompanies the use of these anaesthetics. For them such an anaesthetic as nitrous oxide and oxygen is most necessary. But for all cases want of oxygen should be avoided as much as possible.

Ether and oxygen has been used, but this mixture is highly inflammable. A mixture of such has recently exploded in a patient's throat, probably through overheating of a syringe which was used to dry the teeth in a dental operation.* The trouble about using nitrous oxide and oxygen for a prolonged general operation is that it is impossible to get complete muscular relaxation without bringing into play some want of oxygen by lowering the concentration of oxygen and raising that of nitrous oxide in the inhaled air. A mixture of 40 per cent. air and 60 per cent. nitrous oxide would contain about 8 per cent. oxygen and about 32 per cent. nitrogen, and although the oxygen would just suffice to keep the patient going the 60 per cent. of nitrous oxide would be insufficient to produce tranquil, deep anaesthesia. By using oxygen in place of air we replace nitrogen by nitrous oxide, and the proportion of the latter can be raised to 80 per cent. and better anaesthesia obtained.

* Being the third of three lectures on general anaesthesia delivered under the auspices of the Dental Board of the United Kingdom in 1925. The two other lectures, which were by Professor Yandell Henderson, were published in the BRITISH MEDICAL JOURNAL on December 19th, 1925 (p. 1170), and January 9th, 1926 (p. 41).

* A temperature of 200° C. suffices to effect the explosion.

Nitrous Oxide.

By raising the concentration of nitrous oxide still further deep anaesthesia with full relaxation of the muscles is obtainable. Paul Bert effected this by using a pressure chamber as an operating theatre, in which patient, surgeon, anaesthetist, etc., were all contained. A short length of tube railway would do for such a chamber, closed at one end completely, and provided with a suitable air lock for passing in and out at the other end, and an air pump for raising the pressure and maintaining ventilation. The required increase of pressure is very small—for example, half of an atmosphere. In place of, say, 90 per cent. of an atmosphere of nitrous oxide and 10 per cent. of an atmosphere of oxygen, we should then have $90 \times 1.5 = 135$ per cent. of an atmosphere of nitrous oxide and $10 \times 1.5 = 15$ per cent. of an atmosphere of oxygen, which will give deep anaesthesia and enough oxygen. The circulation and respiration are in no way influenced by this anaesthesia, which may be maintained for several hours. If an animal is kept warm it will, as Dr. H. H. Dale and I have found, confirming Bert, at the termination of anaesthesia be in splendid condition without any sign of shock, and able at once to run across the room fully alert. Such a method of anaesthesia is safe even in the hands of students; there is no difficulty in constructing and using such an operating theatre if the demand for it were made. Through the air lock people could pass quickly in and out, and inside everything would be conducted just as in an ordinary operating theatre, the gas and oxygen mixture being administered in the ordinary way, all inside being submitted to a slightly increased atmospheric pressure. The only precaution to take would be opening of the Eustachian tubes by a deep expiration with mouth and nose shut, so as to equalize the pressure within and without the middle ear. People with tubes swollen by catarrh might be troubled in effecting this. Tunnel workers carry out work in pressures up to two, three, and even four atmospheres in the caissons which are used to keep water out when boring under rivers; half an atmosphere extra pressure is, then, of very little account.

Nitrous oxide and oxygen offer an ideal anaesthetic for dental surgery; the average time of inhalation Hewlett found to be $110\frac{1}{2}$ seconds, the average available time 44 seconds, but varying from 21 to 90 seconds. The addition of a small dose of ethyl chloride is effectual in prolonging the anaesthesia, and appears to be a safe proceeding.

When pure nitrous oxide is administered with the ordinary valved apparatus there results anaesthesia, partly from this gas and partly from deprivation of oxygen. To-and-fro breathing during the last part of the inhalation allows some small amount of oxygen to be retained, and a longer inhalation and period of anaesthesia to be obtained. The exhaled carbon dioxide which is thus re-breathed deepens the breathing. There is, however, a disadvantage in breathing into the bag, because it is difficult to clean and disinfect it for another patient. By using an apparatus such as the Salvus used in mine rescue work, with a caustic soda absorber placed between the mouth mask and the bag, this trouble might be obviated. Each patient would have his caustic soda cartridge to breathe

through. A suitable modification would have to be made to secure sufficiency of oxygen for prolonged anaesthesia.

