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EDITOR'S NOTE

It is a matter of regret that this first published volume of the PROCEEDINGS OF THE CANADIAN ANAESTHETISTS' SOCIETY is presented in incomplete form and at such a late date. Unfortunately the decision to publish the volume was taken after the authors of the papers had dispersed following the meeting of June 1952, and there has been some difficulty in collecting these contributions for publication. I have no doubt that this matter will be much simplified in the future.

I wish to express my thanks to those who have cooperated to make the present volume possible.

R. A. GORDON
Editor

ANAESTHESIA FOR CARDIAC SURGERY

Panel Discussion

Chairman—Dr. R. A. Gordon, Toronto.
Members—Dr. A. J. Kerwin, Toronto.
Dr. C. I. Junkin, Toronto.
Dr. S. M. Campbell, Toronto.
Dr. A. F. Pasquet, Montreal.
Dr. N. Nix, Edmonton.

The contributions of the members of the panel follow in this order:—

(1) Physiological abnormalities and handicaps due to the cardiac disease—Dr. Kerwin.
(2) Evaluation of the operative risk and a basis for choice of the type of anaesthesia to be employed—Dr. C. I. Junkin.
(3) Complications and Emergencies arising during operations on the heart and great vessels, and measures taken to deal with these—Dr. Campbell.
(4) The value of procaine in cardiac surgery—Dr. Pasquet.
(5) Complications and care of the patient in the post-operative period—Dr. Nix.
(6) Questions from the floor, discussed by Dr. Junkin.

(1) INTRODUCTORY REMARKS—PHYSIOLOGICAL ABNORMALITIES

Dr. A. J. Kerwin

Patients with congenital heart disease suffer from one or both of two major abnormalities—

(A) The blood takes an abnormal path through the heart or through an adjoining great vessel.
(B) The normal pressure relationships are grossly disturbed, usually in the direction of elevation.

In cases where venous blood by-passes the lungs and is shunted directly into the systemic circulation, varying degrees of arterial unsaturation add to the difficulties of anaesthesia. In cases where the shunt is from the arterial to the venous side and there is a short circuiting or recirculation of blood through the lungs, the problem is much less difficult since the peripheral arterial saturation is normal. Because these abnormalities are congenital the organism has had a chance to become adapted to them as it grows and gross defects can be fairly well tolerated for some years, defects which if suddenly produced in a normal adult would instantly cause disaster. This fact is easily appreciated but is not so often understood that the sudden correction of gross defects e.g. the occlusion of a very large patent ductus, while it may force the blood to pursue the normal route, may impose such a strain on the circulation that readjustment is very difficult.

The two main objectives of operation are (A) Alteration of intracardiac or extracardiac structures so that the flow of blood is in the normal direction, or alternately, the production of some other defect which permits the oxygenation

of blood which was previously deficient e.g. the Blalock operation for tetralogy of Fallot. (B) The relief of excessively high pressures e.g. resection of coarctation of the aorta. The most important single task of the anaesthesiologist during these procedures is to maintain an adequate concentration of oxygen in the brain cells which in turn implies adequate total cerebral blood flow and adequate oxygenation of the blood. In addition to the usual risks of a fall in blood pressure and failure of the respiratory centre, in cardiac surgery there is the additional risk of producing serious arrhythmias as a result of the local handling of the heart. To some extent this can be obviated by the local application of Procaine and by care on the part of the surgeon in manipulation. The "irritability" of cardiac muscle which causes abnormalities of electrical impulse production and distribution which occurs spontaneously in many individuals either in association with serious cardiac disease or even in otherwise normal individuals, can be ameliorated or prevented by the oral or parenteral use of such drugs as Quinidine, Procaine Amide etc. However these are relatively ineffective when given to prevent arrhythmias caused by local stimulation. The choice of anaesthetic is of some importance. It seems well established that Cyclopropaine sensitizes the ventricular muscle, particularly where Epinephrine may be used and may result in serious arrhythmia. The anaesthetic of preference is probably intratracheal ether.

In cases of intraventricular septal defect with over-riding aorta (Eisenmenger's Complex) the pressure in the right heart is very high, in fact almost equal to that of the left side, as it must be in order to maintain circulation through the lungs where there is a large communication between the pulmonary and the systemic pressure areas. If the systemic pressure is allowed to fall the right side will divert more blood into the systemic circuit thus decreasing pulmonary flow and increasing the amount of cyanosis. It is thus very important to maintain adequate systemic blood pressure levels.

A most important consideration is the amount of blood and saline which should be administered during and after operation. There is an unfortunate tendency to overload the circulation which of course adds considerable strain to an already overworked myocardium. If the patient's haemoglobin is normal to start with only as much blood should be given to make up for the loss during operation. If the patient is polycythaemic probably a little less blood than sufficient for replacement should be administered. A similar attitude should be taken with respect to saline since the retention of sodium makes the retention of water obligatory and the total blood volume may be increased to dangerous levels with the production of cardiac failure and pulmonary oedema.

A well administered anaesthetic is of prime importance in these difficult operations and it may make all the difference between success and failure.

(2) EVALUATION OF OPERATIVE RISK AND CHOICE OF ANAESTHETIC

DR. C. I. JUNKIN

In answer to your request that I discuss the evaluation of operative risk in these (cardiac) patients, and a basis for choice of the type of anaesthesia to be

employed. I would at the outset stress the importance of teamwork between cardiologist, anaesthetist and surgeon. We as anaesthetists must rely on the cardiologist first of all for an accurate diagnosis of the anomaly which exists. We must know what hazards we face and what surgical procedure is to be attempted, if we are to anticipate the difficulties which may arise in any given case.

In congenital heart disease a broad but satisfactory division of cases into non-cyanotic and cyanotic types can be made.

The non-cyanotic group may be required to undergo anaesthesia for the elective or urgent surgical problems which befall children with normal hearts, and these may as a rule be treated as ordinary cases but approached with caution, premedication to reduce the fears and struggles in the induction stage and care in the administration of such agents as Ethyl Chloride or Cyclopropane, if these are used at all.

In the patent ductus arteriosus or adult type of co-arctation of the aorta the same rule applies. Either of these two conditions may however demand urgent operation in infancy through the development of congestive failure, which is often unresponsive to medical treatment. Our experience with these cases is that they withstand surgery very well and that operative correction of the defect is life saving. More and more of these non-cyanotic defects are now coming under surgical care and correction—aberrant vascular channels, pure pulmonary stenosis, and septal defects of all varieties being examples.

On the other hand, the cyanotic group constitutes the gravest operative risk and from the anaesthetist's viewpoint is the most serious of all heart disorders. The commonest such patient is the child suffering from the tetralogy of Fallot who undergoes the Blalock, Potts or Brock procedure. It is interesting that the arterial oxygen saturation as measured by the ear oximeter rises during induction of anaesthesia where the inhalation mixture is at least 50% oxygen, and frequently reaches levels above 80% where endotracheal oxygen by assisted respiration is administered. This cannot be explained on the basis of physically dissolved oxygen in plasma. Opening the chest does not produce any significant lowering of oxygen saturation in the majority of cases, but the use of a suction catheter at any period may produce a precipitous drop in oxygen saturation figures. At any stage, but usually during manipulation of clamps on the pulmonary artery with associated collapse of the lung, a serious sinus brachycardia, unresponsive to atropine, may herald the onset of cardiac arrest. Cardiac massage may or may not effect relief. A sharp reduction in blood pressure at any point in the operation may induce thrombosis revealed only in the post operative period by the appearance of hemiparesis or hemiplegia. Ziegler has shown that electrocardiographic abnormalities of rhythm occur in over 75% of such cases of tetralogy undergoing operation. Only a small percentage are detectable without electrocardiographic aid and the majority are clinically unimportant.

Probably the greatest risk of all is operation in the extremely anoxemic infant with cyanotic heart disease of this type who is submitted for operation at an early age in a desperate attempt to save life.

The use of the extra-corporeal circuit for transposition of great vessels produces a normal arterial oxygen saturation immediately, though other grave problems concerned with cardiac function require to be solved with this mechanical aid. Such cases are therefore in a better state at the time of operation than beforehand because of the high oxygen concentrations used in the inhalation mixture and the use of mechanical assistance to respiration.

Post-operative care is equally important. The anaesthetist—the man who controls the plain fluids, plasma and blood entering the infant's circulation throughout the operation—is the logical person to supervise the fluid balance of such children in the critical post-operative period. Each individual varies in his requirements, but the important principle is to avoid excessively salt solutions and to avoid drowning the patient with too much fluid. In the immediate post-operative period a careful watch for the onset of haemorrhage is more than just an academic principle. The onset of pallor, restlessness and particularly of dyspnoea even in the absence of the classical physical signs of pleural fluid demands a diagnostic thoracentesis and rapid blood transfusion if clinical suspicion is confirmed. Such measures are literally life saving.

Most infants are tardy at resuming oral feedings after such procedures and it is often necessary to keep intravenous fluids running at 15 to 20 ccs. per hour or less for two or three days before discontinuing completely. In hot weather the demands for fluid in a small infant are greater and the risks from dehydration are proportionately more significant. Though operation is avoided in hot summer months pre-operative, operative and post-operative attention to the increased fluid requirement may prevent disasters from thrombus formations.

There is no doubt that surgery for congenital heart disease depends on

1. Accurate diagnosis and selection by a medical team.
2. Modern techniques and precautions by the anaesthetist.
3. Rapid and successful surgery by the operator.

No one member of this team is more important than the other, and a breakdown on the part of one jeopardizes the patient's chances of successful progress.

For small patients ether-oxygen plus pentothal and syncurine are used.

But for cardiac cases, as for all transthoracic operations, the first problem the anaesthetist has to solve is adequate oxygenation of vital organs during procedures which may become prolonged. He must be prepared to assist, to control and to carry on mechanical respiration if necessary over considerable periods of time, and the outcome may depend to a great degree on his attention to this one factor, setting aside entirely his roll as a supplier of analgesia and relaxation, the latter being much less a factor than in abdominal surgery. If he fails in this first duty he may wind up with a vegetable on his hands rather than a vital human organism. The heart may resume its rhythmic rate and carry on for hours with higher centres which fail to take back their responsibility for maintaining respirations. There can be no gain in this type of surgery which is commensurate with the efforts expended, materials used and time wasted, unless the organism has a reasonable chance of survival.

(3) COMPLICATIONS IN CARDIAC SURGERY

DR. S. M. CAMPBELL

(*a*) *Acute Pulmonary Oedema.*

The two factors causing this condition are first—sustained increase in pulmonary capillary pressure and second—anoxia. The latter causes increased permeability of lung capillaries and transudation of fluid into the interstitial spaces and through the alveolar walls into the alveoli, thus decreasing the area available for oxygen absorption and thereby increasing the anoxia.

Immediate treatment.

1. Clear out quickly the free fluid in the bronchial tree by suction through the endotracheal catheter.
2. Increase the amount of oxygen available for absorption by giving 100% Oxygen under intermittent pressure through a clear airway.

(*b*) *Alterations in cardiac rate and rhythm.*

(1) Bradycardia—a vagal effect. If the heart rate goes below 66 administer Atropine Sulphate 1/50–1/75 grain intravenously to subdue the vagal preponderance and thus prevent a drop in cardiac minute output.

(2) Tachycardia—A sinus tachycardia may be caused by haemorrhage, general vasodilatation, paralysis or severance of the vagus, a preponderant atropine effect, splanchnic dilatation or shock.

The treatment for haemorrhage, general vasodilatation, splanchnic dilatation or shock is obviously blood transfusion. Where the haemorrhage is massive, intra-arterial transfusion under pressure is necessary to maintain cardiac output and coronary circulation. In order to prevent cerebral anoxia the head of the table should be lowered as well as inflating the lungs with oxygen.

In addition to transfusion methedrine 10 mgm. intravenously or vasoxyl can be used to counteract vasodilatation.

Where the vagus has been interfered with intravenous procaine or procaine amide may be of some value in reducing the irritability of the automatic conductive mechanism of the heart. For a preponderant Atropine effect intravenous Digoxin .25 mgm. is safer than the use of Quinidine which may have a depressant effect on the heart muscle.

(3) Auricular Fibrillation. The patient who has been having Auricular Fibrillation is usually digitalised during the pre-operative preparation. Some of these people in spite of digitalis still fibrillate and the operation is carried out. In these cases the anaesthetist must maintain circulation and oxygenation in spite of the fibrillation.

Where auricular fibrillation developes during the course of the operation intravenous digoxin is justified.

(4) Ventricular fibrillation is a most serious development which in its effect is the same as cardiac arrest. Immediate use of 1% procaine solution on the surface of the heart, 1/50th grain of Atropine sulphate intravenously and intermittent manual compression of the heart at a rate of sixty per minute will usually

return the heart to a nodal rhythm. The use of the electrical defibrillater is certainly indicated here.

(5) Cardiac arrest—The contracted heart is in ventricular fibrillation and I have dealt with that. The heart stopped in diastole is another matter. In this case intracardiac adrenalin is of value to produce a contraction along with rhythmic manual cardiac compression. The electrical stimulator may also be used.

(*c*) *Massive haemorrhage* is best treated by rapid intra-arterial transfusion.

(*d*) *Shock* following transient interception of circulation. For this the head of the table is lowered, the intravenous transfusion is speeded up and vasoxyl or methedrine may be added to the transfusion.

(4) PROCAINE—ASSESSMENT OF ITS VALUE IN CARDIAC SURGERY

DR. A. F. PASQUET AND DR. R. G. B. GILBERT

In the analysis of the present status of Procaine therapy during all types of Surgery, with particular reference to Cardiac Surgery, a number of factors have to be considered. (In this instance, its action on the heart is that which is to be discussed and not its general anaesthetic effect.)

(1) What is the action of Procaine on the heart of animals and man?

(2) What is the action of Procaine breakdown products?

(3) What clinical evidence is there concerning Procaine therapy?

(4) What types of abnormal heart beat are to be expected and in what way may Procaine be of help?

(5) Has Procaine Amide advantages over Procaine?

All are familiar with the early work of Goyanes, Bier, Leriche and Fontaine. The use put to Procaine by Gordon and the early attempts at using Procaine as a general anaesthetic agent are other stepping stones.

Before Cardiac Surgery had reached its present high standard of efficiency and before Procaine had been considered as a prophylactic and curative drug in the treatment of arrhythmias, heart beat irregularities were commonly found following the use of anaesthetic drugs and combinations.

Levy (1) in 1911 reported sudden death occurring under chloroform anaesthesia following the use of adrenalin. Meek, Hathaway and Orth (2) in 1939 reported on Cyclopropane and Adrenalin. In 1936 Kurtz, Bennett and Shapiro (3) reported on the evidence of cardiac irregularities by E.C.G. following a variety of anaesthetic agents, the occurrence appeared remarkably high in all types of anaesthesia.

The story of the use of Procaine in the present text may start with the observation in 1936, of Mautz (4) that Procaine applied locally to the epicardium, reduced cardiac irritability and he felt that it might be of use in Cardiac Surgery.

I. *The Action of Procaine on the Cardio Vascular System of Animals and Man.*

Burstein, Marangoni, DeGraff and Rovenstine (5) in 1940 described the prophylactic and therapeutic uses of Intravenous Procaine to avoid and combat

Ventricular Fibrillation in dogs under Cyclopropane to which adrenalin was administered.

Stutzman, Allen and Orth (6) in 1945 failed to reverse this Ventricular Fibrillation in similar dogs by the use of Procaine.

In 1950 Wedd and Blair (7) using the Turtle heart found that Procaine raised the threshold for stimulation and prolonged the conduction time in heart muscle.

Long, Oppenheimer, Webster and Durant (8) investigated the action of Procaine on the normal heart of dogs. They found E.C.G. changes suggesting depression of conduction with Procaine in small doses, while larger quantities produced Ventricular Fibrillation. Furthermore, they described its action on the Respiratory Centre.

The action on the peripheral circulation of Procaine (9) has been shown to cause vasodilation and thereby a fall in B/P.

In man the evidence is similar, there being two main actions. Firstly the Quinidine like action on the heart itself, depressing conductivity and decreasing irritability, and secondly the peripheral vasodilation which may cause a severe fall in Blood Pressure. This latter (10) can be avoided by the selection of the dose and not repeating it within twenty minutes.

II. *Hydrolysis of Procaine.*

Procaine hydrochloride is hydrolysed by Procaine Esterase into Para-amino benzoic acid and DiEthylamino Ethanol very rapidly after injection.

P.A.B. was found by Burstein to be of use prophylactically in dogs with induced ventricular fibrillation, but not therapeutically. The same worker showed that D.E.A.E. which is an adrenergic blocking agent, produced similar results as did procaine. Its prophylactic use was confirmed by Kraatz, Gruber and List. (11)

D.E.A.E. however, is fairly rapidly transformed and the resultant hypotension following its use, precludes it. (12)

III. *Clinical Evidence*

In 1946 Burstein (13) described the use of Intravenous Procaine in decreasing the cardiac irritability and conduction in the presence of Cardiac Dysfunction associated with Anaesthetic and Surgical procedures. The same year Barbour described his experiences with the prophylactic use of .1% Procaine for thoracic battle casualties.

Barbour and Tovell (14) in 1948 reported beneficial results using .1% Procaine before and during general surgical procedures when rapid and irregular cardiac rates were observed and also in those procedures involving Intrathoracic structures.

In 1949 Burstein (15) stated regarding the position of Procaine, that its efficacy in the treatment of certain cardiac irregularities during general anaesthesia was established and although he cited many satisfactory cases, he did

state that Procaine was not a cure-all and that it does have its limitations. (His views were not universally accepted.)

His views were that cardiac irregularity is due to sensitization of the Cardiac conducting mechanism which gives rise to increased cardiac irritability. He felt that I.V. Procaine tended to minimize this sensitization. He advocated 100 mgms. to be used in 1% solution as a prophylactic or Therapeutic dose, which could be repeated after an interval if necessary. Burstein also conformed to the opinion that Electrographic evidence should be available in all likely cases in order correctly to diagnose the arrhythmias.

Burstein, Piazza, Knapp and Rovenstine (16) in another paper the same year recount various reflexes during thoracotomy which give rise to cardiocirculatory disturbances. The treatment recommended was 100 mgm. Procaine I.V. combined with local application of procaine to the pericardium before the manipulation commenced.

Burstein has also made observations concerning vagovagal reflexes during intubation and extubation; Procaine D.E.A.E. and Procaine Amide reducing the incidence of arrhythmia caused by such. (Anoxia)

IV. *Types of abnormal rhythm.*

Various types of arrhythmia may arise during Heart Surgery. McQuiston (17) has divided the causation of these into those initiated by handling the Heart, those due to asphyxia and those secondary to Heart Strain. To this list might be added those caused by anaesthetic drugs, (Anoxia and CO_2 retention during anaesthesia.)

The first type can be minimized by local action of Procaine and by gentle handling, the second by general principles of anaesthesia and by Intravenous procaine, while the frequency of the third must be guarded against by avoiding additional strain on an already overtaxed organ.

Evidence has been presented to show that the incidence of arrhythmia caused by the anaesthetic per se may in some measure be lessened by the judicious use of I.V. Procaine prophylactically, while therapeutically there is also evidence that the drug may be of benefit.

Harken and Norman (18) have divided arrhythmias into benign and malignant types, the former the "Prefibrillation States" being shifted pacemaker, extrasystoles and (ventricular tachycardia) the latter being Ventricular fibrillation and Cardiac Standstill. They stress the importance of preoperative control, diagnosis during operation and identification of the cause. They concurred with the opinion that local and systematic administration of Procaine was of great value, from both prophylactic and therapeutic aspects. Concerning dosage of Procaine in the benign type, they suggested 200–300 mgms. which could be repeated up to three times in two hours. Therapy for the malignant type was also discussed but it is not relevant to this summary, except to state that they have found intracardiac Procaine had been used successfully in conjunction with Cardiac Massage.

V. *Procaine Amide.*

Owing to the rapid rate of Hydrolysis of Procaine and its Central Nervous System stimulation effect, the breakdown products have been assayed in respect to their cardiac action. Mention has been made of B.A.B. and D.E.A.E. In addition the Amide has also been used and reported on. (19, 20, 21, 22).

It has been shown that it has a much more prolonged action and that it can be used by oral administration. It possesses a quinidine like action in a manner similar to Procaine. Cardiac irritability is reduced, the refractory period is prolonged, there is slowing of the heart and an increased cardiac filling. It does not, however, possess the toxic action of quinidine nor the local anaesthetic action, rapid hydrolysis nor central nervous system stimulating effect of Procaine. It resembles D.E.A.E.

Experimentally, the majority of reports suggest it is capable of protecting the heart against Ventricular Tachycardia induced by adrenalin in dogs but Morris and Haid (23) were unable to reproduce this effect.

In spite of its desirable properties, the question of dosage gives rise to difficulty. That which will control an arrhythmia in one case may be either ineffective or excessive in another. Concerning the intravenous administration, not only is the dosage of importance but also the rate at which it is given.

As with Procaine and D.E.A.E., Procaine Amide appears more active in the treatment of arrhythmias of Ventricular origin; such are the more malignant and are those in which endeavour must be made to treat.

Summary

1. The evidence analysed demonstrates that Procaine applied locally to the heart decreases irritability.

2. The majority of evidence shows that I.V. Procaine in requisite dosage diminishes irritability of the heart muscle and depresses conduction (a) Its prophylactic use diminishes the frequency of arrhythmias, especially those of Ventricular origin. (b) Therapeutic use in the benign types of arrhythmia will usually result in restitution of normal rhythm. (c) In the malignant types of arrhythmia, exceptionally it can promote normal rhythm.

3. Procaine is rapidly hydrolysed. D.E.A.E. presents the same action on the heart and is longer acting, but it may produce hypotension.

4. Procaine amide is longer acting and has neither local anaesthetic nor central stimulating action. It may be given by mouth or intravenously and has similar action on the cardiovascular system to Procaine. However, the assessment of dosage is difficult.

Conclusions

Procaine given locally and intravenously is of use in Cardiac Surgery, as may also be the Amide. But the inclusion of this type of drug as an additional arrow in our quiver must not prevent the practice of the basic principles of

anaesthesia as oxygenation; assistance to respiration and maintenance of Blood Flow during Intrathoracic Operations.

REFERENCES

1. Levy, A. G. J. Physiol. 43:3, 1911.
2. Meek, W. J., Hathaway, H. R. and Orth, O. S. J. Pharmacol & Expt. Therap. 61:240, 1939.
3. Kurtz, C. M., Bennett, J. H. and Shapiro, H. H. J. Am. Med. Assn. 106, 434, 1936.
4. Mautz, F. R. J. Thoracic Surgery 5:612, 1936.
5. Burstein, C. L., Marangoni, B. A., Degraff, A. C., and Rovenstine, E. A. Anaesthesiology 1:167, 1940.
6. Stutzman, J. W., Allen, C. R. and Orth, O. S. Anaesthesiology 6:57, 1945.
7. Wedd, A. M. and Blair, H. A. Anaesthesiology 12:261, 1951.
8. Long, J. H., Oppenheimer, M. J., Webster, M. R. and Durant, T. M. Anaesthesiology 10:406, 1949.
9. Beutner, R. Anaesth. and Analg. 27:197, 1948.
10. Taylor, I. B., Stearns, A. B., Kurtz, H. C., Henderson, J. C., Sigler, L. E., and Nolte, E. C. Anaesthesiology 11:185, 1950.
11. Kraatz, C. P., Gruber, C. M., List, A. G. Jr. Phar. & Exp. Therap. 98:111, 1950.
12. Rosenberg, B., Kayden, H. J., Lief, P. A., Mark, L. C., Steele, J. M. and Brodie, B. B. J. Pharm. & Expt. Therap. 95:18, 1949.
13. Burstein, C. L. Anaesthesiology, 7:113, 1946.
14. Barbour, C. M. and Tovell, R. M. Anaesthesiology 9:514, 1948.
15. Burstein, C. L. Anaesthesiology 10:133, 1949.
16. Burstein, C. L., Piazza, T. L., Kapp, L. A. and Rovenstine, E. A. Surgery 25:36, 1949.
17. McQuiston, W. O. Arch. of Surgery 61:892, 1950.
18. Harken, D. E. and Norman, L. R. Anaesthesiology 11:321, 1950.
19. Peters, N. S., Newman, W., Burstein, C. I. Anaesthesiology 12:673, 1951.
20. Sadove, M. S., Wyant, G. M., Kretchmer, H. E. and Gittleson, L. A. Anaesthesia & Analgesia 31:45, 1952.
21. Miller, H. Nathanson, M. H., and Griffith, G. C. J.A.M.A., 146:1004, 1951.
22. Joseph, S. I., Helrich, M., Kayden, H. J., Orkin, L. R., Rovenstine, E. A. Surg. Gyn. and Obs. 93:75, 1951.
23. Morris, L. E. and Haid, B. Anaesthesiology 12:328. 1951.

(5) POST-OPERATIVE COMPLICATIONS IN CARDIAC SURGERY

Dr. Nelson Nix

These have been divided by McQuiston into three groups:

(*a*) *Complications probably due to anaesthesia:*

Cough
Cough with mucus
Cough with mucus and blood
Laryngeal edema
Pulmonary edema

(*b*) *Complications with anaesthesia a possible factor:*

Cerebral anoxia (followed by coma for hours or days).
Cerebral anoxia (followed by temporary spasticity).
Extrasystoles
Pulmonary rales

Pneumonia
Atelectasis
Bronchitis with effusion

(*c*) *Complications probably unrelated to anaesthesia:*
Thrombophlebitis
Subcutaneous emphysema
Pneumothorax
Hemothorax
Pulmonary edema (transfusion reaction)
Anuria (transfusion reaction)
Wound infection

McQuiston strongly advises the steam room, with temperature not over 75° for laryngeal edema therapy, warning that just a steam kettle is not enough. For pulmonary edema the trachea is aspirated and oxygen under pressure advised. Closed drainage of the chest with negative pressure largely prevents atelectasis postoperatively.

ELECTROCARDIOGRAPHIC CHANGES DURING EXTUBATION:

Arrhythmias are more common in thoracic surgery and on biliary tract. It is advantageous to have patient as reactive as possible at conclusion of surgery; but if a very light anaesthesia at time of extubation promotes serious cardiac disturbances, then perhaps it is better to have patient react more slowly.

There are fewer disturbances (as shown by E.C.G.) when pentothal is used with gas anaesthetic.

There are more disturbances when ether is used alone or when cyclo used alone.

Intravenous procaine does not prevent all arrhythmias, nor does topical pontocaine applied to larynx. *The practice of ventilating the patient's lungs with oxygen 100% before extubation seemed to offer the most protection against arrhythmias at this time.

THE POSSIBLE USE OF E.C.G. POST-OPERATIVELY:

The major problem is recognition or diagnosis. A few dramatic crises occur which might be prevented if E.C.G. was used during and just after a surgical procedure, or when a change for worse appears.

1. Death from *S–A block,* due to excessive *vagal tone*—E.C.G. *might have revealed an A–V block,* a warning. One may have treated this with *atropine* or *ephedrine early.*

2. Death from *ventricular fibrillation.*—May have been treated with procaine amide, quinidine, or electrical stimulation (defibrillation).

3. *Slow Pulse*—Is it sinus bradycardia (*use atropine*) or is it *coupling* due to ventricular premature beats? (use quinidine or procaine amide).

4. *Cardiac Damage* which *cannot be detected clinically.*
First degree heart block

Right or left bundle branch block
Intraventricular block

It is essential to recognize these, since certain drugs (digitalis, quinidine, pronestyl) *increase* the conduction defect and caution is required.

I.V. procaine rarely effective in the *un*anaesthetized patient.

I.V. Procaine amide may stop *auricular fibrillation* during anaesthesia but not afterwards. Most cardiac reflexes are altered with various degrees of anaesthesia, types of anaesthesia and differ from those in the conscious patient. For this reason therapy differs with the stage and condition of the patient.

THE GENERAL RULES:

1. *Congestive Heart Failure*:
   (*a*) Shortness of breath
   (*b*) Râles in chest
   (*c*) Cardiac enlargement (shown by x-ray or clinically) often a failing heart.
   (*d*) Ventricular strain
   Suggested therapy is 1.2 mgm. digitoxin (12 hours pre-operatively).
2. *Asthenic Individuals*:
   (*a*) Older age group
   (*b*) Those with tuberculosis
   (*c*) Bed-ridden (several months or years).
   Again, digitalization recommended.
3. *Multiple Premature Ventricular Contractions*; (As shown by E.C.G.)
   Pronestyl (procaine amide) is useful.
4. *Vaso-Vagal Reflex*:
   This complication is less frequent in the digitalized, anaesthetized group.
   Prophylaxis with Banthine is recommended by Fisher and Travis.
5. *Coronary Insufficiency*: (Shown by E.C.G. or by angina pectoris)
   Aminophyline orally or suppository
   Gr. 1½ q4h x 24 hours
   Gr. iii one hour pre-operatively (oral) or gr. $\overline{viiss}$ by rectum
6. *Supraventricular Arrhythmias*:
   (*a*) Auricular fibrillation
   (*b*) Auricular tachycardia
   (*c*) Ectopic beats
   (*d*) Nodal tachycardia
   Rx Pre-op: Quinidine gr. vi
   During operation: pronestyl!! (In spite of general opinion)
   Post-op: quinidine.
7. *Ventricular Arrhythmias*:
   (*a*) Ectopic beats
   (*b*) Tachycardia

(*c*) Fibrillation

Treatment same as for auricular types.

8. *Digitalis Intoxication*:

(*a*) S-A block — From overdigitalization

(*b*) First or second degree heart block

May be treated by as little as 5 mgm. Banthine I.V.

THE ROLE OF PHYSIOTHERAPY IN POST-OPERATIVE CARE:

From the beginning of the post-operative period, the patient must be encouraged to keep his tracheobronchial tree clean:

Regular coughing
Breathing exercises
Posture if possible

Rigid supervision is usually necessary by a trained physiotherapist if possible. Continue this into convalescent and rehabilitation period to lessen the mortality and morbidity of the thoracotomy.

REFERENCES

McQUISTON. Anaesthesiology, Sept. 1949.
CONVERSE, *et al.* Anaesthesiology, March, 1952.
FISHER and WINSOR. Anaesthesiology, March, 1952.
FISHER and TRAVIS. Anaesthesiology, March, 1952.
DAFOE, C. S. Alberta Medical Quarterly, July, 1951.

(6) QUESTIONS FROM THE FLOOR

1. Will you tell us how you control fluid administration in small children undergoing operations on the heart?

Answer—Dr. Junkin.

It is known that supportive treatment will be required and therefore a cut down is done immediately before operation and both suitable blood and plasma are available. If the hemoconcentration is high, plasma is run in during operation at a sufficient volume to maintain blood pressure levels at or near normal. If the hemoconcentration is below normal, whole blood is used for replacement at a rate sufficient to maintain an approximately constant volume. The rate of flow will be judged by observation of blood loss and also by pulse and blood pressure readings. On rare occasions two cut downs may be necessary to keep up to blood loss, the second one being started during the operation, if it becomes necessary.

At the end of operation with the necessary blood replacement complete, the intravenous is continued with Dextrose in saline. Fluids are given by mouth as soon as they can be tolerated and the intravenous drip is discontinued when the intake by mouth becomes sufficient for the patient's needs.

2. Will you describe in some greater detail your anaesthetic techniques in these patients?

Answer—Dr. Junkin.

In small patients ether, oxygen plus pentothal and syncurine are used. In older children cyclopropane, pentothal, and syncurine are used. High oxygen concentrations are maintained during the induction stage. Pentothal and syncurine are used and inhalations of oxygen plus ether or cyclopropane are then given up to the point where intubation can be accomplished without difficulty. On the establishment of an endotracheal system, as soon as possible, the patient is carried to a stage of controlled respiration, thereby ensuring an adequate ventilation with a minimum of movement of lungs and mediastinum. Topical application of procaine solution, by the surgeon after he has entered the chest, to block vagal reflexes is desirable in many cases and is in my opinion more effective than including procaine in the intravenous administration.

Syringes loaded with atropine, coramine, epinephrine and procaine are kept available for immediate use.

Desirable adjuncts would be an apparatus for defibrillating a Ventricular Fibrillation and a pace maker for starting a heart that is in a state of arrest–but these we do not have at our disposal at the present time.

3. Do you use refrigeration in these patients?

Answer–Dr. Junkin.

I would prefer to use the term controlled temperatures. We employ a thermocouple type of rectal thermometer from which readings may be taken at any time. At the beginning, we used a crude form of air conditioning, with buckets of ice with a fan blowing over their surface, to lower the temperature of the room, when operating on very hot days. At the moment we are investigating the possibilities of a water mattress for directly cooling or heating a patient and maintaining a desirable level of body temperature. In the cardiac cases of course it is desirable to suppress metabolism by lowering the temperature a degree or so below normal during the operative period. Temperatures above normal definitely are detrimental to the success of the operation.

HEXAMETHONIUM INDUCED HYPOTENSION TO REDUCE SURGICAL HEMORRHAGE

KATHLEEN W. LANGSTON, M.D., D.A.(Eng.)*

The problem of hemorrhage during surgery always has been of deep concern to surgeons and anaesthetists. Since the realization, during and after World War II, of the importance of adequate blood replacement, it has become even more important. Hemorrhage during surgery has two consequences. Firstly, when it is so profuse as to endanger the safety of the patient. Secondly, when small in itself, it can interfere with the successful performance of the surgery by obscuring the field of operation, and prolong the operative time by that required to control the hemorrhage.

Hypotension, to a level where only arteries bleed, will control both types of dangerous or annoying hemorrhage. This is achieved by providing a systolic pressure of 55 to 65 mmg. of Hg.

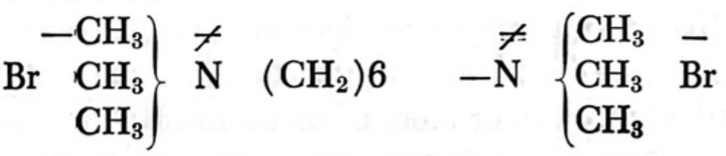
Since 1948, Gillies of Edinburgh (1) has been using total spinal anaesthesia to produce this degree of hypotension for the control of surgical hemorrhage. This necessitates spinal block to T.1 or about level of the clavicle.

Another method of hypotension reported by several writers (2) (3) is by arterial bleeding until the blood pressure falls to 60 to 80 mm. of Hg. The cannula is kept in the artery and the blood replaced by arterial transfusion at the end of operation, or before, if the systolic pressure falls below 60 mm. of Hg.

A third method is by epidural anaesthesia, which acts in the same way as spinal block (4). These methods of producing hypotension are all effective but so inherently dangerous that they have not been widely used.

With the introduction of the methonium compounds by Paton and Zaimus in 1949 as autonomic ganglionic blocking agents, a new field of investigation was opened. In the literature which immediately began to appear on these drugs for the treatment of hypertension and control of gastric secretion, postural hypotension was presented as an annoying side effect (13–17). Enderby at East Grinstead (5) saw the possibility of adapting this to surgery and producing a safer hypotension than methods already in use. In June, 1950, he reported his first 50 cases using Pentamethonium iodide and in March, 1951, a further 250 cases with Hexamethonium bromide. He considers the latter a more effective drug. Reporting on the use of both drugs, Shackleton (7) in May, 1951, with 250 cases, and Lewis in July, 1950, with 80 cases of thoracic surgery, agree that Hexamethonium bromide is most constant in reducing blood pressure. Well over 1,000 cases have been recorded in the British literature.

The drug used in this series was "Vegolysen" brand of Hexamethonium bromide supplied by Poulenc Ltd. It is one of a series of Polymethylene Bistrimethylammonium salts. The formula is:—



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The pharmacological properties are related to the number of carbon atoms in the polymethylene chain. C_{10} or decamethonium has a curarizing action blocking the neuromuscular junction. C_5 and C_6; Pentamethonium and Hexamethonium show a sharp maximum potency in ganglionic block. The site of action is at the pre-ganglionic synapse of both sympathetic and parasympathetic nerves. There is, therefore, a block of the entire autonomic system. The effects are; mainly, generalized vasodilatation with postural hypotension. Given intravenously, in effective dosage, to an erect patient, the blood pressure would probably fall to zero. By placing him in a horizontal or slightly head-down tilt, it would as quickly return to 80 or 90 mm. of Hg. or higher. Other effects are dry mouth, blurring of vision from dilated pupils, occasional drowsiness and decreased motility of gut and bladder. 60–70% of the drug is excreted in the urine unchanged (11). No chronic toxicity has been observed in laboratory animals even after prolonged administration of relatively large amounts of Hexamethonium.

Hexamethonium acts by blocking the sympathetic supply to the vasoconstrictor mechanism. This makes it impossible for the body to compensate for blood loss either by hemorrhage or into its own capillary reservoir. "The vascular system in essence is composed of a pump and arterial conduit leading to a capillary reservoir and a return or venous conduit. The capillary reservoir which has a huge capacity in comparison to the total blood volume is capable of considerable variation in volume. If the reservoir were to enlarge to its full capacity, blood would be lost from the large conduits, whereas if it became smaller, an excessive amount of blood would be forced into the central circulation. If this closed system were static, blood loss would produce a fall in pressure throughout and a diminution of venous return. However, since the normal vascular system is dynamic, the capillary reservoir is capable within certain limits of reducing its capacity in proportion to the degree of blood loss. Thus the venous conduit remains filled and venous pressure and return are maintained. On this depends the extremely sensitive homeostatic mechanism of the vascular system.

"Certain hypotensive agents by doing away with the ability to reduce the volume of the capillary beds, convert the vasculature into a more static system, with the result that during blood loss the pressure falls throughout and venous return fails." (25)

When a patient is given an adequate dose of Hexamethonium the blood pressure will fall, but not alarmingly if he is kept horizontal or slightly head-down. However, if the feet are lowered or the whole body placed in a foot-down slope, the blood pressure can be lowered almost proportionately to the degree of tilt. In effect the patient is bleeding into his own capillary reservoir where the blood may be safely kept until the end of the operation. It is then returned to his circulation by the simple expedient of raising the foot of the table and if necessary, using a slight head-down position. This may also be done at any time during the operation, so that a relatively complete control of blood pressure can be maintained.