The circulation is well maintained during nitrous oxide anaesthesia, providing too great a degree of asphyxiation is prevented; the pulse increases in frequency and loses in fullness; flabby, stout patients become deeply cyanosed; the moment air is breathed the pulse becomes less frequent and fuller; the muscular spasms which occur are due to want of oxygen. To obtain the best results, then, it is necessary to eliminate asphyxial symptoms, and mix oxygen with nitrous oxide. All the deaths which occur from use of this gas appear to be due to fright or to want of oxygen, and not to the poisonous nature of the gas. If the air leaks into a mask during inhalation of nitrous oxide, the oxygen in this air is quickly absorbed by the patient, and the nitrogen, which forms four-fifths of the air, remains and dilutes the nitrous oxide; thus want of oxygen increases, and anaesthesia results more from this than from nitrous oxide. It is necessary to have a close-fitting mask to avoid such leaks.

Chloroform.

Chloroform is a poison to living cells, acting perhaps on the fatty surface film which controls permeability and adsorption. On the heart it causes dilatation and weak action, and under certain conditions fibrillar contraction, the cause of sudden syncope and death. Added to oxygenated blood it dilates the blood vessels of surviving organs, but constricts these if the blood lacks oxygen or is replaced by Ringer's solution. It relaxes the skeletal muscles which support the veins, thus enlarging the capacity of these great veins into which the whole of the blood can easily drain from the upper part of the body when the body is put in the upright position. The cross circulatory experiments of Gaskell and Shore showed that chloroform does not affect the vasomotor centre so as to lower the arterial pressure in the early stages. It may even excite this and raise the arterial pressure at first. In very deep anaesthesia the vasomotor centre is depressed in action just as the respiratory centre is, and arterial

dilatation may take place. With ether also, after an hour or so, of anaesthesia, progressive dilatation sets in. The respiratory pump helps the circulation greatly, furthering the return of blood by rhythmic compression of the abdomen and rhythmic enlargement of the lungs, in which enlargement the pulmonary vessels share. When the respiration pump is enfeebled by deep chloroform anaesthesia everything tells against the circulation—the dilated feeble heart, the relaxation of the

abdominal veins, and the feeble action of the respiratory pump. Hence there is danger in the upright position of the blood draining into the abdominal veins, the heart almost emptying, and the circulation in the brain almost ceasing. Such conditions help to stop the respiration, the respiratory centre itself being partly poisoned by chloroform and much less responsive to the natural stimuli of breathing.

The capillaries—there are, according to Krogh, some 2,700 in each square millimetre of diaphragm muscle—are supported by the contractile cells of Rouget and by the tissues in which they run. They may become dilated by chloroform, and the blood pool within them as well as in the

FIG. 1.

FIG. 2.

FIGS. 1 AND 2.—Record of blood pressure in aorta and vena cava of a dog anaesthetized with ether, and placed in the vertical position (head up) with the cannulae inserted in the vessels in the axis of rotation. The vena cava pressure falls and the respiratory waves are heightened; the aortic pressure at first falls and then rises, and compensates the effect of gravity for a time; finally it slowly falls, with variations produced by increased depth of respiration as shown by the vena cava record.

FIG. 3.—Record of aortic blood pressure of a dog under morphine. A, The animal was placed in the vertical posture (head up) and the preliminary fall in blood pressure was largely compensated until, B, chloroform was breathed in concentrated doses. The blood pressure then fell steeply. After stopping the administration of chloroform at C, the abdomen was compressed between D and E and the blood pressure thereby largely restored. Placing the animal in the head-down position at F had the same effect.

big veins. In favour of this is an observation of Argyll Campbell that chloroform anaesthesia lowers the tension of oxygen in the tissues, while ether may slightly raise it. Many capillaries in resting organs such as muscle are normally closed. In states of shock the capillaries are dilated, probably by absorption of histamine into the blood from damaged tissues; chloroform and ether increase this effect. Fluid also passes out then from the capillaries into the tissue spaces. The whole body confined by the skin is kept taut and hard, or should be, by the inhibition pressure of the living cells of the tissues. Each organ has its capsule of connective tissue limiting its expansion, in addition to the skin which confines the whole, the brain, spinal cord, and marrow being confined by bone; expansion is thus limited. The tissue cells thus confined, when healthy and in good tone, are swollen with fluid so as to produce a certain tissue pressure, and the capillary pressure is adjusted so as just to balance this and exceed it just to the amount sufficient to cause the flow of blood into the veins, an amount which need be, and is, very small. The conduction of the arterial pulse wave and its driving force depends largely on the tone of the tissues which support the arteries. In shock the tone of the body is generally relaxed, and the blood stagnates in dilated veins and capillaries and sinks to the lower parts of the body. Histamine, a product of tissue damage, dilates the capillaries, and is probably closely associated with the causation of shock. To both pituitrin and adrenaline has been ascribed the function of supporting capillary tone.

Chloroform, by poisoning the tissue cells and relaxing their tone, makes the whole body flabby, turns the circulation from a vigorous one through confined channels in a taut body to a sluggish one in a relaxed body, and the blood in consequence stagnates in dependent parts—hypostatic congestion. The failure of the circulation in deep anaesthesia can be got over by supporting the vascular system, by putting the body horizontal or with the legs and abdomen slightly the higher, or by compressing the abdomen, while failure of respiration can be met by artificial respiration.

Far more dangerous, and the main cause of deaths under chloroform, is cardiac fibrillation, which occurs in the initial stages of anaesthesia, and may be a consequence of too light an anaesthesia and of struggling or sensory excitation. The distinguished medical statistician M. Greenwood has concluded that the condition called status lymphaticus, often made an excuse for fatalities arising from anaesthesia, is a make-believe.

MacWilliam first pointed out that dilatation of the heart occurred, and that this was due to the action of chloroform on the heart itself, and that fall of blood pressure was concomitant with this. In some cases he found the heart thrown into a state of inco-ordinate fibrillar contractions. These waves are quite ineffectual in maintaining a circulation. These facts I confirmed. Goodman Levy has shown that the reapplication of chloroform in concentration of about 2 per cent. or over after an interval may act as a powerful excitant to the upper air passages, and the sensory excitation therefrom may throw a heart, already made irregular by chloroform, into fibrillation. In deeply morphinized dogs inhibition of the heart, reflexly

excited by breathing strong chloroform, may help to embarrass the heart. Prolonged intense stimulation of afferent nerves which reflexly arrest the respiration and inhibit the heart may cause in animals a failure of respiration, to be followed by that of the heart if unrelieved by artificial respiration.

Want of oxygen is another cause exciting the whole sympathetic system, including the accelerator nerves, and causing, probably, increased adrenal secretion, which helps to produce the fibrillation, this being a most powerful cardiac stimulant. An injection of adrenaline may throw the weakly chloroformed heart into fibrillation; but according to MacWilliam, while a small dose has this effect, augmenting excitability, a large dose has the opposite effect, and may be used to stop fibrillation. Direct excitation of the accelerator nerves may provoke fibrillation; painful stimuli in the half-anaesthetized patient reflexly excite these nerves. A cardiac depressant, such as chloroform in full dose, prevents these irritable phenomena—hence the danger of light or irregular administration and safety of deep, full anaesthesia. The latter damps down or cuts out the reflex exciting mechanism, and so protects the heart from any sensory excitation acting reflexly on the accelerator

nerves. Fibrillation is due to a disturbed relation between the conduction time of the stimulus and the refractory period, resulting in the setting up of circulatory excitations in the musculature of the heart. The heart tends to recover spontaneously from fibrillation, but spontaneous recovery in animals does not occur after the first minute or minute and a half. Such spontaneous recovery has even been observed in man by means of an electro-cardiographic record.