In the normal patient, 500 c.c. or more of blood must be lost before any adverse

effect is seen upon the blood pressure. However, using Hexamethonium, should any degree of external hemorrhage occur the body cannot compensate and the blood pressure falls further. Even a loss of 100–200 c.c. will show a decrement in blood pressure.

It cannot be emphasized too strongly that an essential part of this technique is the maintenance of constant blood volume. The only patients whose blood pressure did not rise promptly on changing position to a 5° or 10° Trendelenburg, were those whose blood replacement did not keep pace with the blood loss.

Consequently, therefore, it is apparent that the site of operation is important. Ideally the surgical site should be the highest point in the body, while allowing for the legs, particularly, to be dependent. In this way the blood drains out of the operative area into the dilated vessels of the more dependent parts of the body. In addition to hypotension, there is also what Enderby calls "a postural ischaemia of the surgical site."

The lateral jack-knife position of thoracic surgery is a typical example. This position can be exaggerated by raising the kidney bar and having the legs at a steep angle. In head and neck surgery, the whole body can be placed in any desired degree of foot-down slope. Many other sites can be used by some thought and ingenuity in posturing.

The cases on this series have been:—

Pneumonectomies—5.
Lobectomies—9.
Decortication of a lung—1.
Thoracoplasties—27.
Lumbodorsal sympathectomies—2.
Craniotomies—3.
Plastic surgery—4.
Spine fusion—2.

TOTAL: 53.

Type of individual.

Patients with hypertension react much more readily to Hexamethonium and with a more profound hypotension than the normal group. Old age groups over 70 and known arteriosclerotics have so far been avoided. As with many drugs, the young, healthy adult requires a maximum dose and the elderly asthenic type a minimum. When in any doubt about the patient's reaction to the drug it can be given in small divided doses to obtain the desired effect. The minimum total dose has been 12.5 mg. and the maximum 150 mg. The initial dose used in the average vigorous, young adult is 50 mg.

Anaesthesia has been produced with a combination of Pentothal, Curare, intravenous Demerol, Nitrous oxide and Oxygen. Induction is with 0.5 gm. of Pentothal and 15 mg. of Curare. An endotracheal tube is then passed, and anaesthesia maintained with Nitrous oxide and Oxygen usually four litres to two. If there is any question of the patient's oxygenation, the nitrous oxide is decreased or even

discontinued. Many of the thoracic surgery patients have been carried on pure oxygen. The provision of adequate oxygenation is a fundamental principle in anaesthesia, even more essential when the blood pressure is low, for in this condition oxygen deficiency will strike with dreadful accuracy (26). Pentothal and Demerol are used intravenously either in a continuous drip or in intermittent doses. Our most recent method is to use 25 mg. of Demerol and 2 c.c. of 2.5% Pentothal, alternating at about 20 minutes interval for the first two hours. After that the intervals are lengthened to one-half hour or more. In this way an operation can be carried through with very small amounts of anaesthetic agents. The average total for lobectomies and pneumonectomies was 1.2 gms. of Pentothal and 100 mg. Demerol. Thoracoplasties used an average total of 0.96 gm. Pentothal and 50 mg. Demerol per case.

After being intubated, the patient is given an initial dose of Hexamethonium and postured. A moderate foot-down tilt is tried and the degree of hypotension assessed in two or three minutes. An attempt is made to produce a systolic blood pressure of 55 to 65 mm. of Hg. This would seem to be the best range for maximum control of hemorrhage with reasonable safety to the patient. Once stabilized, the blood pressure tends to remain at that level for one or more hours. Should it begin to rise a prolongation of effect can sometimes be had by increasing the degree of foot-down tilt. If this is not sufficient, a further dose of Hexamethonium may be given. All these patients have been carried on controlled respiration which also assists in keeping the blood pressure low (23).

Placing the table in a 5° or 10° Trendelenburg position at the end of the operation is usually sufficient to bring the blood pressure back to 90 or 100 mm. of Hg. We consider this a safe level for the patient to leave the operating room. However, the blood pressure is very labile for three to four hours and can be readily altered by raising or lowering the foot of the bed. The patient's condition must be closely observed during this postoperative period.

Complications in the operating room have been:

I. One case of auricular fibrillation. This was reversed in about 15 minutes with 5 c.c. 1% Procaine intravenously and 0.2% Procaine intravenous drip for the remainder of the operation. The concentration of oxygen was increased by decreasing N_2O and the blood pressure raised to 80 mm. Hg. by decreasing the tilt down of the legs. The patient was a malignant hypertensive having his second lumbodorsal sympathectomy under hypotension. The first had been uneventful. This was the only complication relating to the heart.

II. A severe bronchospasm in a known asthmatic undergoing pneumonectomy. This responded fairly well to intravenous aminophylline.

III. At the beginning of the series there were a few cases in which the blood pressure was slow to rise after the table had been put in 10° Trendelenburg. In the first case 6 mg. of Methedrine was given intravenously without effect. At that point the estimation on blood loss was completed and it was evident that blood replacement had been inadequate. As soon as the blood loss was replaced the blood pressure came up promptly. Since that time a blood

pressure that does not return to a reasonable level with horizontal or head-down position has been considered evidence of inadequate blood replacement. Again, this point cannot be too strongly stressed. It is of paramount importance to the technique to replace blood loss! The patient's vasomotor control having been abolished, the body very quickly reacts adversely to the loss of even small amounts of blood.

IV. Inability to reduce the blood pressure sufficiently after giving Hexamethonium and consequent danger of increased bleeding due to vasodilatation. The effective dosage has proven so variable that it may be simply a case of giving the drug until the desired effect is obtained. Some investigators claim that if an effect is not reached with 100 or 150 mg. it is useless to give further drug. It may be due to insufficiently extreme posture. In one case the blood pressure had not fallen below 90 mm. of Hg. in spite of 100 mg. of Hexamethonium. By placing the patient in an almost upright position for a minute or two the blood pressure fell to a satisfactory level and remained. Since that time this practice has been followed in the resistant cases with better results.

V. The greatest technical difficulty has been accurate recording of blood pressure at low levels. The auscultatory method sometimes fails because of weakness of the sound. Oscillometers have been tried but there is too much interference with movement about the table. We are now working on a direct method of recording intra-arterial pressure (25).

Postoperative complications have been pleasingly few. Most of the patients have returned to consciousness as soon as or before similar types of operations with other anaesthetic techniques.

Two patients had delayed recovery not becoming fully conscious until seven hours postoperative. One was a man of 67 with advanced bronchogenic carcinoma, who had a pneumonectomy. It was a difficult case and in spite of a hypotension of 60 to 70 mm. of Hg. for the four hours of the operation, he lost 1350 c.c. of blood. This was our single largest blood loss with Hexamethonium. His anaesthetic consisted of Pentothal 1.58 gms., Demerol 50 mgs. and Curare 30 mgs. The only gas used was oxygen because of concern about his oxygenation. The other was a case of pulmonary tuberculosis with a long history of severe illness. A pneumonectomy was done as a last resort because of a persistent focus of infection. This patient had a severe hemorrhage immediately postoperative, losing 800 c.c. of blood into the drainage bottle in 3 hours. Neither of these patients showed any headache or mental confusion on recovering consciousness.

Three patients in the control and four in the Hexamethonium series had postoperative hemorrhage with a considerable fall in Hb. (Fig. I). An attempt has been made to analyze the pre- and post-operative hemoglobin records in both groups, to assess if possible the relationship of post-operative bleeding. Up to a 14% fall in hemoglobin occurred in 45.9% of the control and 67% of the Hexamethonium. A fall of 15–29% in hemoglobin occurred in 43% of the control and 19% of the Hexamethonium group. Falls of 30–44% hemoglobin occurred in 10.8% of the control and 13.5% of the Hexamethonium group. There seems to be essentially

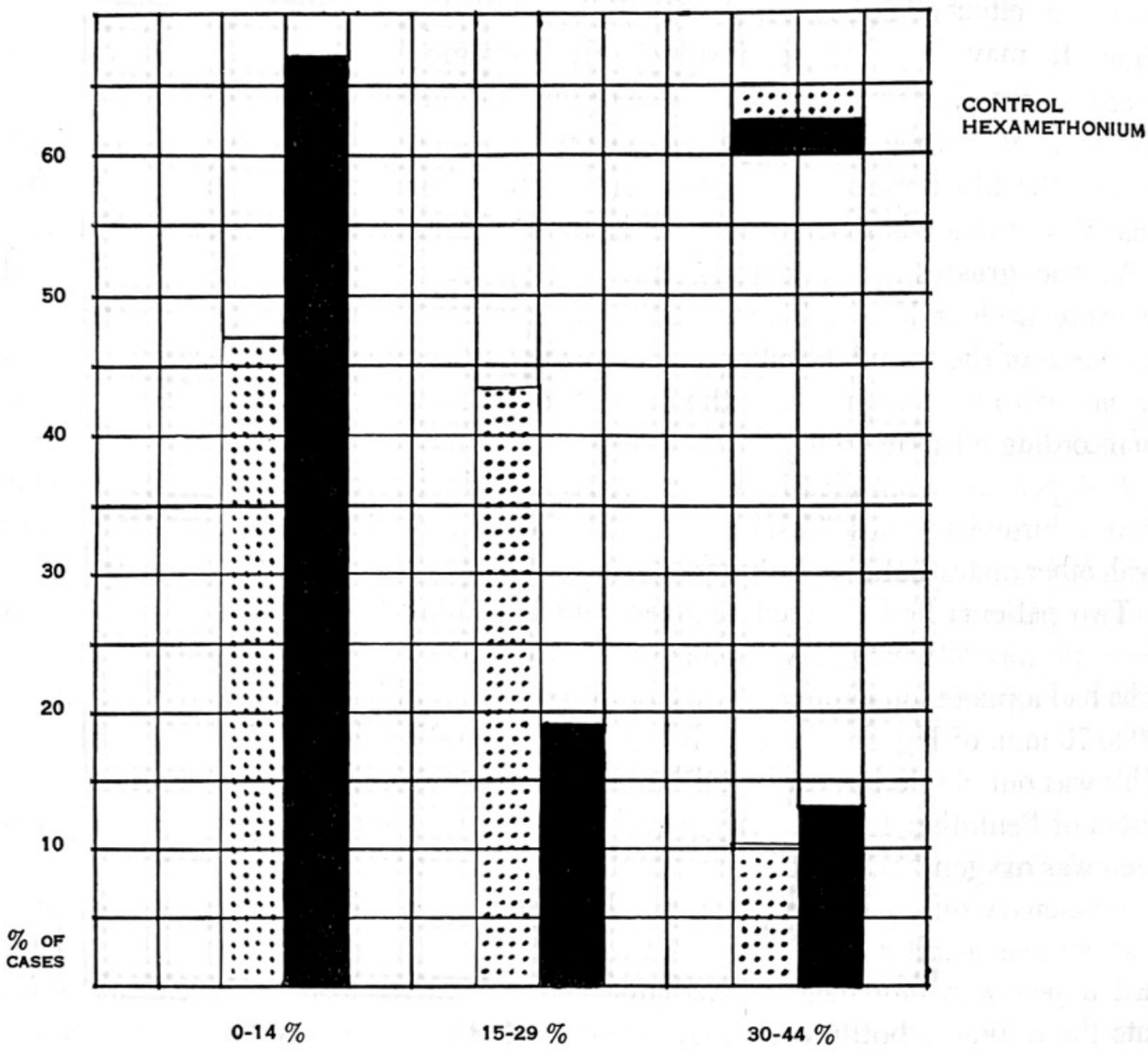
little difference in the two groups. Reports by other writers claim reactionary hemorrhage as being rare.

One patient complained of severe low back pain after both his operations. This was probably due to his position on the table.

Two patients complained of headache the first postoperative day.

Aside from this, the clinical impression is that these patients have felt particularly well postoperatively.

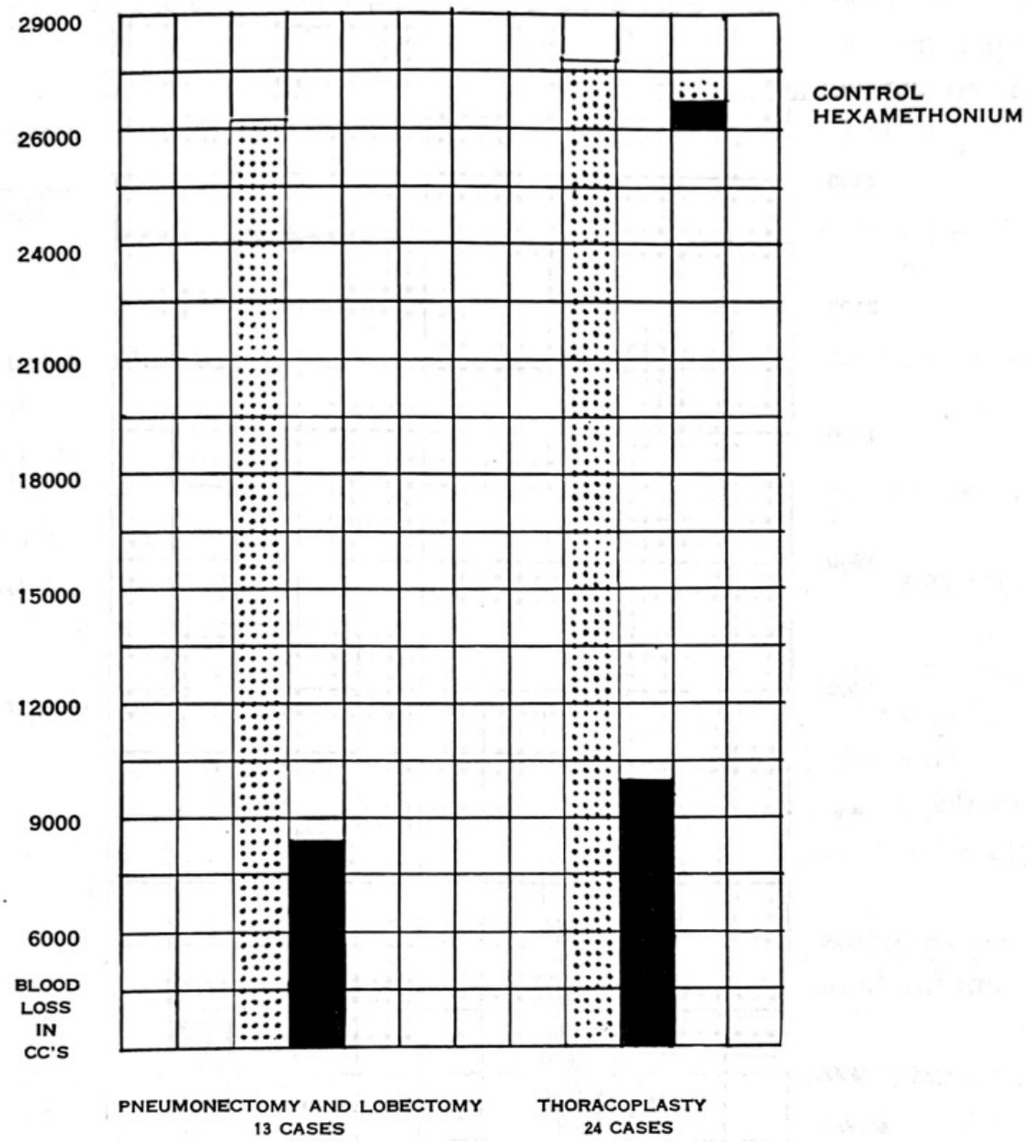
THORACIC SURGERY



POST-OPERATIVE DROP IN HB.

Assessment of results has been made easier because for the past two years in the Chest Unit at Shaughnessy Hospital blood loss in the operating room has been estimated by the gravimetric method. All sponges, towels, gowns and other linen is weighed before sterilizing and the weight in grams marked on each piece. They are then used dry and weighed after use, each extra gram of weight being considered as 1 c.c. of blood loss. Sponges are weighed as the case progresses.

Fig. II shows the combined total amount of blood lost in 13 cases of lobectomy and pneumonectomy. In the control it is 26,225 c.c. and in the Hexamethonium group, 8,455 c.c. In 24 cases of thoracoplasty on the right, the control group lost a combined total of 27,946 c.c. and the Hexamethonium group 9,930 c.c.



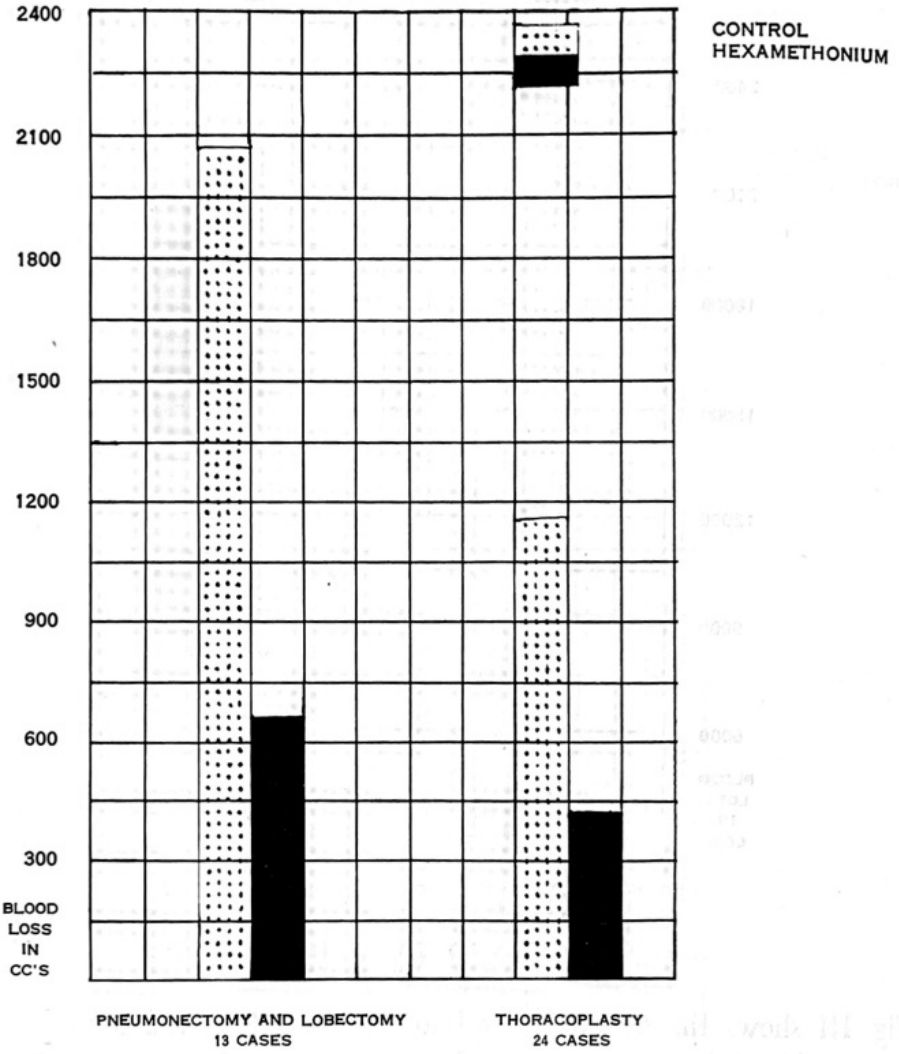
COMBINED TOTAL BLOOD LOSS

Fig. III shows the average blood loss per case. In the lobectomy group the average blood loss per case in the control was 2,017 c.c. and in the Hexamethonium group 650 c.c. In the 24 thoracoplasties, the average blood loss per case in the control was 1,164 c.c. and in the Hexamethonium group 413 c.c. This proves beyond doubt that it is possible to markedly reduce surgical hemorrhage by this method. But a great deal of work needs to be done before we can be assured of the safety of the method and adopt it wholeheartedly. The patients must be selected carefully and watched with the greatest detail during the operation and in the immediate postoperative period.