Bathing the chick embryo in a suitable nutritive solution to which chloroform is added shows the poisoning action on the heart. So, too, does perfusion of the isolated mammalian heart through

the coronary vessels with blood to which chloroform is added; Sherrington and Sowton found that the heart is weakened progressively as the strength of the chloroform in the perfusion fluid is raised: finally the heart stops beating and does not recover. The strengths of chloroform used for inhalation, however, do not produce this stoppage, because the respiration fails first when too concentrated a dose occurs, and then the heart can be recovered by artificial respiration. The fatal cases of syncope are due to fibrillation, and these can be recovered, according to MacWilliam, by injecting 0.1 to 1 mg. of adrenaline (using a 1 in 10,000 solution) into the cavity of the left ventricle, or by rhythmic massage of the heart. To effect this massage through the chest wall is very difficult in an adult man. The method then to follow is to make an abdominal incision and through this cut the attachment of the diaphragm to the left costal margin for two inches. The left hand is inserted into the left pleural cavity and the heart grasped through the pericardium and rhythmically squeezed, the wrist preventing air entering the pleural cavity. The rate of compression should be about that of the normal heart. Massage should not be abandoned under an hour, short pauses being allowed at intervals. An occasional intermittent compression of the abdomen to fill the heart helps. Perflation of the lungs by mouth-to-mouth method, or by a stream of air or oxygen blown in by

FIG. 4.—Respirations and blood pressure records showing the reaction to adrenaline in a cat under chloroform. *a*, Respiration. *b*, Blood pressure (mercury manometer). *c*, Level of zero pressure. *d*, Signal line. *e*, Time, indicated in seconds. *A*, intravenous injection of half a minim of 1 in 1,000 adrenaline solution when the cat is under 1.5 per cent. chloroform. The blood pressure rises, but the beat remains regular. *B*, A similar injection when the cat is under 0.2 per cent. chloroform. The beat becomes rapid and irregular, and finally ceases abruptly. Following the cardiac collapse the respirations are first greatly exaggerated and then cease entirely. (Levy.)

intubation of the larynx, must be kept up. Artificial respiration can be first tried, but in this desperate condition there must not be more than a very few minutes' delay before direct massage of the heart is begun, because if delay is too long, even though the heart recover, the nervous system is damaged by prolonged oxygen starvation, and the patient dies from this reason. When the heart gradually fails from overdosage, artificial respiration, conducted by squeezing the chest rhythmically, will recover it by bringing fresh blood into the coronary arteries and through the lungs, and so getting rid of the chloroform and admitting a supply of oxygen. MacWilliam finds that injection of 0.5 gram per kilogram of body weight of urethane beforehand prevents fibrillation in animals during chloroform anaesthesia.

Ether.

When ether is the anaesthetic, the symptoms of poisoning are in general the same, but fibrillation or failure of the heart from overdosage is much more unlikely because of the far lower toxicity of the drug. The anaesthetic partial pressure of ether is 3.7 to 4 per cent. of an atmosphere. The solubility of the brain tissue for ether is practically the same as that of blood. Large pulmonary ventilation and rapid blood flow through the brain results in quick anaesthesia.

In every operating theatre there should be available a cylinder containing oxygen and 5 per cent. carbon dioxide. By using this with ether at the beginning of anaesthetization deep breathing is obtained and quicker narcosis. A lower percentage of ether can be used and less irritation of the bronchial mucosa caused. After prolonged ether anaesthesia the colour and fullness of the veins in the skin are restored by breathing this mixture (Yandell Henderson and Haggard). By using it alone after operation the deep breathing excited by the carbon dioxide washes ether out of the body and introduces oxygen; this greatly lessens the after-effects and helps recovery. It is an advantage to give a liberal dose of morphine half an hour before initiating anaesthesia. This takes away anxiety, lessens the period of excitement, decreases pain after anaesthesia, and is an excellent preventive of shock. The breathing of the oxygen CO₂ mixture

after operation counterbalances the depressive action of morphine on the respiratory centre.

In animals chloralose or urethane (6 c.cm. of a 25 per cent. solution per kg. body weight) injected subcutaneously, or into the rectum, gives narcosis with a high blood pressure, and is very convenient.

Ether in oil introduced into the rectum, after washing this out, produces anaesthesia which may be long lasting. This plan has been suggested for use when no help is at hand to give this anaesthetic by inhalation.

Local Anaesthesia.

Turning to local anaesthesia, we know that cocaine is a general protoplasmic poison, and its poisoning action is exerted on the central nervous system, the symptoms being excitement, convulsions passing into coma, and death from asphyxia. The blood vessels are contracted, blood pressure raised considerably, the heart also being accelerated in the stage of excitement. A local contraction of the vessels is caused by the application of cocaine. The artificial alkaloids used as a substitute for cocaine are much less poisonous, and have not the same constricting effect on the blood vessels. Thus novocain is quickly destroyed in the liver, and is for this reason much safer. Adrenaline increases the toxicity of cocaine according to the findings of the committee of the American Medical Association. This committee believes that local anaesthetics may be applied safely in the following concentrations and total amounts: Cocaine in the mouth 5 per cent., in the nose up to 10 per cent., and in total amounts of from 10 to 15 minims containing at most 1 to 1.5 grain. In the eye not over 5 per cent. should be used. In the larynx 10 per cent. and not over 15 minims, containing 1 to 1.5 grain. Novocain in concentrations not greater than 1 per cent. Adrenaline or epinephrin should not be used with cocaine in concentrations greater than 1 in 10,000, and not more than 10 minims of this—with novocain up to 1 mg. of adrenaline—is allowed; but even this

dose may be unsafe in patients suffering with hyperthyroidism (exophthalmic goitre). In poisoning cases the heart must be massaged by artificial respiration to keep up the circulation through the liver; the injection of any other drugs is probably useless, and may increase the danger.

FIG. 5.—Ventricular fibrillation initiated during a fit of struggling in a cat under light chloroform anaesthesia. Upper record: respiratory. Lower record: blood pressure (mercury manometer). Time marked in seconds. The period of struggling is indicated by the irregular respiration record within the dotted bracket. The cardiac collapse is followed by intense dyspnoeic gasps. The heart was inspected immediately on the cessation of respiration, and the ventricles were seen to be fibrillating vigorously. (The declination on the left of the respiratory record is accidental.) (Levy.)

RECOVERY AFTER MASSAGE OF THE HEART.

BY

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THE following case is one of considerable interest, as it illustrates the value of massage of the heart when it has ceased to beat during the performance of a major surgical operation.

History.

A man, aged 50, was admitted under my care to St. Bartholomew's Hospital on September 28th, 1925. His story was as follows: In October, 1913, he was admitted to the hospital with perforation of a gastric ulcer, which was sutured by myself; a posterior gastro-enterostomy was performed at the same time. He made an uninterrupted recovery, and departed for West Africa, where he has been living up to this time. In 1916 he had a bad attack of dysentery. In 1918 he began to have a grinding pain in the neighbourhood of the umbilicus on the left side of the abdomen, extending towards the left iliac fossa; the pain usually came on about three hours after food and lasted for an

hour or two. From time to time this pain would disappear for a period of two or three months; since 1920, however, it had gradually become more persistent, and as a result thereof his life had become very miserable.

On admission to hospital he looked fairly well, but his facies suggested that of a man suffering from constant pain. His teeth had all been removed, so that oral sepsis was excluded as a cause of his symptoms. There was well marked tenderness to the left of the middle line in the epigastric region; over this point of tenderness there was an area of superficial hyperaesthesia. His pain was only partially relieved by placing him on a convalescent diet. The character of the pain suggested that he might have a gastro-jejunal ulcer, and I decided to explore his abdomen to settle this point.

Operation.

On October 2nd he was anaesthetized by the endotracheal gas-oxygen-ether method. No ulcer or scar could be found in the region of the site of the old perforation, but there was thickening around the gastro-jejunal junction on the proximal side of the stoma, which was involving the transverse colon and was obviously the site of a gastro-jejunal ulcer. The jejunum was divided proximally and distally to the stoma and an end-to-end anastomosis made. The transverse mesocolon was then separated from the anastomosis and its adhesion to the colon divided. The ulcer had not actually perforated into the colon; the point of