A less dramatic result has been the reduction of operative time. The average time in the control group for lobectomies was 3.5 hours, and with hexamethonium

2.6 hours. For thoracoplasties the average time was reduced from 1.8 hours to 1.5 hours. For three carniotomies the average time was 2.06 hours.

Postoperative urinalysis showed only an occasional trace of albumin and an occasional leucocyte or granular cast. The postoperative urinary output was not abnormal. By catheter placed in the bladder one hour preoperatively the amount of urine secreted in that time averaged 87 c.c., while the average secreted during the operative period of hypotension was 48 c.c. The urinalysis on the operative specimen showed an increase in specific gravity and occasional trace of albumin.



AVERAGE BLOOD LOSS PER CASE

All the thoracic surgery cases had preoperative and postoperative electrocardiograms, and ten cases had electrocardiograms during operation with hypotension. There was no evidence of coronary ischaemia or postoperative cardiac damage from anoxia.

In using a technique of this kind, which in so many ways is contrary to our accepted ideas on physiology, one would wish to have as much support as possible from current medical literature. In reviewing the available publications I was struck by how little work has been done on hypotension.

Gillies of Edinburgh claims that a systolic pressure of 60 mm. of Hg. will maintain a capillary circulation sufficient for cellular respiration and metabolism in all the vital organs provided the blood is well oxygenated and provided vasodilatation is assured.

Henry, Gauer and Kety writing on "Factors Maintaining Cerebral Circulation During Gravitational Stress" show that in various types of postural hypotension in the conscious patient the mean arterial pressure can fall to 30 mm. of Hg. before consciousness is seriously impaired, but that syncope develops at 20–25 mm. Hg. Mean arterial pressure was obtained by adding one-half pulse pressure to the diastolic pressure.

Kety and King (19) reporting on high selective spinal block in hypertensive patients, show that with a severe reduction in blood pressure, there is a significant reduction in cerebral blood flow and evidence of cerebral anoxia.

Hafkenschiel and Crumpton used dihydroergocornine as a sympatholytic drug in both hypertensive (20) and normotensive (21) patients. They record that in spite of a significant decrease in mean arterial pressure, the cerebral blood flow, cerebral arterio-venous oxygen difference and the cerebral oxygen uptake were essentially unchanged.

This would agree with Gillies statement that it is the vasodilator state that is important to the safety of the patient.

In the kidney hypotension has usually been associated with vasoconstriction as in shock from hemorrhage or trauma. In fact a renal vasoconstrictor substance has been demonstrated in shock. There are reports of relief of the renal anoxic syndrome by splanchnic block or spinal anaesthesia (22). This is corroboration of the vasoconstrictor factor in renal ischaemia and would support the theory of the safety in vasodilatation.

Phemister in 1944 demonstrated that animals would tolerate prolonged periods of hypotension provided there was no associated reduction of blood volume and anoxia (27).

The deleterious effect of prolonged vasoconstriction was re-emphasized by Wiggins and his group in 1947. They administered dibenamine, a sympatholytic drug to dogs subjected to hemorrhagic shock. This prevented the onset of irreversible shock and 80% of the treated animals survived compared with 30% of the untreated (28).

A severe fall in blood pressure can initiate an anginal attack in coronary heart disease and this is a definite contra-indication for hypotension. Dr. Douglas Robertson (26) has done electrocardiographic studies in normal and hypotensive states. In the small series which he has so far investigated, there has been no evidence of coronary ischaemia in either young or old patients when the pressure has been between 60–70 mm. Hg.

SUMMARY:

A report has been made on 53 cases where Hexamethonium bromide has been used to produce hypotension during anaesthesia.

ANAESTHESIA FOR HARELIP AND CLEFT PALATE REPAIR

C. I. JUNKIN, M.B.*

The available records at the Hospital for Sick Children show that since 1924 some 1,250 harelips have been repaired and approximately 1,300 cleft palates have received surgical treatment. Of these cases, four harelips and two cleft palates died during operation—a total of six operative fatalities in 2,250 cases, which is a mortality rate a little more than twice the operative mortality rate for all surgical cases operated upon. My own impression is that the number of deaths over this period was even higher than that revealed by our records, since all the recorded deaths occurred since 1935—none being recorded for the eleven years prior to that date. I am sure that while our records are accurate as to the number of cases, they are not very dependable and err on the side of leniency with regard to operative fatalities. I am also sure that these cases are among the most difficult with which one has to deal in Paediatric Anaesthesia. There has always been a high operative mortality rate which has steadily declined as our methods have improved.

Among the causes of high operative mortality rate one might enumerate:

(1) The presence of other congenital defects, such as congenital cardiac and great vessel anomalies, in these cases;

(2) The technical difficulties to be overcome in giving an anaesthetic when the lips and palate are the site of operation;

(3) The fact that the areas operated upon are extremely vascular, and the blood loss can be relatively severe;

(4) And finally, that, at least in our experience, the operation takes from two to three hours.

An account of the evolution of our methods may be of interest. On my introduction to these cases, the anaesthetic was administered by way of a mouth hook, and consisted of ether and air—the source of the air being a double hand bulb which one kept going as vigorously as possible throughout the operation, the other hand being occupied with the almost impossible task of coping with haemorrhage with a mouth sucker, in an effort to keep the patient from drowning in his own blood. A three-bottle Jaunker apparatus was used with chloroform in one bottle, ether in another, the third bottle being used as a trap. When the patient, in spite of one's vigorous squeezing on the bulb, showed signs of returning consciousness—one switched temporarily from ether to chloroform, and the patient was quickly subdued by the more potent drug. At this stage in our evolution, transfusions were resorted to only in dire extremities. These were direct transfusions, usually from parent to child. It must have been almost as much of a trial to the parent as it was to the anxious anaesthetist who hoped that the transfusion would be in time to turn the tide in his favour.

The first improvement to be recorded was the discovery that an air pump could be used as a source for vapourizing ether. Later we became bright enough

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to use an oxygen tank with a reducing valve for this purpose, the higher oxygen percentage being of some advantage to patients who had heretofore always been a bit anoxic, either from intermittent laryngeal spasm, or from partial flooding of the pharynx with blood, or from deliberate interference by the surgeon with the function of respiration.

The next forward step was the establishment of a blood bank in the hospital, and we were made somewhat more hopeful of the outcome by having a cut-down done at the beginning of the operation and transfusions were done as a prophylactic measure, rather than as a last resort to overcome haemorrhagic shock.

Next, with the advent of portex catheters, we became bold enough to intubate these small patients—harelips by the oral route and cleft palates by nasal intubation. We had come a long way toward overcoming the chief difficulties and risks of these cases. The anaesthetic was still ether oxygen and reasonably satisfactory, but with the drawback that the patient subjected to an ether anaesthetic for anywhere from two to three hours suffered some ill-effects from this intoxication and was slow in returning to consciousness and to the stage where feedings were tolerated.

Finally, we took the obvious step of resorting to intravenous pentothal in combination with endotracheal nitrous oxide-oxygen, since with an intravenous established there were no difficulties in introducing the barbiturate. At the outset, intubations were done with pentothal, curare, or syncurine anaesthetic combination, but after a few attempts it was decided that since intubation in these cases is not always as easy or as simple as one could wish, an ethyl chloride ether sequence up to the point of intubation was a much safer procedure. With these small patients it was not always easy to guage the dosage of barbiturate and relaxant drug accurately and a dosage sufficient to depress respiration put one in difficult dilemma if the intubation could not be accomplished with facility and speed.

Our present procedure, therefore, is as follows. A cut-down is done immediately before operation. This is to insure that the patient will not be overloaded with fluid, run in at an uncontrolled rate, or for too long a period. I have had two cases which developed acute pulmonary oedema from what I considered to be too much fluid run in too rapidly. Fortunately these cases were brought under control, the operation postponed for two or three days, and then accomplished without embarassment. Therefore, it is necessary to keep a watchful eye on the surgical interne doing the cut-down, and to make sure that he does not deal with an infant as he would with an adult and that he has a little regard for the amount of fluid he is introduoing into a circulation of limited capacity.

An ethyl chloride ether sequence is employed up to the point of intubation. When intubation has been accomplished, an immediate switch-over to pentothal and syncurine intravenously with nitrous oxide and oxygen by endotracheal catheter by inhalation is made. A solution of 2½% pentothal with 1 c.c. syncurine added to a 20 c.c. solution is used intravenously. A continuous flow of 2 litres

nitrous oxide and 1 litre oxygen per minute is usually found satisfactory as the analgesic agent in this combined light level anaesthetic. A very simple hook-up is used for the administration of nitrous oxide and oxygen. A length of rubber tubing runs from the gas machine to a No. 16 Foregger bag. From this a short length of tubing, about 10 inches long, runs to the connection with the catheter. This tube has a linear slit about 1¼ inches long, about 1 inch from its distal end, which acts as a very simple and satisfactory blow-off valve, provided that care is taken to see that no torsion is present in the tubing in the neighborhood of the blow-off slit. In the case of harelips, the endotracheal catheter is introduced *per os* and is brought out over the tongue and strapped with adhesive in the midline and a direct connection of rubber tubing and catheter is made, reducing the dead space to a minimum. It is important that the tube does not distort the angles of the mouth, as these are landmarks upon which the surgeon depends in making his repair.

In the case of cleft palates, a nasal intubation is done. The catheter is just long enough to reach the vestibule of the nasal passage. I have found a Rowbottom right-angled and tapered connector most satisfactory in these cases. I prefer them to the curved Magill nasal connectors, because they present less difficulty in breaking down the connection and reestablishing it, if suction becomes necessary; and I have so far not been impressed with the argument that disturbing turbulence occurs in the right-angled turn. These connectors—whichever type is used—are connected to the split tube, and again the dead space is reduced to a minimum. Only a trial of one or two passes at blind intubation is made. If these fail of their object a laryngoscope is used and the catheter directed visually into the trachea without or with the assistance of forceps if necessary.

These cases, once under weigh, are surprisingly easy to control. Depression of respiration by over-dosage of intravenous anaesthetic can be coped with by placing a thumb over the slit in the tube and bagging until the respiration returns to normal. A pentothal pusher which delivers 1 c.c. with each complete turn of the dial makes fractional intermittent additions of the barbiturate very easy to control.

The two to one mixture of nitrous oxide and oxygen rarely needs to be changed. As the end of the operation approaches it may be desirable to increase the nitrous oxide percentage, rather than to give more intravenous barbiturate.

Three signs indicate a lightening of hypnosis from the barbiturate and suggest additions of the drug during operation. First, as hypnosis lightens, the rate and amplitude of respiration increases and is obvious in the movements of the bag directly under ones eye. Secondly, as hypnosis wears off, the palpebral fissure, which has been closed, begins to show a bit of eyeball. And thirdly, the introduction of a sucker into the pharynx will reveal the return of pharyngeal reflexes and will indicate that additional fractions of pentothal are required.

With practice and with a knowledge of the surgeon one works with, it is possible to have a patient wide awake within a minute or two of the termination of operation. There is no hangover from the ether induction and the patients

are ready and willing to take feedings within a few hours after operation. This is of particular advantage in the cleft palate cases, in which healing takes place much more rapidly if the patient is able to take sterile fluids soon after operation, which get rid of accumulated blood clots, etc., which formerly became a nidus of infection.

From records which we have kept recently of blood loss, harelips lose from 100 to 200 c.c.; cleft palates, 200 to 300 c.c. of blood. It is, therefore, necessary to have sufficient blood of a suitable type available before operation. The transfusion should start as soon as possible after commencement of the operation and should be adequate to make complete replacement of the blood lost.

Three difficulties we have not yet completely eliminated:

(1) Over-loading of the circulation by intravenous started too early or run in too fast;

(2) Tracheal oedema from long intubation; bucking; or from rough technique; or latent infection in pharynx;

(3) The endotracheal tube, particularly in cleft palates, must be guarded against attack by the surgeon. He may, if he is not watched and warned, slit the tube during his cutting, and then one is likely to have difficulties with an endotracheal catheter plugged with blood clot, and a danger rather than a safeguard.

But with these exceptions, the method is one which is better, in my opinion, than any previously used. Since we started dealing with these cases by this method over five years ago, we have had experience of some morbidity—three cases have required tracheotomy, but there have been no deaths. In by far the large majority of the cases, recovery is almost immediate at the completion of operation, and the patients show little evidence of disturbed physiology.

(A brief movie illustrating the method above described was shown at the termination of the paper.)

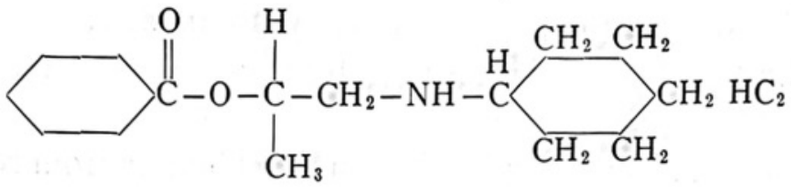
CLINICAL STUDIES ON "CYCLAINE"

F. HUDON, M.D. AND A. JACQUES, M.D.*

Our modern pharmaceutical companies produce each year many local anaesthetic drugs, of which only a few withstand clinical tests.

We would like to report in this paper the results of our experience in 271 cases, using a new anaesthetic drug called "Cyclaine."

This product was brought to our attention by Dr. Strickland of the Sharp and Dohme Co. . . . It is I-cyclohexyl-amino-2-propyl benzoate hydrochloride and has the following formula:



This hexylcaine hydrochloride was synthetised by Cope and his associates (1), recognized as a local anaesthetic by Kuna and Seeler (2), and was used experimentally on the animal by Beyer and his associates (3). Wylde, Walters and Orth (4) have used it for spinal anaesthesia in man. Ruben and Anderson (5) published the first reports on its clinical use.

Cyclaine has a molecular weight of 297.85, is soluble in water up to 12% concentration, has a pH of 4.4 in a 1% solution, and is not altered by sterilization. According to the authors just mentioned, its toxicity is comparable to procaine and metycaine when used subcutaneously, intravenously, or in the spinal canal. On topical application it is as effective as cocaine and for regional anaesthesia it is slightly inferior in strength to "pontocaine".

According to Ruben & Anderson, cyclaine is comparable clinically to procaine and their results are based on 515 cases of spinal anaesthesia using a 1% solution and on 200 cases of regional anaesthesia using concentrations varying between ¼ to 1%. In their 715 cases, they have not encountered any toxic reactions, either local nor general.

As mentioned before, we have used this drug in 271 cases, and to better assess its value we have used procaine and xylocaine in an equal number of cases and during the same period of time.

Following is a record of our findings: In spinal anaesthesia, cyclaine was used in concentration of 1% and we have found this solution very satisfactory, analgesia lasting approximately 90 minutes. In regional anaesthesia, we first used the 2% solution and then the 1% solution with or without Adrenalin. For example: in a brachial plexus block, this solution, without Adrenalin, is adequate for the reduction of a dislocated shoulder or elbow joint, or for the reduction of fractures of the clavicle, the arm or the fore-arm. But, as analgesia of the hand is not always complete, the addition of 2 drops of Adrenalin to 40 c.c. of the solution will give complete anaesthesia in 3 to 8 minutes and lasting well over 2 hours.

The same applies to caudal anaesthesia. For operations on the anal region, the

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1% solution without Adrenalin is effective; but for circumcision, castration, vaginal repair and such operations, 2 drops of Adrenalin should be added to the 40 c.c. of the solution.

As a topical anaesthetic in the throat, we have used 5 to 7cc of a 5% solution as a spray. We were impressed by the rapidity of action, the complete analgesia, and the absence of anxiety of the patient. During induction with pentothal anaesthesia, cyclaine seems to control a laryngospasm quicker than any other drug. Intubation therefore, may be performed in a few seconds without curarisation, 200 to 500 mgms of pentothal being sufficient to produce hypnosis and allow spraying. There is no "bucking", respiratory rate is slowed to about 16 but amplitude remains normal. The patient's color remains good and assisted respiration, if necessary, is very easy. Curare may then be added according to the surgical needs. With this technique, pulmonary ventilation is autonomic during induction and the physiological respiratory exchanges are maintained. We believe this is the result of slight dilatation of the bronchi and bronchioles. The blood lost figured by weight seems remarkably decreased.

As a result of the foregoing observations, we have modified our technique of anaesthesia in tonsillectomies. Previously, intubation was performed under pentothal-curare and anaesthesia was maintained with N_2O and Trilene. Now, intubation is performed under a slight amount of pentothal intravenously, and Cyclaine in topical application. As no curare is used, there is no respiratory paralysis, the bronchioles are not constricted but are slightly dilated, thus eliminating lower obstruction with subsequent congestion. The venous pressure is therefore not increased and oozing is decreased.

"Cyclaine" has been used topically in the throat as a 5% solution in over 50 cases without any toxic reaction. During the same period, we had 4 cases of circulatory collapse after spraying the throat with a 2% pontocaine solution.

The only complication we can report are two cases of convulsions after injection of 60 c.c. of a 2% solution of "cyclaine". Both patients appeared normal, were not in any state of anxiety, the pulse and blood pressure had remained unchanged when suddenly, while talking with the anaesthetist, they went into convulsions. This was immediately controlled by intra-venous pentothal. As premedication, they had received morphine gr. 1/6 and atropine gr. 1/150. It was following these two reactions that we gave up the 2% solution.

During the same period of time, we have used 2% xylocaine and have noticed some cases of nervous reactions and emotional disorders such as tears and agitation lasting for approximately 30 minutes. Doses in these cases were about 40 c.c.

Aside from these two cases of convulsions, there is nothing to be mentioned in the chapter of complications. In other words, we did not encounter anxiety, fear, dyspnea, pallor, diaphoresis, or changes in pulse. As for blood pressure, there often appeared a slight rise of 10 mm Hg.

Briefly, we may say, and this is to the advantage of "cyclaine", that the circulatory side effects are absent, that emotional disorders if pre-existing are

not increased, and that the nervous reactions such as convulsions are caused by concentrations and doses unnecessary for adequate anaesthesia.

Analgesia is rapid and sufficiently long lasting.

We have found it very satisfactory in topical analgesia, in caudal and spinal anaesthesia and in brachial plexus blocks.

The patient does not experience the disagreeable feeling of weakness, anxiety and nausea, so common with other local anaesthetics. As a general rule, there is a parallelism between potency, toxicity and irritation in local anaesthetic drugs. In "cyclaine" it seems that potency is higher compared to its toxicity. The authors mentioned above give a therapeutic index in animals of 584 as compared to 353 for procaine.

Procaine as compared to 54 for cocaine is still the least irritating of all local anaesthetic drugs and this is the reason why we prefer this drug to any other for repeated therapeutic infiltrations. "Cyclaine" as other local anaesthetic agents gives a transient burning sensation following the injection, but after its use, we have not met with any local reaction in the tissues, in the nerves nor in the spinal cord. To our knowledge, no cases of neuritis have been reported.

We were impressed by a case of pain of the upper limb appearing 4 days after the repair of lacerated fingers. Three weeks later, after failure of all ordinary therapeutic measures, a brachial plexus block using 30 c.c. of 1% solution of "cyclaine" completely and definitely relieved the pain.

In conclusion, we believe that "cyclaine" deserves further study. Compared to procaine, it is more rapid and two or three times more potent. It produces less general reactions and less emotional disorders. It seems especially applicable as a topical anaesthetic in the pharynx and larynx and, therefore, would seem very valuable in bronchoscopic work in replacing other drugs which so often give circulatory or nervous reactions. It should be preferred to cocaine which when swallowed is quickly absorbed by the gastric mucosa, causing severe toxic reactions. "Cyclaine" is much less toxic and can even be administered intra-venously. The drug has been given intra-venously to man in doses of 1000 mg. by slow intra-venous drip in the same manner as procaine.

It is our impression that "cyclaine" will improve our technique of intubation, that it will favour respiratory exchanges by decreasing the use of curare. With its use intubation is performed very easily. We cannot measure as yet all the dangers or the side effects of the drug, but since the benzoic acid portion contains no amino grouping, it is our belief that the side effects are minimized. As the plasma esterase has no effect on the product, it is thought that the drug remains longer in the blood stream. Apparently this is another advantage. This paper is based on clinical impressions. There was no electrocardiographic control.

REFERENCES

1. Hancock, E. M., Hardy, E. M., Heyl, D., Wright, M. E., and Cope, A. C. Amino-Esters, J. Am. Chem. Soc. *66*: 1747 (1944).
2. Kuna, S., and Seeler, A. O. Studies on Local Anaesthetic Properties of 131 Alkylamino alcohols and Their Esters, J. Am. Chem. Soc. *66*: 1747 (1944).

3. BEYER, K. H., LATVEN, A. R., FREYBURGER, W. A., and PARKER, M. P. Study of Activity and Toxicity of Hexylcaine; New Local Anaesthetic Agent. J. Pharmacol. & Exper. Therap. *93*: 388–400 (1948).
4. WYLDE, R. M., WATERS, D. M., and ORTH, O. S. Cyclaine (D109), a new local anesthetic agent. Am. J. Med. *4*: 477 (1948).
5. RUBEN, J. E. and ANDERSON, E. Hexylcaine Hydrochloride. Am. J. Surg., *78*: 843–846. (1949).

SUCCINYLCHOLINE: A NEW APPROACH TO MUSCULAR RELAXATION IN ANESTHESIOLOGY*

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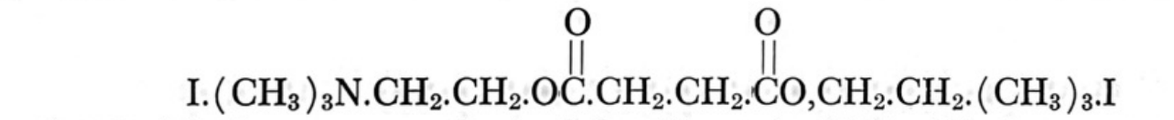
JOSE M. BORREGO-HINOJOSA, M.D.†

It is generally accepted that the ideal muscle relaxant should have: 1. specificity of action; 2. rapid onset of action; 3. readily controllable intensity; 4. wide margin between muscular relaxation and respiratory arrest; and 5. rapid and complete recovery following the cessation of its administration. The agents hitherto employed for the production of muscular relaxation in surgical anesthesia all fell short of the above requirements. Depending on the agent used, 3 to 8 minutes were required for the development of maximal effect. (1–3) There was more or less marked respiratory depression with all agents, and in a certain percent of patients adequate muscular relaxation could not be obtained without transient respiratory arrest. (1–3) Salivation, excessive bronchial secretion, bronchospasm, tachycardia, hypotension, hypertension, were encountered in various combinations with the different muscle relaxants. (1–6) The duration of action of a single effective intravenous dose varied from 10 to 30 minutes with these agents, and even longer time was occasionally required for the complete restitution of neuromuscular conduction after prolonged administration of fractional doses. Occasionally profound respiratory depression of long duration could be observed. (1–3, 7)

Observations made on anesthetized patients with a recently introduced synthetic muscle relaxant, succinylcholine, indicated that this agent, administered in the form of continuous intravenous infusion, approximated most closely the definition of the ideal muscle relaxant. Its chemical formula is as follows:



Succinylcholine was synthesized by Hunt in 1906. (8) Its neuromuscular blocking activity was first observed in 1949 by Bovet, (9) and reported simultaneously and independently by Phillips. (10) Subsequently it was investigated by Castillo and de Beer (11) and Castillo et al. (12) in various laboratory animals. Succinylcholine belongs to the "depolarizing" type of neuromuscular blocking agents. (13) Its effect is intense, brief and reproducible. The short duration of the action of a single intravenous dose was attributed to the rapid enzymatic hydrolysis of succinylcholine. (10–12, 14–16) The duration of action of succinylcholine could be prolonged by physostigmine. (12)

Clinical trials with succinylcholine were reported by Brücke et al., (17) Thesleff (18) and Dardell and Thesleff (19) and with a closely related com-

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pound, di-(B-ethyl-dimethylammonium-ethyl) succinate di-iodide by Voldoni (20) and Ottolenghi (20) and also by Scurr. (21)

Material and methods.

The observations to be presented were made on 202 unselected consecutive patients in whom the use of muscle relaxant was indicated. Of these 79 were males and 123 were females. The age distribution of the patients is presented in Table I.

TABLE 1

AGE DISTRIBUTION OF PATIENTS

Age Group	*Number*	*Per cent*
11–20	5	2.5
21–30	17	8.4
31–40	35	17.3
41–50	57	28.3
51–60	48	23.8
61–70	26	12.8
71–80	10	4.9
81–90	4	2.0
	202	100.0

Anesthetic technique: Patients, following premedication with pentobarbital, atropine or scopolamine, and morphine, were anesthetized by the intravenous injection of a 2.5% Pentothal Sodium solution administered through the rubber sleeve of an intravenous infusion of 5% dextrose started previously. After the depth of anesthesia progressed to plane 1 or 2 of stage III, 10 to 45 mg. of succinylcholine diiodide* were administered intravenously. Forty five to sixty seconds later the cords were visualized by direct laryngoscopy, sprayed with a 1% Pontocaine solution, and an endotracheal tube was inserted. The intubation was usually completed within 1 to 2 minutes from the end of the administration of succinylcholine. Following intubation, a continuous intravenous drip of an 0.2% solution of succinylcholine diiodide in normal saline or in 5% dextrose in water was started immediately after intubation through a no. 23 needle also inserted into the rubber sleeve of the intravenous infusion started before the induction of anesthesia. The degree of muscular relaxation required was obtained by regulating the rate of flow of the succinylcholine solution with a "tunnel clamp".* From then on the anesthesia was conducted as described previously. (1–3)

The cases in which succinylcholine was used for the production of muscular relaxation are listed in Table 2.

*Succinylcholine diiodide was made available to us through the courtesy of Dr. E. J. de Beer of Burroughs Wellcome & Co.

*The tunnel clamp can be obtained from the Harvard Instrument Company of Dover, Massachusetts.

Results

The rapid intravenous injection of 10 to 45 mg. of succinylcholine diiodide was occasionally followed in 30 to 45 seconds by muscular twitchings of 5 to 15 seconds duration. At the same time the respiratory depth also increased. Muscular relaxation developed in 45 to 60 seconds and reached its maximum in 60 to 90 seconds. After 180 seconds, the effect of a single injection of succinylcholine started to decrease, and disappeared within 300 seconds.

Muscular relaxation could be maintained by the continuous intravenous infusion of 1.1 to 9.2 mg. of succinylcholine diiodide per minute. The average per minute dose of succinylcholine diiodide was 4.0 mg. The degree of relaxation could be changed within 30 seconds by regulating the flow rate. It was our

TABLE 2

TYPES OF OPERATIONS

Operation	*Number*	*Percent*
Gastric surgery	43	21.3
Gallbladder surgery	57	28.2
Other upper abdominal surgery	38	18.8
Large bowel surgery	20	9.9
Appendectomy	8	4.0
Intra-abdominal gynecological operations	36	17.8
	202	100.0

clinical impression that good muscular relaxation could be obtained with less respiratory depression than with any of the other muscle relaxants. Assisted respiration was seldom necessary and respiratory arrest never developed, except when produced deliberately with excessive flow rates. The effect of succinylcholine on muscular relaxation and respiratory depth completely disappeared in all cases within 2 to 4 minutes after the intravenous infusion was discontinued.

The quantities of succinylcholine and Pentothal Sodium used are summarized in Table 3.

TABLE 3

SUCCINYLCHOLINE DI-IODINE AND PENTOTHAL SODIUM REQUIREMENTS

	Range	*Average*
Duration of anesthesia	35–363 Min.	126 Min.
Initial dose of succinylcholine di-iodide	10–50 Mg.	29.2 Mg.
Total dose of succinylcholine di-iodide	66–1830 Mg.	507 Mg.
Dose of succinylcholine di-iodide per min.	1.1–9.2 Mg.	4.0 Mg.
Initial dose of pentothal sodium	250–1000 Mg.	560 Mg.
Total dose of pentothal sodium	540–3500 Mg.	1432 Mg.
Dose of pentothal sodium per min.	2.3–34.8 Mg.	13.1 Mg.

The quantity of Pentothal Sodium per minute necessary for the maintenance of an adequate depth of anesthesia, characterized by the absence of breath-holding, was found to be the same as with Syncurine (1) but somewhat greater than with Mytolon Chloride (2) or Flaxedil. (3) The significance of this finding

will be discussed later. Despite this, 50% of the patients reacted to stimuli after the removal of the endotracheal tube. The average recovery time in the rest of the patients was considerably longer (79 minutes) than after the use of other relaxants. (1–3) In contrast to this, respiratory depression seen in 12 to 21% of the patients after the use of other muscle relaxants, (1–3) was not observed in any of the patients after the use of succinylcholine.

Postoperatively atelectasis was observed in 2, and moderately increased bronchial secretions were found in 29 of the 202 patients who received succinylcholine for prolonged periods. The only other serious respiratory complication was bronchopneumonia in a debilitated patient with peritoneal carcinomatosis. This patient came to the operating room with a temperature of 103.4°F. Through a small laparatomy incision 8000 cc. of fluid was removed. Bronchopneumonia, diagnosed a few hours postoperatively, responded promptly to intramuscular penicillin,

The incidence and severity of other postoperative complications did not differ significantly from those observed after the use of other muscle relaxants. There were 10 postoperative deaths in this series. The deaths occurred 2 to 49 days postoperatively. In no case was there any connection between the patient's death and the use of succinylcholine.

Comments

Compared to other muscle relaxants used in anesthesiology, succinylcholine possesses several advantages. In our experience the outstanding advantage of succinylcholine is its easy controllability which permitted almost instantaneous changes in the degree of muscular relaxation. There is no other agent used in anesthesiology which can be as readily controlled as succinylcholine. Though it is possible to increase rapidly the depth of intravenous barbiturate anesthesia, recovery is never prompt. Even with the most rapidly acting inhalation agents, such as cyclopropane and ethylene, it takes minutes to deepen or lighten the depth of anesthesia. With succinylcholine, both the increasing and decreasing of muscular relaxation took less than a minute.

The reason for this ideal controllability is the fact that succinylcholine, in contrast to the other generally employed muscle relaxants, is rapidly hydrolyzed by both cholinesterase (plasma cholinesterase, non-specific cholinesterase) and acetylcholine esterase (true cholinesterase). (15) It has been shown previously that the hydrolysis of procaine and 2-chloroprocaine by the plasma cholinesterase (22) is a zero order reaction. (23) It seems permissible to assume that succinylcholine is hydrolyzed in an identical manner. This would mean that above a certain critical concentration the quantity of succinylcholine hydrolyzed per minute in the body would be independent of its concentration. Consequently, a more rapid continuous administration of succinylcholine will cause an elevation of its concentration and produce a more profound muscular relaxation. On the other hand, the enzymatic hydrolysis of succinylcholine diiodide is so rapid in vivo that in the average patient, the effect of a 30 mg. dose, that produces com-

plete muscular relaxation, completely disappears within 5 minutes. This rapid, and to all appearances, complete hydrolysis of succinylcholine explains the lack of residual respiratory depression frequently seen with other muscle relaxants that are not similarily disposed of within the organism. The residual respiratory depression with other muscle relaxants that can be profound and prolonged in the occasional patient presented a serious enough problem to necessitate the use of various "anti-curare" drugs. (24, 25) These drugs, physostigmine, prostigmine, Tensilon, etc. are only effective against the "anti-depolarizing" group of muscle relaxants (d-Tubocurarine, Metubine, Flaxedil), and their use can also be accompanied by various unwanted side effects (26) and even fatalities. (27,28) Recently a compound was synthetized that is capable of antagonizing the effect of Syncurine on neuromuscular conduction. (12) The dangers that might be encountered with the use of these drugs have been discussed elsewhere. (29) Because of its rapid and complete hydrolysis, these problems are non-existent with the use of succinylcholine.

It was observed that the rapid intravenous injection of 100 mg. of procaine in patients receiving a muscle relaxing dose of succinylcholine increased the degree of muscle relaxation and produced respiratory depression. Since procaine is also hydrolyzed in the organism by the non-specific choline esterase, (22) the additive effect of procaine administration on succinylcholine activity can be looked upon as substrate competition for the same enzyme. Further observation on this phenomenon will be presented elsewhere. (30)

Another advantage of succinylcholine is its relative sparing effect on respiration. With comparable muscular relaxation, there is definitely less diminution of respiratory depth, and the respiratory movements are smooth, in contrast to the jerky respiration frequently seen with other muscle relaxants. At this time, we have no explanation of this favourable circumstance. It is possible that the depressant effect of succinylcholine on the central nervous system is less marked than that noted with other muscle relaxants. (31) Ellis et al. (32) found that the depressant effect of succinylcholine on the respiratory center was less than that of Syncurine or d-Tubocurarine.

The lack of unwanted side effects with the use of succinylcholine was very gratifying. No marked circulatory changes were seen (see Table 4). The slight variations in pulse rate and blood pressure seemed to be dependent on surgical manipulation. If anything, there was a tendency for a slight elevation pulse rate and systolic blood pressure. Salivation, excessive bronchial secretions, laryngeal spasm, bronchospasm, or other evidence of histamine release were not observed. There was no indication of blocking or stimulating action on the autonomic nervous system.

The disadvantages of succinylcholine are not serious. The first of these is that its administration requires greater attention on the part of the anesthesiologist. The rate of its administration has to be regulated by adjusting the drops delivered per minute. The rate of flow has to be checked periodically and adjusted to the degree of relaxation required by the surgeon. The use of a

tunnel clamp and stop watch or Flowrator (33) facilitates the rapid regulation of the flow.

The second disadvantage of succinylcholine in our experience was that somewhat higher per minute doses of Pentothal Sodium were required for the maintenance of comparable depth of anesthesia than with the other synthetic muscle relaxants. (29) Due to this, though the percent of patients who reacted to light stimulation at the end of anesthesia compared favorably with that seen with other muscle relaxants, the average recovery time of the remainder of the patients was considerably longer (79 minutes as compared to from 33 to 48 minutes).

TABLE 4

CIRCULATORY CHANGES

	Pulse Rate		*Systolic Blood Pressure mm Hg.*		*Diastolic Blood Pressure mm Hg.*	
	Range	*Average*	*Range*	*Average*	*Range*	*Average*
Initial	58–136	85	80–300	122	40–142	75
10 minutes after start of anesthesia	48–180	89	74–228	120	38–142	76
Lowest value observed during anesthesia	48–120	81	68–222	106	34–122	69
Highest value observed during anesthesia	70–180	100	84–280	139	50–160	87
At the termination of anesthesia	60–140	93	85–260	123	40–122	75

Technic	*No.*	*Concentration*	*With or without epinephrine*	*Failures*	*Complication*	*Headache*	*Lumbar pain*
Brachial plexus block	80	1%–2%	1/200,000	7 (9%)	convulsion (1)		
Caudal	55	1%–2%	1/200,000	6 (11%)	convulsion (1)	3	
Spinal	57	1%		2 (4%)		3	2
Peridural	5	1%					
Therapeutic infiltrations	20	0.5%–1%					
Topical	50	5%					
Total	267						

Note: Up to the present, our statistics cover 530 cases; 202 of which were brachial blocks, 110 caudal anesthesias, and 132 topical anesthesias.

That the per minute dose of Pentothal Sodium was greater with succinylcholine than with the other muscle relaxants studied previously might be due to the fact that the succinylcholine molecule has less depressant action on the central nervous system. (22)

The available data on the effects of succinate on barbiturate anesthesia are controversial. (34) It was suggested by Soskin and Taubenhaus (35) that succinate might be a physiologic antidote in barbiturate anesthesia. Recently, Price demonstrated that the intracarotid injection of sodium succinate increased the electrical activity of the brain in rabbits. (36) On the other hand, Carson et al. (37) found that the recovery time in barbiturate anesthesia was not significantly shortened by succinic acid. The investigations of Tucci et al. (38) and Vandewater and Gordon (39) also gave negative results. Lamson et al. (40) in guinea pigs found that succinates prolonged the duration of sleep after the administration of barbiturates. On the basis of presently available evidence, it seems more likely that the less marked central depressant effect of the succinylcholine molecule, and not the antagonistic effect of the succinate molecule is responsible for the somewhat increased Pentothal Sodium requirements.

The occasional muscle twitching that can be seen on rapid administration of succinylcholine also occurs, though less frequently with the use of Syncurine. (13) This transient muscular twitching is due to the initial stimulating effect of these depolarizing compounds on skeletal muscle. A slower rate of intravenous injection of Syncurine and succinylcholine will usually prevent these fasciculatory movements.

The advantages of succinylcholine as a muscular relaxant in anesthesiology far outweighed its disadvantages. The greater attention required on the part of the anesthesiologist was amply rewarded by the greater flexibility afforded by this agent. Similarly the occasionally observed prolonged sleeping time due to the increased quantity of Pentothal Sodium used was more than compensated for by the complete absence of residual respiratory depression and the lack of side effects.

On the basis of our experience, succinylcholine is the muscle relaxant of choice, especially so in the debilitated, dehydrated, and aged patients in whom prolonged postoperative respiratory depression with other agents is most common (29).

It is highly significant, both from the pharmacological and clinical point of view, that succinylcholine, exerts its desired effect rapidly, is readily controllable, and is easily hydrolyzed into substances that in the dose range employed have no harmful effects. The need for such ultra-short acting agents in anesthesiology is readily apparent, and it is hoped that the clinical use of succinylcholine will stimulate the search for other agents, e.g. barbiturates, with ultra-short activity.

Summary

1. Succinylcholine is an ultra-short acting "depolarizing type" muscle relaxant.
2. It has a rapid onset and short duration of action.
3. This short duration of action is due to the rapid enzymatic hydrolysis of succinylcholine within the organism.
4. For prolonged muscular relaxation it has to be administered in continuous intravenous infusion. The degree of muscular relaxation can be easily controlled.

5. It has a relative sparing effect on respiration during its administration, and because of its rapid hydrolysis its use is not followed by respiratory depression.

6. Its use is not accompanied by unwanted side effects and the incidence of postoperative complications after its use is low.

7. On the basis of limited experience, succinylcholine seems to be the muscle relaxant of choice of all similar agents so far investigated.

Addendum:

Since this paper was submitted for publication several instances of prolonged respiratory depression have been reported following the use of succinylcholine. (41,42,43,44) As pointed out elsewhere (45,46,47) unnecessarily high doses of succinylcholine were used in these cases. It has also been shown that the respiratory depressant effect of a given dose of succinylcholine is inversely proportional to the plasma cholinesterase activity of the patients. (48,49) According to our experience in over 500 cases in which succinylcholine was administered in the manner described in this paper, no prolonged respiratory depression was observed. Low plasma cholinesterase activity could be detected from the effects of the initial dose of succinylcholine and if the maintenance dose was adjusted to the needs of the patients the restitution of respiration after the discontinuation of the succinylcholine administration was not markedly different from that in patients with normal plasma cholinesterase activity. On the other hand, if patients with low plasma cholinesterase activity receive unnecessarily high doses of succinylcholine, the development of prolonged respiratory depression cannot be avoided.

REFERENCES

1. Foldes, F. F., and Machaj, T. S. Syncurine (Decamethonium-bromide): Its use with Pentothal Sodium and nitrous oxide-oxygen anesthesia in abdominal surgery. Anesthesiology 12:366–375, 1951.
2. Foldes, F. F., Hunt, R. D., and Ceravolo, A. J. The use of Mytolon Chloride with Pentothal Sodium and nitrous oxide-oxygen for abdominal surgery. Anesth. and Analg. To be published.
3. Foldes, F. F., Machaj, T. S., and Chapman, P. L. The use of Flaxedil with Pentothal Sodium-nitrous oxide-oxygen anesthesia in intra-abdominal surgery. To be published.
4. Comroe, J. H., and Dripps, R. D. The histamine-like action of curare injected intracutaneously and intra-arterially in man. Anesthesiology 7:260–262, 1946.
5. Grob, D., Lilienthal, J. L., and Harvey, A. M. On certain vascular effects of curare in man: The "histamine" reaction. Bull. Johns Hopkins Hosp. 80:229–322, 1947.
6. Landmesser, C. M. A study of the bronchoconstrictor and hypotensive action of curarizing agents. Anesthesiology 8:506–523, 1947.
7. Harris, L. C., and Dripps, R. D. Use of decamethonium bromide for the production of muscular relaxation. Anesthesiology 11:215–233, 1950.
8. Hunt, R., and Taveau, R. deM. On the physiological action of certain choline derivatives and new methods for detecting choline. Brit. M.J. 2:1788–1791, 1906.
9. Bovet, D., Bovet-Nitti, F., Guarino, S., Longo, V. G., and Marotta, M. Proprieta farmacodinamide di alcumi derivati della succinilcolina dotati di azione curanica. Rendiconti 1st. Sup. Sanita 12:106–137, 1949.
10. Phillips, A. P. Synthetic curare substitutes from aliphatic dicarboxylic acid aminoethyl esters. J. Am. Chem. Soc. 71:3264, 1949.

11. Castillo, J. C., and de Beer, E. J. Neuromuscular blocking action of succinylcholine (diacetylcholine). J. Pharmacol. and Exper. Therap. 99:458–464, 1950.
12. de Beer, E. J., Castillo, J. C., Phillips, A. P., Fanelli, R. V., Wnuck, A. L., and Norton, S. Synthetic drugs influencing neuromuscular activity. Ann. New York Acad. Sc. 54:362–372, 1951.
13. Paton, W. D. M., and Zaimis, E. J. Actions and clinical assessment of drugs which produce neuromuscular block. Lancet 2:568–570, 1950.
14. Glick, D. Some additional observations on the specificity of cholinesterase. J. Biol. Chem. 137:357–362, 1941.
15. Bovet-Nitti, F. Degradazione di alcune sostanze curarizzanti per azione di colinesterasi. Rendiconti Ist. Sup. Sanita. 12:139–157, 1949.
16. Whittaker, V. P. Idrolisi della succinilcolina da colinesterasi: Utilizzazione simultania della cromatografia su carta e della tecnica di Warburg. Experientia 7:217–218, 1951.
17. Brücke, H., Ginzel, K. H., Klupp, H., Pfaffenschlager, F., and Werner, G. Bis-cholinester von Dicarbonsauren als Muskel relaxantien in der Narkose. Wien. Klin. Wochenschrift 63:464–466, 1951.
18. Thesleff, S. Pharmacological and clinical tests with L.T. 1 (0.0-succinylcholine iodide). Nordisk Med. 46:1045, 1951.
19. Dardel, O. V., and Thesleff, S. Clinical results with succinylcholine iodide, a new muscle relaxant. Nordisk Med. 46:1308–1311, 1951.
20. Report on congress of anaesthetists. Brit. M.J. 2:668–669, 1951.
21. Scurr, C. F. A relaxant of very brief action. Brit. M.J. 2:831–832, 1951.
22. Kalow, W. Identity of procainesterase and pseudocholinesterase. Feder. Proc. 10:312, 1951.
23. Aven, M., and Foldes, F. F. The chemical kinetics of procaine and choloprocaine hydrolysis. Science 114:206–208, 1951.
24. Wescoe, W. C., and Riker, W. F., Jr. The pharmacology of anti-curare agents. Ann. New York Acad. Sc. 54:438–459, 1951.
25. Randall, L. D. Synthetic curare-like agents and their antagonists. Ann. New York Acad. Sc. 54:460–479, 1951.
26. Artusio, J. F., Jr., Marbury, B. E., and Crews, M. A. A quantitative study of d-tubocurarine, tri-(diethylamino-ethoxy) 1,2,3 benzene (Flaxedil) and a series of tri-methyl and dimethylammonium compounds in anesthetized man. Ann. New York Acad. Sc. 54:512–529, 1951.
27. Macintosh, R. R. Death following injection of neostigmine. Brit. M.J. 1:852, 1949.
28. Clutton-Brock, J. Death following neostigmine. Brit. M.J. 1:1007, 1949.
29. Foldes, F. F., Machaj, T. S., Hunt, R. D., Carberry, P. L., and McNall, P. G. The use of synthetic muscle relaxants in anesthesiology. To be published.
30. Foldes, F. F., McNall, P. G., Davis, D. L., Ellis, C. H., and Wnuck, A. L. Substrate competition between succinylcholine and procaine for plasma cholinesterase. To be published.
31. McIntyre, A. R., Bennett, H. L., and Hamilton, C. Recent advances in the pharmacology of curare. Ann. New York Acad. Sc. 54:301–306, 1951.
32. Ellis, C. H., Norton, S., and Morgan, W. V. Central depression by drugs which block neuromuscular transmission Feder. Proc. 11:42–43, 1952.
33. Graubard, D. J., and Peterson, M. C. Clinical uses of intravenous procaine. 63 pp. Springfield: C. H. Thomas.
34. Quastel, J. H., and Wheatley, A. H. M. Narcosis and oxidations of brain. Proc. Royal. Soc. London 112:60, 1932.
35. Soskin, S., and Taugenhaus, M. Sodium succinate as antidote for barbiturate poisoning and in control of duration of barbiturate anesthesia (including its successful use in care of barbiturate poisoning in human). J. Pharmacol. and Exper. Therap. 78:49–55, 1943.
36. Price, R. D. Montreal Neurological Institute. Personal Communication.
37. Carson, S. A., Koppanyi, T., and Vivino, A. E. Studies on barbiturates: effect of succinate and fumarate in experimental barbiturate poisoning. Anesth. and Analg. 24:177–192, 1945.
38. Tucci, J. H., Brazier, A. B., Miles, H. H. W., and Finesinger, J. E. A study of pentothal sodium anesthesia and a critical investigation of the use of succinate as an antidote.

Anesthesiology 10:25–39, 1949.
39. Vandewater, S. L., and Gordon, R. A. Sodium succinate in control of Pentothal anesthesia. Anesth. and Analg. 31:97–104, 1952.
40. Lamson, P. D., Greig, M. E., and Hobdy, G. J. Modification of barbiturate anesthesia by glucose, intermediate metabolites and certain other substances. J. Pharmacol. and Exper. Therap. 103:460–470, 1950.
41. Love, S. H. S. Prolonged apnoea following scoline. Anaesthesia 7:113–114, 1952.
42. Gould, R. B. Succinylcholine chloride. Correspondence Brit. Med. J. 1:440, 1952.
43. Harper, J. K. Prolonged respiratory paralysis after succinylcholine. Correspondence Brit. Med. J. 1:866, 1952.
44. Hewer, C. L. Prolonged respiratory paralysis after succinylcholine. Correspondence Brit. Med. J. 1:971–972, 1952.
45. Foldes, F. F. Prolonged respiratory paralysis after succinylcholine. Correspondence Brit. Med. J. 1:1352, 1952.
46. Foldes, F. F. Prolonged apnoea following scoline. Correspondence Anaesthesia 7:181–182, 1952.
47. Foldes, F. F. Succinylcholine. Letters to the Editor Lancet 2:245–246, 1952.
48. Bourne, J. G., Collier, H. O. J., and Sommers, G. F. Succinylcholine (succinoylcholine) muscle-relaxant of short action. Lancet 1:1225–1229, 1952.
49. Evans, F. T., Gray, P. W. S., Lehman, H., and Silk, E. Sensitivity to succinylcholine in relation to serum-cholinesterase. Lancet 1:1229-1230, 1950.

THE ANAESTHETIST AND THE BRONCHOSCOPE*

R. A. Gordon, M.D.*

I. Introduction

The bronchoscope was introduced by Killian in 1897. Modern instruments which are widely used today have been designed by Jackson, Negus, and others. The usual instrument used on the North American continent is the Jackson bronchoscope. Until recent years the use of the bronchoscope was considered to be the sole prerogative of the Otolaryngologist, and only too often this attitude persists in our hospitals today. Because this attitude is so persistent, I think that it is timely to discuss those situations in which it is imperative that the anaesthetist should be able to use this instrument with some facility.

II. The Bronchoscope in Anaesthesia—Historical

The use of the bronchoscope in anaesthesia was pioneered by Dr. Ivan Magill of London, England, who in 1930 described his technique of bronchial occlusion by a cuffed suction tube under direct vision. In 1936 Magill described his technique of endobronchial intubation under direct vision. Since about 1939 there has been increasing advocacy for the treatment of post-operative pulmonary atelectasis by bronchoscopic aspiration of obstructing secretion.

It is my purpose to discuss with you what I consider to be the indications for use of the bronchoscope by the anaesthetist in various situations occurring before, during and after anaesthesia.

III. Use of the Bronchoscope by the Anaesthetist

(1) *Before Operation*

Not infrequently the anaesthetist will encounter circumstances in which the use of the bronchoscope immediately before operation will so improve the respiratory function of a patient as to make an otherwise impossible anaesthetic a smooth and orderly procedure, sometimes changing an otherwise inoperable patient into a fair operative risk and at other times preventing serious post-operative respiratory complications. These circumstances arise in patients who have large accumulations of secretions in the tracheo-bronchial tree, or who have aspirated foreign bodies or blood as a result of facial injuries. In my experience this procedure has been useful and in some cases indispensable in paraplegic patients with high lesions and in unconscious patients who have had head injuries or other intracranial catastrophies. The paraplegic patient with a high lesion of the spinal chord is unable to clear secretions from the trachea and bronchi by coughing, and is not infrequently presented for an exploratory laminectomy with respiration embarrassed not only by paresis, but also by a great accumulation of secretion in the tracheo-bronchial tree, and perhaps actual pulmonary atelectasis. Unconscious patients, particularly those with mid-brain injury with a suppressed cough reflex, may present with the same accumulation of secretions in the respiratory tract. The clearing of the tracheo-bronchial tree through a bronchoscope immediately after induction of anaesthesia in these

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patients will usually permit of a smooth operative course with adequate oxygenation. I do not believe that adequate results can be obtained in these patients by any other method.

Every anaesthetist who has had much experience of thoracic surgery has met the bronchiectatic patient who presents with a great quantity of secretion in the bronchiectatic areas of the lungs, which drifts into the bronchi and trachea during induction or positioning of the patient, cannot be removed by suction through an endotracheal tube, and by obstructing respiration prejudices the whole course of the operative intervention. In these patients a thorough cleansing of the tracheo-bronchial tree as soon as may be reasonable after the induction of anaesthesia saves much subsequent embarrassment of respiratory function, and I am sure will prevent many operative deaths.

The patient who has suffered facial injuries not infrequently aspirates fractured teeth and other foreign bodies into the respiratory tract. Whenever such an aspiration is suspected (such suspicion should be aroused by missing teeth for which no accounting can be made), these patients should have immediate radiological investigation of the chest and any foreign body demonstrated should be removed immediately by the anaesthetist after induction and before insertion of the endotracheal tube. It may be profitable, too, to employ the bronchoscope to recover blood clot from the bronchi of such patients, and every such patient who has signs of atelectasis before operation should be bronchoscoped before the introduction of the endotracheal tube.

(2) *During Anaesthesia*

The technique of bronchial occlusion as practised for the operations of lobectomy and pneumonectomy requires the placement of an occluding cuff under direct vision through the bronchoscope. No other method can be considered satisfactory. While endobronchial anaesthesia can be readily accomplished without the use of the bronchoscope, I feel that the endobronchial tube should always be placed under direct vision by Magill's technique where there are quantities of secretion or blood within the tracheo-bronchial tree.

In certain emergencies arising during anaesthesia the use of the bronchoscope is life saving. This is true in occlusion of the glottis by laryngospasm or edema after induction, in tracheal obstruction becoming complete after induction of anaesthesia, and in aspiration of vomitus. Occlusion of the glottis due to laryngospasm during induction is an ever present danger in certain operations performed on patients with paralysis of the vocal cords following division of the recurrent laryngeal nerve. We are all aware too that it may occur under other circumstances, and that the problem of intubation in these circumstances may be most serious. It is well to remember that a rigid bronchoscope can always be inserted between the cords under these circumstances, and if used with care will produce no damage to the cords. Laryngeal occlusion may also be the result of edema, either post-traumatic or inflammatory in origin. Two common inflammatory causes are Ludwig's angina and laryngotracheitis in children. In these circumstances I feel most strongly that a bronchoscope should be introduced rather than pro-

ceeding with many futile attempts at intubation with an endotracheal tube, and the bronchoscope will then serve to fix the trachea while a tracheotomy is done. Tracheotomy is mandatory in occlusion of the glottis due to edema.

Occasionally one encounters tracheal obstruction due to tumours of the thyroid or tumours in the superior mediastinum, and such obstruction may suddenly become complete on induction of anaesthesia. In some of these cases immediate endotracheal intubation may be successful in re-establishing the airway, but this is not always true, and blind passage of a tube in these cases may be dangerous and result in rupture of the trachea. I feel that in a case of this type of obstruction the introduction of the bronchoscope and its passage under direct vision beyond the point of obstruction is a safer and more certain procedure, and it will undoubtedly be successful in those cases where ordinary endotracheal intubation fails to re-establish the airway.

The aspiration of vomitus during anaesthesia is a major catastrophe which calls for immediate bronchscopy with removal of all foreign material which can be visualized. To illustrate the value of immediate bronchoscopy in these patients I wish to report briefly three cases of a number which have occurred in my own hospital in which it was life saving.

Case 1 Parturition. Para I. Gr. II.

Anaesthesia commenced as head came on to perineum–N_2O/O_2 with Trichlorethylene. As the head was born the patient vomited large quantities of dark coloured fluid and went into spasm with the jaws firmly closed. Vomitus continued to pour from her nose, and she became acutely anoxic. Twelve mgm. of d-tubocurarine was given to relax the spasm, an endotracheal tube was passed and vomitus poured from the tube. Suction was applied by catheter through the tube, and then the patient was inflated with oxygen. Pulse immediately improved, but the patient remained cyanosed. By this time the bronchoscope was available, and vomitus was removed from both main bronchi and from the openings of the segmental bronchi, under direct vision. The bronchoscope was maintained in position until the patient commenced to cough, when further vomitus was recovered from the lower lobe bronchi. During this time the obstetrician completed the delivery and repaired the episiotomy.

On the following day this patient's maximum temperature was 99.2° Fahr., and her chest was clear on physical examination. She complained of a little sore throat. Post-partum course was otherwise uneventful.

Case 2–Lobectomy–Anaesthesia Ether with Oxygen

The operation had been completed and tracheo-bronchial toilette and inspection of the stump had been done. (bronchoscopy). The patient's cough reflex had returned. Just as he was returned to bed this patient vomited a moderate volume of material containing solid food particles, aspirated the vomitus and became acutely anoxic and flaccid. He was returned promptly to the table, the bronchoscope re-inserted, and the tracheo-bronchial tree was thoroughly cleared under direct vision. His post-operative course was entirely uneventful.

Case 3

This patient reported at mid-morning as an out-patient for cystoscopy. It proved impossible to manage him with topical anaesthesia, and the anaesthetist on the Service was called. The patient was questioned directly concerning food both by the Urologist and the Anaesthetist, and told both that he had taken no breakfast. He was given a small quantity of pentothal, and promptly regurgitated a full breakfast, of which a good portion was aspirated. He became acutely anoxic and flaccid and the peripheral pulse was no longer palpable. Fortunately the anaesthetist carried with him a Magill bronchoscope, and proceeded immediately to clear the food from the trachea and bronchi, following which the lungs were inflated with oxygen. The patient recovered. His reason for lying about the food—he was afraid the anaesthetist would refuse to give him the anaesthetic if he told the truth!

(3) *Post-Operative*

Tracheo-bronchial toilet can in most cases be performed satisfactorily by blind suction through an endotracheal tube. There are some circumstances, however, where the greater certainty of removing all blood and secretion makes the use of the bronchoscope preferable. We feel that all patients who have had a resection of any part of the lung should be given the benefit of a bronchoscopic examination before leaving the table. In several instances where this has not been done, and in spite of thorough suction through the endotracheal tube, patients have been asphyxiated after leaving the operating room by the transfer of a blood clot or a large plug of muco-pus from a bronchus into the trachea. On one occasion examination of the bronchial stump after lobectomy found half of a bronchial ring protuding from the stump, and this was so readily detached with a Jackson's forcep that it was quite obvious that it would very shortly have been free in the bronchial tree as a foreign body.

The use of the bronchoscope to clear the respiratory tree at the end of operation is also frequently advisable in patients who develop atelectasis on the table during long operations. I have found this complication occurring most frequently in patients who have had long craniotomies in the lateral position. I have previously pointed out that some of these patients will require bronchoscopy before operation.

In the post-operative period bronchoscopic aspiration should be employed in all cases of pulmonary atelectasis which do not yield promptly to simpler methods. The persistence of the obstruction predisposes to acute infection, and undoubtedly in many cases to the development of bronchiectasis. The use of antibiotics is no substitute for the immediate relief of the bronchial obstruction.

Conclusion

I have endeavored to draw attention to the fact that the bronchoscope has become an essential instrument for the anaesthetist. I would suggest that the modern anaesthetist cannot be considered adequately qualified until he has acquired facility with this instrument.

A REPORT ON ANAESTHESIA IN A NON-UNIVERSITY CENTRE

J. N. KYLES, M.D.*

It is my privilege to bring greetings from my colleagues in the City of Hamilton. Our city, fifth in size in the Dominion, is the home of McMaster University, which has no Faculty of Medicine. This paper is a report of our endeavours in the practice of Anaesthesia in a centre of over 200,000 population, which is Canada's largest city lacking a Medical School.

The ancient Greeks had the Oracle at Delphi—we in Hamilton have London, Toronto and Buffalo nearby, with Kingston and Montreal not too far distant.

We have two fine modern hospitals with their associated Maternity divisions—St. Joseph's with 550 beds—the Hamilton General with 950 beds. Since both are open hospitals, many anaesthetics are administered by other than certificated anaesthetists. Last year's totals, for all forms of anaesthesia including blocks and locals were, at St. Joseph's, approximately 12,000; and at the Hamilton General, approximately 16,000.

Our group consists of 14 certificated anaesthetists all doing full-time Anaesthesia. Each of us is independent, for we have no group or associate practices, though four men restrict their work to St. Joseph's Hospital. Some are the products of Armed Services training with wide experience gained during wartime. The majority are general practitioners re-conditioned by post-graduate training in a variety of places.

Each month, as a Section of the Academy of Medicine, we have a Scientific session, following which our wives join us for a social hour. This meeting, plus the fact that, by common agreement, we take no "double-bookings" in our daily work, is a big factor in keeping us friendly and co-operative. Each year, too, we have a picnic when all our families get together.

By independent bookings in advance, we give general coverage to all units of both Hospitals. By regular schedule we have a system of emergency coverage, so that two of us are always available at both institutions. Also by regular schedule we maintain 24 hour coverage of the two Maternity units, where we are furnished with separate sleeping accommodation, bed-side phone and so forth. Four of us rotate at the Mountain Sanatorium three afternoons weekly; more will be said of this later. Occasionally we go to the Ontario Hospital to assist with the surgical care of Mental patients, and quite frequently there is an outing to nearby Grimsby or Oakville where there are small hospitals.

Our relationship with Surgeons and Obstetricians is most friendly and, we feel, mutually beneficial. None of them endeavours to dictate concerning our procedures or techniques, and for the most part all co-operate with us in regard to the emergency coverages mentioned above. In fact, the Anaesthetists run their own show.

For some time now we have presumed ourselves to be the only surviving exponents of Spinal (or lumbar) Anaesthesia. Despite what is read and heard to

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the contrary, we, from our local experience, see no indication for abandoning it. Post-operative headache—and backache—continue to be infrequent minor irritatations for all of us, and always shall. But, in the past five years there have been reported only two major complications—

(1) A Ward obstetrical patient developed Pneumoccal meningitis following a spinal anaesthetic administered by the Resident in Obstetrics. With Antibiotic therapy, recovery was complete.

(2) A not uncomely female Ward patient apparently failed to regain function of lower limbs, bladder and bowel following lumbar anaesthesia with 5 mgms. Pontocaine and glucose for Hemorrhoidectomy. (She did, however, shortly after return to bed from the O.R. require Morph S. gr. ¼ for relief of rectal pain.) Pampered by Hospital authorities, she was sent to Toronto neurologists, and to other consultants, at City expense. For two years she underwent routine enemata, bladder catheterization and physiotherapy and during this time of course, entered suit against both surgeon and anaesthetist who took counsel with the Canadian Medical Protective Association. Some 5–6 months ago she disappeared from bed and was found hiding in the Hospital basement. On return to bed she was mentally upset and required physical restraint. Bowel, bladder and locomotor system returned to normal.

It might be mentioned here that Continuous Spinal is now a rare procedure with us, although St. Joseph's was possibly the first Canadian Hospital to employ it in 1939. Light Nupercaine, too, has fallen by the wayside.

In all our institutions for the past few years our solutions for intra-thecal injection have been auto-claved with the spinal trays. No longer are they stored in antiseptic solution or handled with lifting forceps. To this chiefly we attribute our comparative freedom from sequelae. Other contributing factors are—

1. Almost exclusive use of Pontocaine-glucose solution.
2. Low spinal puncture and posturing.
3. Supplemental gas—O_2 for upper abdominal procedures, or Pentothal drip.
4. No fancy procedures, such as threading plastic catheters into spinal canal.
5. Surgical preparation of patient and anaesthetist.
6. Use of spinal anaesthesia is pretty well restricted to the more major abdominal procedures.

There are some exceptions to this last, since we do use spinal for

(*a*) Rectal surgery—hemorrhoids, fissures, fistulae—for Sphincter relaxation and jack-knife position.

(*b*) Cystoscopy and pyelogram—for co-operation of patient in securing satisfactory X-ray films.

(*c*) Obstetrics—generally for delivery of Prematures—also frequently for patients with full stomach or those markedly over-sedated.

On the whole, for Obstetrical Anaesthesia, Cyclo plus O_2 plus N_2O is the popular choice, about 7:2 as compared with spinal. However, of all the Obstetrical Anaesthetics given, somewhat more than ⅓ are open ether given by the Interne staffs. For Caesarean section, we employ in nearly every instance Spinal plus

Pentothal or gas O_2 supplement. In passing it might be stated that for some years prior to 1951, the Mountain division of the General Hospital had the most active Obstetrical unit in all Canada—average 5,029 cases a year over the past 5 years. In 1951, the Sisters of St. Joseph's opened their up-to-date Maternity Wing and deliveries are now pretty equally divided between the two places in proportion to the available beds.

The Mountain Sanatorium, mentioned earlier, is the largest in the British Empire, having 760 beds. Our Anaesthetic service there is a busy one, as Thoracoplasties are customarily three or four stage procedures. They tell us, there, that about ⅛ of all the Sanatorium patients come to operation under the present day treatment of Tuberculous disease. It is here that occasionally one gets involved with "double-booking" for lately, at a first stage Thoracoplasty three ribs may be removed in the one O.R., ground in a special gadget, then packed into the spinal column of a recipient lying prone in the O.R. next door.

For our Chest Surgery, induction is by Pentothal 5% (occasionally with added Curare), with maintenance by Cyclopropane via endotracheal tube). (Just offhand it strikes me that 5% Pentothal makes for readier intubation than the 2½% Pentothal, put up by the Dispensaries in 200 c.c. bottles.) We use packing but seldom and inflatable cuffs not at all. Procaine drip has been employed spasmodically in chest work but is not routine. It probably does no harm. There has been one case of Cardiac arrest reported from the Sanatorium with satisfactory response to Cardiac massage.

In both our Hospitals the main operating Rooms are of recent construction, with a Recovery room contained centrally within the suite. There is no need to enlarge upon the advantages of such a location. The chief disadvantage encountered is that trained personnel is on hand during regular day-time hours only. The Sanatorium, too has an excellent Recovery room.

The Canadian Red Cross for 2½ years has supplied the blood at all our institutions. Group O, Rh negative and Rh positive blood is always on hand. Blood for elective surgery is ordered beforehand. The service has many obvious advantages and certain disadvantages which need not be gone into here.

Since the General Hospital is a civic institution it is apparent that the overwhelming bulk of the Indigent and Welfare work is done on the Public Wards there. This fact, plus the fact that the Staff in Anaesthesia is approved by the Royal College, some time ago led to this Hospital being granted recognition for Training in Anaesthesia. Our Resident, under direct staff supervision is responsible for all Ward anaesthetics. He is assisted by Junior internes in their rotation. One drawback for him is the necessity of travelling twice weekly to Toronto for the lecture course leading to Certification.

Recently the Department of Anaesthesia at St. Josephs Hospital has obtained Royal College recognition. At the present time, in this Hospital some of the newer drugs such as Decamethonium, Tensillon and Cyclaine are being investigated by the Chief of the Department.

During the past 6 or 7 months the group as a whole and the Resident at the

General have been running a series of Epidural blocks using Xylocaine for a variety of procedures. Dr. R. M. Stringer gave a preliminary report at Sudbury in April, stating uniformly good results; the advantages in poor risk cases and so forth. This small series now totals about 100.

For the past three years two of our members in particular have been employing epidural anaesthesia in infant abdominal surgery, e.g. pyloric stenosis, atresia of bowel, inperforate anus, strangulated hernia etc., with marked success. Earlier 1–2% Novocaine was used: lately 2% Xylocaine with ephedrine, injecting 3½–6 c.c. depending on the size of the patient. Occasionally, but not usually light supplement of gas–oxygen or a trace of Pentothal is required. With the patient held in sitting position the drug is introduced through a short-bevel 20 gauge I.V. needle and reaches optimum effect in 10–15 minutes. Dr. F. G. Ruston lately reported on this work at Sudbury. Since then he has added, amongst others, a day old premature weighing 4 lbs. 7 oz. This case was recorded for posterity on Kodafilm by the demon photographer of our group. The total of this series in infants is now approximately 30 cases.

We do the usual run of nerve Blocks, as stellates, lumbar sympathetic, splanchnic and so forth on occasion. Xylocaine has been employed for some of these but no follow-up has been done in this regard. Paul de Kruif and the *Saturday Evening Post* think more of the stellates for cerebral afflictions than we do!

During the past year our E.N. and T. men have reported seeing an occasional granuloma of the cords where there is a history of earlier endotracheal intubation. Strangely enough, at the Sanatorium, where intubation is routine, the bronchoscopist, doing approximately 500 examinations a year, has not encountered this condition.

Locally, the fields of Neurosurgery and Cardiac surgery at present lie fallow, so in these we toil not, neither do we reap. Otherwise our work offers us a wide range of interest and much variety and from it we derive much satisfaction and pleasure. Through the arranging of our work amongst ourselves, as earlier outlined, we are enabled to derive from our leisure more satisfaction and pleasure than might otherwise be the case.

THE TEACHING OF ANAESTHESIA IN CANADA
PANEL DISCUSSION

Chairman—Dr. S. M. Campbell—University of Toronto.

Members—Dr. J. A. Blezzard—University of Western Ontario
Dr. E. A. Gain—University of Alberta
Dr. George Cousineau—University of Montreal
Dr. H. B. Graves—University of British Columbia
Dr. Fernando Hudon—Laval University
Dr. Donalda Huggins—University of Manitoba

The objects of the panel discussion were an attempt to assess the methods of teaching anaesthesia in the various medical schools in Canada, to bring out the ways in which teaching can be improved in each centre and by co-operation between the schools, having in view both the training of physicians for general practice and for the specialty of Anaesthesia. Unfortunately, Dalhousie, University of New Brunswick, McGill, Queens and the Universities of Ottawa and Saskatchewan were not represented. However, outlines of the courses given in McGill and Queens were made available for this discussion.

Undergraduate Teaching

It was disclosed that undergraduate teaching of anaesthesia varies from none in some of our schools to an amount comparable to that given in the other specialties in a few of our larger Universities. Opinions vary greatly, even amongst anaesthetists not only in Canada, but also in the United States as to the amount of teaching in anaesthesia which should be given the medical undergraduate. The chief difficulty seems to have been in getting sufficient time for lectures and demonstrations in a very crowded time table where the Departments of Surgery and Medicine and the other specialties each feels it is the most important subject on the curriculum.

The following is a brief outline of undergraduate teaching in some of our schools.

McGill—

(*a*) Demonstrations in the laboratories during the pre-clinical years of the pharmacological effects of some anaesthetic drugs.

(*b*) A series of 15 lectures illustrated by moving pictures and followed by discussion during the final year. A written examination is set.

(*c*) Each final year student spends one week on Anaesthesia in one hospital and two days in another hospital.

Toronto—

(*a*) A few laboratory demonstrations of the effect of drugs in the pre-clinical years.

(*b*) A series of eight lectures on anaesthesia during the second last year. This will be increased to sixteen lectures in 1953–4.

(*c*) In the final year each student is required to be present at six anaesthetic administrations and to attend six afternoon clinics on anaesthesia following which he takes an oral examination.

Laval–A series of lectures is given in the final year.

University of Montreal–The final year of the medical course is a rotating internship during which the student receives practical teaching in Anaesthesia.

Queens–

(*a*) In the pre-clinical years during laboratory demonstrations in Physiology and Pharmacology and Anatomy, special reference is put on anaesthesia where applicable.

(*b*) In the second last year a one hour lecture is given weekly on Anaesthesia.

University of Western Ontario–Lectures in Anaesthesia during the final year.

University of Manitoba–Lectures in Anaesthesia are given during the final year.

Universities of Saskatchewan, Alberta and British Columbia–have not yet given undergraduate teaching in Anaesthesia.

The conclusion to be drawn from above were:–

1. Teaching of the Basic Sciences with reference to Anaesthesia is not receiving the attention it should.
2. The teaching of Anaesthesia to the undergraduate in some schools is insufficient to prepare him for hospital interneship. Some will no doubt question the value of this suggestion.

Graduate Training

This varies even more than undergraduate training but there is a very excellent increase in the number of centres where training is provided for qualifying candidates for Certification and Fellowship in the Royal College.

The McGill three year course consists of two years senior interneship in one of the Montreal hospitals under the teaching staff of McGill University during which lectures are given in the basic sciences, medicine and anaesthesia and weekly seminars are held. The third year is spent as a junior staff member in a hospital either in Montreal or as arranged by the chief in another hospital, usually in Canada. In addition there is an exchange arranged for short periods in Hartford, Conn.

The post graduate course in Toronto consists of two years in Toronto hospitals with basic science, medicine and anaesthesia lectures and seminars, somewhat similar to the McGill course. A third year in anaesthesia is obtained by means of Fellowships either in Toronto or in the British Isles or by a junior staff appointment. Residencies in anaesthesia to cover a third year are being established in several of the University of Toronto teaching hospitals. Arrangements are also made whereby a graduate going on to Fellowship examinations may take a year in medicine or a basic science before entering on a senior interneship in Anaesthesia.

In addition General practitioners from time to time are given instruction for periods of 2 weeks to a month in practical anaesthesia.

The University of Montreal provides a two year course in basic sciences followed by two years in anaesthesia. A third year must be obtained at some other centre.

Laval in addition to the two year course provides a year in basic sciences and a residency in anaesthesia leading to the degree of Master of Anaesthesia.

At Queens and Western the post graduate courses are being developed to extend beyond one year senior interneship.

At the University of Manitoba we are given to understand that senior internships in anaesthesia were not yet available, but junior staff appointments on salary are available.

In the University of Alberta the junior interne receives 2 months on anaesthesia. Following a second year taken in Medicine or Surgery he may then take a residency for one or two years in Anaesthesia during which he spends 2 afternoons a week in the laboratory in Physiology and Pharmacology. It is then necessary for him to take another year in anaesthesia at another centre before being eligible for Royal College Exams.

In the University of British Columbia, 6 residencies are available leading to Fellowship Examinations. Dr. Digby Leigh has his own animal laboratory for teaching and research purposes in addition to the teaching in basic sciences by other departments of the University. The Department of Anaesthesia also conducts a one month course for General Practitioners for a fee of $50.00.

On the Atlantic coast at Dalhousie University a strong Department of Anaesthesia under Professor C. C. Stoddard has been organized and post graduate training leading to certification and Fellowship is under way for a limited number of candidates.

In practically all the University hospitals junior internes serve one or two months on anaesthesia. This was stressed by some of the representatives as part of the training for men and women going out to general practice.

In some of the smaller universities there has been considerable difficulty in arranging sufficient basic science lectures and demonstrations by other departments of the University. This is partly due to shortage of staff, partly to lack of funds and also to the small number of post graduate students.

In the smaller teaching centres there are very few senior interneships, let alone residencies, available in hospitals affiliated with the University. This makes it very difficult to organize classes and teaching time tables. Until these universities gain sufficiently in size and their affiliated hospitals have bed capacity to provide enough senior interneships in anaesthesia, the obvious remedy is for the larger centres to accept post graduate students into their courses who have completed one year of their training in the smaller centres.

Remuneration for senior internes varies considerably across the country. In some centres the internes administer anaesthetics to none but public ward non-paying patients. In others, especially where the majority of patients come under a prepaid scheme or a Provincial Health scheme the interne gives anaesthetics

to patients for whom a fee is paid and in this latter situation, whether it is strictly ethical or not, the interne usually receives a larger monthly allowance. However for teaching purposes, it would seem to be a better situation where the interne is limited to the non-paying case. We understand that the Royal College in assessing a hospital for recognition as an acceptable one for post graduate training takes into account the number of public non-paying beds available for teaching purposes. This will certainly limit the number of hospitals put on the approved list and with the increase in prepaid and semi socialised medicine some allowance may have to be made for changing conditions.

Certainly with the rapid growth of this country in industrialisation and population many more trained anaesthetists are required for the new and enlarged hospitals and the medical schools must keep ahead of the situation in providing the necessary training programme. In doing this they must not forget that the general practitioner also requires a knowledge of anaesthesia and provision must be made for his training.

SERVEL OR FEEDBACK CONTROLS IN ANAESTHESIA

H. V. Rice, M.D.*

The purpose of this paper is to review the possible application of recent developments in the field of physiology and related branches of science to anaesthesiology. It is in the nature of a hypothetical proposal, or what might be called a far-fetched prediction. It is intended, not to review in detail the procedures which have been developed, but rather to touch upon the procedures themselves, and to indicate how they might be applied to the field of anaesthesia.

The basic techniques of the art and science of anaesthesiology have changed little in fundamental respects since anaesthetics were first administered. There have, of course, been numerous refinements in the efficiency of giving anaesthetics, but these have been relatively minor improvements in a scientific sense and have involved largely an improvement in the technique of the individuals using them and of the anaesthetic agents, and to a lesser extent in the apparatus used. The administration of an anaesthetic still depends largely upon the perception, awareness, experience, and judgment of the anaesthetist who uses his natural senses to detect changes which must, to be thus detected, be relatively gross. Critical analysis of the biological state of the patient is necessarily limited. Functional or physiological analysis is very incomplete during the course of a routine anaesthetic. One might almost say that functional analysis under the conditions of the operating room is almost as incomplete as was morphological analysis prior to the introduction of the microscope.

In the course of many anaesthetics, questions arise which cannot be answered relative to the condition and progress of the patient. There is frequently an inability to predict accurately the course of events both during and after the anaesthetic. It would appear that in order to obtain information that is necessary to eliminate these questions it is necessary to develop procedures which indicate more accurately what is going on. What is needed in short is a physiological microscope.

The first part of this paper, therefore, deals with the possible means by which a more accurate indication of the actual state of the patient both during and after the anaesthetic may be obtained. The second part will stem from this and will deal with the way in which such detection devices may be linked to control mechanisms which will automatically assist in the maintenance of the best state of the patient.

It is true that anaesthesia as routinely conducted is amazingly efficient and successful in spite of the paucity of information available to guide the anaesthetist as he conducts his work. But the administration of an anaesthetic is not yet perfect, or free from unexpected danger. There are two reasons for this. The first is the fallibility of the anaesthetist, who may not be able, with the senses provided by nature, to detect changes which are actually starting. There is also

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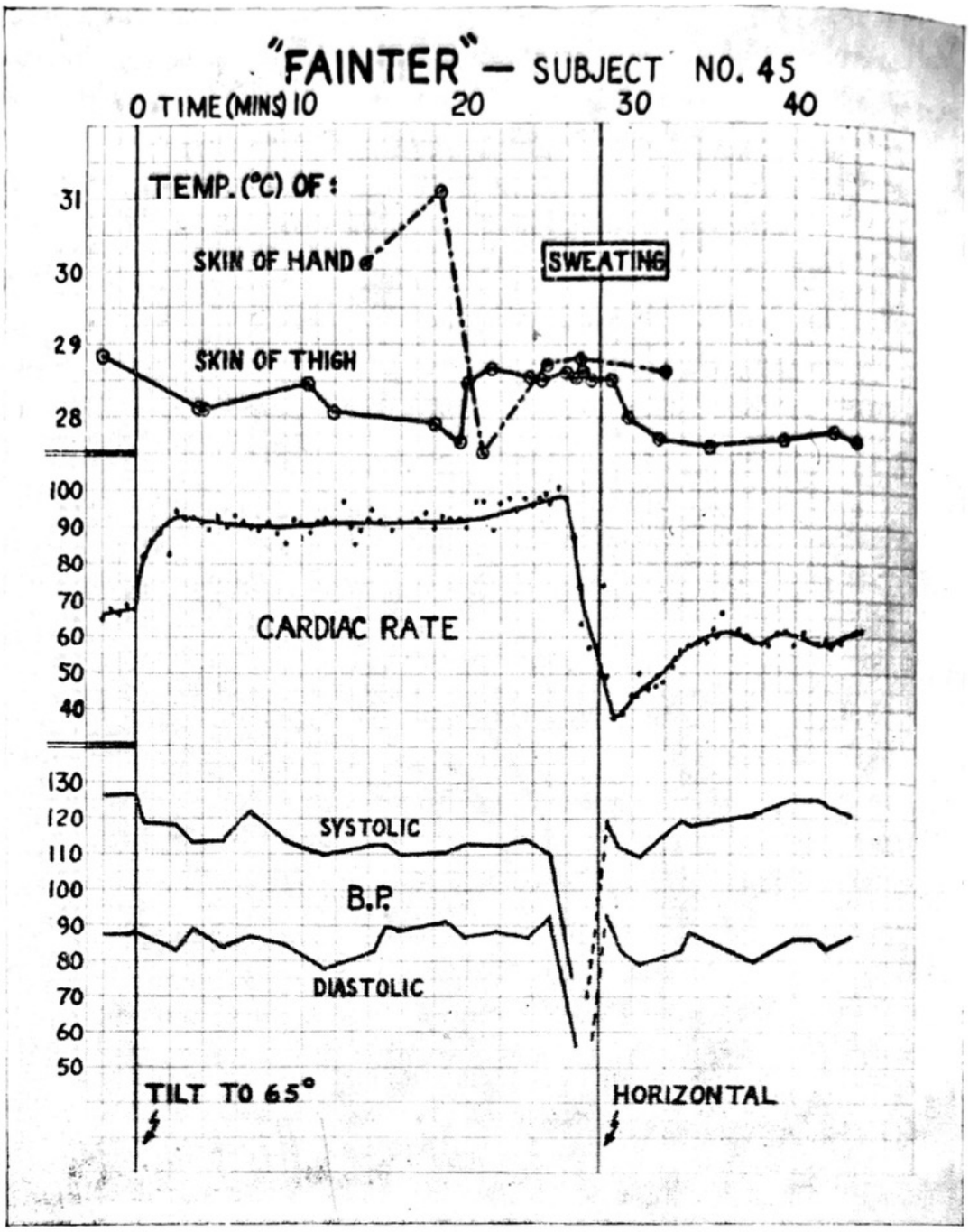
the factor of momentary lapses of attention at a critical moment. And finally there is the variation of ability of the anaesthetist, which is referred to usually as his skill and which may in some instances be of a much higher order than in other instances though each may be considered adequate.

Anaesthesia, having developed to the stage where it is a very highly developed art and a very efficiently conducted one (although not perfect), is in the stage where, to attain further progress in improvement, a degree of development out of all proportion to that which was necessary in the early stages of the art is involved. Improvements over the best results now obtainable will require the determination of physiological changes which have up to date been obscure. With recent developments, however, they have become observable through the use of the appropriate apparatus. It is the use of this apparatus, already established as necessary in research laboratories, and which has been applied in other fields of applied medicine such as aeronautics, with which this paper is concerned.

The first proposition thus deals with the possible methods for more critical detection of both normal and abnormal function during the course of an anaesthetic. The research worker in many fields is fully aware that our senses are in many instances ineffectual in determining variations of function. On the other hand, such variations can be made evident to us through the use of electronic devices which have greater sensitivity than we naturally possess. They are capable of revealing a variety of physical and even chemical phenomena. Any one who has worked on animals will realize that simple observations of gross function are indeed inadequate and sometimes fallacious. For example, blood pressure changes are frequently used as an index of a change of function. But this is a poor index or at best a late one. An illustration of this is shown in the accompanying figure. This is a graphic representation of information obtained from a normal individual who was being studied in connection with the development of postural syncope.

The background of the experiment illustrated is as follows. The patient was first lying horizontally on a comfortable bed with apparatus attached to him to permit the detection in detail of various bodily functions. These included changes in the distribution of the circulating blood through the use of leg plethysmographs. In addition continual records of the electrocardiograph and the electroencephalograph were made. Blood pressure readings were taken every minute or two and frequently repeated readings of skin temperature were taken on some ten or more parts of the body including the fingers, the skin of the arm, body, face, leg, etc. After a basic period of control with the patient horizontal had been established he was then tilted to a near vertical position and the changes in all the observable functions were followed. The patient was allowed to remain in this vertical position until a fainting reaction developed. At this time the patient was returned to a horizontal position and the subsequent course of events was followed still further.

The composite findings shown on the graph prove that while the patient



Study of Postural Syncope in normal individual held passively at 65°. Vasomotor changes in hand and leg precede fainting response by 8 minutes.

ultimately did show a fainting response and lost consciousness, and while this state was preceded for a minute or two by a sense of constriction in the chest, nausea, and considerable sweating, significant blood pressure and pulse rate changes did not occur until the actual development of the fainting reaction. The most significant point of all is that some six to eight minutes prior to the onset of any other changes there were recorded from the skin of the hand and the thigh significant changes in skin temperature, indicative of a prodromal vasomotor change which preceded the actual fainting reaction. There was a sudden constriction of the blood vessels of the fingers resulting in a reduction of temperature in this area associated at approximately the same time with a dilation of the vascular tree of the leg as indicated by a rise of temperature. It would appear that under conditions in which a subsequent development of a failing circula-

tion were to occur one might have early evidence of it if there were attached to the patient adequate devices for showing the vasomotor changes which might precede this circulatory collapse.

This experiment is cited in detail to illustrate how unreliable are the gross changes upon which we ordinarily rely to observe the state of an individual during various stressful conditions. The gross changes are actually a poor index, or at least a late one of the true state of the individual. This is due to the fact that there are so many compensatory adjustments at work in the body which maintain blood pressure and other bodily functions. They tend to mask the primary disturbances. There can thus in fact be a wide fundamental disturbance either of vasomotor control or of cardiac action or of respiratory action prior to the development of obvious disturbances of function.

In recent years, new physiological techniques, based largely on physical apparatus of new types, have been developed which permit the detection of changes which are otherwise not detectable. For example, the development of the photocell has permitted the successful use of the oxymeter during the course of anaesthesia and experimental work to determine the state of oxygenation of the blood. These have been employed with particular success in the field of aviation medicine. Efforts have also been made recently to apply knowledge of the brain wave patterns to control the depth of anaesthesia. The changes in the electroencephalogram that occur during other types of interference with cerebral metabolism have previously been followed in great detail but it is only recently that a practical use of the changes occurring in the brain during the course of an anaesthetic have been applied to the field of anaesthesiology.

During the war a new type of gas analyzer was made available which permits automatic and instantaneous determination of the gas mixtures which the patient is breathing including such mixtures as nitrous oxide, oxygen and carbon dioxide. With the use of such apparatus one should be able to adjust immediately the level of anaesthesia, and detect changes in metabolism, or technical procedure which disturb CO_2 exchange.

Electrocardiography has in recent years become a much more practical procedure than previously owing to the development of instantaneous-recording apparatus. The electrocardiogram will not provide all information regarding cardiac function, but it does indicate certain changes which are not evident through pulse or blood pressure observations, sometimes well in advance of the development of a serious cardiac deficiency. It would appear that the use of the electrocardiogram in the operating room by the anaesthetist might offer early evidence of cardiac changes which might later be serious if not detected at that time. The same arguments may be applied to the cardiostethophone, a device which permits the continual recording or hearing of heart sounds without the requirement of putting the manual stethoscope to the patient's chest. Recent use has been made of this in following the course of the fetal heart sounds during labor.

In experimental work on animals continuous recording of respiratory move-

ments is a routine requirement in laboratories where respiratory activity is being observed. While it is quite true that the gross respiratory movements of a patient are evident in the operating room from the movements of the patient or the anaesthetic bag, the true respiratory activity is certainly not capable of sharp analysis by such observation. Without an accurate record of respiration, detailed assessment of respiratory function, and the indications which suggest later respiratory distress, are not possible.

These are illustrations of types of observation of specific function which can already be made through the application of special apparatus which is now available. My first proposition therefore is that continuing improvement in anaesthesiology will depend upon the application of such techniques, which involves, of course, the training of the anaesthesiologist in their theory and use.

Once methods have been devised for the practical application of these methods of physiological observation it is only a brief step further to link them with control devices which will maintain the normal situation as nearly as possible, or automatically correct any abnormal developments which may occur. This is what is meant by servel or feedback mechanisms. Such apparatus is already in use in the research laboratory. Whenever the normal ranges of function might be exceeded the detection systems would institute two types of response. The first would be the activation of warning devices to advise the anaesthetist that something was wrong, and frequently to tell him *what* is wrong. The second would be to cause appropriate adjustments, through servel control systems, to correct the abnormal state. For example, the control valve of an anaesthetic gas machine would be automatically adjusted to modify the flow of anaesthetic gas or oxygen so that a potentially dangerous situation would be corrected by the detector-servel system.

The description of such proposals in general terms as outlined above sounds complicated. In actual fact some of the apparatus is relatively complicated. But the use of it is relatively simple. The simplification of its use is largely a matter of design and standardization. Refinements of design and technique in such scientific developments spell the difference between success and failure in their use.

An important point with respect to such applications in the field of anaesthesiology is that the successful use of this apparatus will hinge upon the knowledge of the principles on which it works, possessed by the person who uses it. Practically all of these developments are electronic in nature. That is, they employ radio tubes in one form or other. The use and design of the apparatus therefore entails a fundamental knowledge of electronics, or difficulties will arise which cannot be interpreted or corrected. Most of such apparatus appears impractical and complicated to the untrained and inexperienced. But to the person with adequate training and knowledge of its operation, such apparatus is actually simple to use. The implications of this statement to the training of anaesthesiologists is obvious.

There are other electronic developments which justify these proposals, and

would eliminate many of the objections that might be raised. To those who, with the tradition of the art of medicine in mind, say that we cannot duplicate the efficiency of a competent and alert human, there is much evidence that in well engineered electronic systems, technical equipment is less fallible; and is capable of much more critical analysis and synthesis than are the human mind and body. Further, by the use of such devices as limit controls, and time-delay systems, detection and servel apparatus can be set to come into operation only when required. Limits of tolerance can be pre-set, so that the robot goes into action only when these limits are exceeded. Thus with the cardiotachomoter, (a device which records continually the interval between heart beats, and thus the instantaneous heart rate), a time-delay relay would prevent an occasional missed beat from activating the servel controls, whereas a more prolonged cardiac slowing or arrest would call them into play. Similarly a limit control might be set at a cardiac rate of 120 per minute, if rates below this were considered permissible. Below this rate, the limit control would prevent any intervention by the apparatus. But above this rate the control would activate whatever mechanism it was connected with.

These developments would, of course, be impossible with the present arrangements of operating rooms. To pile more equipment into an O.R. is not feasible. They foretell, therefore, the re-organization of operating rooms to suit the scientific planning. There may be a bank of operating rooms with a control room into which the recording devices are channelled. Here the anaesthetists work. A panel corresponding with each operating table will be before the operator. On this panel will be the appropriate signals indicating the progress of the anaesthetic and the state of the individual. Further, there will be automatic servel controls linked to these panels so that not only may an anaesthetist be immediately dispatched to the appropriate O.R. at the first sign of danger, but adjustments will automatically be made even before the observer is aware of the danger or could himself correct it. Such apparatus will be less bulky and less cumbersome than the present anaesthetic accountrements in the O.R.

If this prediction is true, anaesthesiology will soon find itself confronted with a revolution of ideas and organization. This revolution will conceivably and even probably involve the following stages.

(1) A change in the nature of training of anaesthesiologists, with greater emphasis on the basic sciences, especially physiology and biochemistry; and more important, with the institution of training in physics, particularly electronics.

(2) Much greater emphasis on research, especially of team research between medically trained anaesthetists and physicists and others.

(3) Reorganization of the operating room to permit the use of future techniques of anaesthesia, which will embroil you more deeply even than now, in the problems of hospital organization and economics.

The first reaction amongst medical men with whom I have discussed these ideas has been an assumption that many anaesthetists will be put out of a job and that anaesthesiology will become mechanical. The converse is, of course,

the real truth. What will be required is that anaesthesiologists be trained first to develop and then to handle the truly scientific apparatus. Rather than the anaesthesiologists finding less work to do and less requirement of true scientific insight, it is obvious to me that he will find himself a more complete scientist and using scientific methods with more thoroughness and with more accuracy A given anaesthetic staff will be able to do more work with greater safety.

The proposals outlined above therefore involve a considerable change of view point, and a considerable change in the training, of anaesthesiologists, and probably will cause a reorganization of the operating room. In other fields parallel developments have already occurred and have proven their worth, for example in aviation. They cannot fail to develop in our own science. They will increase the scope of work and increase the safety in doing it. These are the essentials of progress. The biggest step in their attainment is the recognition of their probability. The concept is father to the attainment. This is the phase with which this paper has been concerned.

SOMATIC ABDOMINAL PAIN

F. A. WALTON, M.D.*

Although intractable pain anywhere in the body is rapidly becoming the province of the modern anaesthetist, by virtue of his special knowledge and training, it has been necessary in the interests of time and space to confine this address to somatic abdominal pain. (A previous communication, "Coeliac Ganglion Syndrome," (1) dealt with abdominal pain of visceral origin). However, many of the concepts expressed here apply with equal force to the other parietes of the body with the possible exception of the area supplied by the fifth cranial nerve. An important consideration in choosing the abdomen for this presentation was the growing realization of just how common somatic abdominal pain is and how often it is mistaken for visceral disease, both by the doctor and the patient. This often leads to a long series of expensive investigations and futile laparotomies; the patient going from doctor to doctor and sometimes ending up with a severe psychoneurosis, drug addiction, or in the hands of irregular practitioners. Multiple hospital admissions and ineffectual operations are not uncommon in these cases, because of the amelioration of symptoms that not infrequently occurs post-operatively. This amelioration is only temporary however, in most cases, and is due to the muscular relaxation conferred by the anaesthetic, and the interruption of the nervous reflex arcs if a regional anaesthetic, such as a spinal, is given. It is also enhanced by the bed rest, the sedation, and the lack of domestic harassment when in hospital, as well as by the conviction that "something is being done at last." The patient is also helped by being reassured that nothing serious, such as cancer, had been found.

Somatic abdominal pain is very commonly caused by a distortion of the musculo-skeletal system, and this is not surprising when one considers that by assuming the erect posture mankind has placed the whole weight of the body on the two caudal extremities, thus depriving the spinal column of the support of the cephalad extremities and necessitating the development of powerful paraspinal muscles along with an improved coördination of balancing power to oppose the ever-acting force of gravity. Again, the erect posture has made it necessary, for health, for the abdominal musculature to be of sufficient strength and tone to support the viscera, which otherwise will sag caudally and drag on their attachments with ensuing dysfunction, pain, and disability of varying degree. Radiological studies in the erect posture (2) have shown that the stomach can rise a full six inches, the transverse colon three inches and the gall bladder four or five inches by improving the body posture and especially the abdominal muscle tone. The kidneys, small intestine and diaphragm can also be elevated. It is apparent also that any increase in the normal slight antero-posterior curvatures of the spine and/or any scoliosis must increase the normal constant strain and effort of maintaining the erect posture, as these deviations in greater or lesser degree, remove the spine from the perpendicular, which position is, of course, best suited to withstand the action of gravity. Also, a consideration of the

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nerve supply of the abdominal wall, thoracic VI to lumbar I, inclusive, sixteen nerves in all, each of which has a long course and similar anatomical relations makes evident the many possibilities for abdominal somatic pain of neuritic origin. The commonest site for the nerves to be afflicted is where they are enlarged by the posterior root ganglia, as they lie in the intervertebral foramina. Any deviation of the vertebrae (e.g., scoliosis, lordosis or kyphosis) one on the other can narrow these foramina to some extent, leading *in some individuals* to irritation of the nerves and ganglia. This will cause pain in the distribution of the nerves involved which in turn will reflexly set up muscle spasm on the affected side, which in turn increases the deformity and the pain so that a vicious circle of pain–muscle spasm–pain is set up, which can, and often does, spread to neighboring segments. This vicious circle is compounded by sympathetic nerve reflexes (afferent and efferent sympathetic fibres travel in the nerve) which cause arteriolar spasm, which gives rise to hypoxia of the nerves and ganglia resulting in increased capillary permeability and oedema. This, in turn, further aggravates the discrepancy between the nerves and their foramina. The involvement of the sympathetic nerves (afferent and efferent) which supply the viscera, also explains the common visceral symptoms from which these patients suffer, and also the response to treatment by block therapy (vide infra).

A consideration of the above factors gives an understanding of the many somatic pain syndromes that are encountered. The commonest clinical type encountered here is postural in origin and is most frequent in women, especially after childbirth (vide infra). The patient often has a lumbar lordosis with a compensatory thoracic kyphosis, with or without some scoliosis.

The next commonest type is also postural in origin and is due to unequal leg length. This unequal leg length inevitably produces some scoliosis. The difference may be only 3/16″ or as much as 1–1/2″. Even the 3/16″ difference can give rise to symptoms. Special x-ray technique (2) is necessary to accurately measure these small differences in leg lengths. A not uncommon cause, also, is acute trauma involving either a direct blow paravertebrally, or sudden marked flexion or twisting (Case No. 8). Another not uncommon cause is old healed empyemata (Cases No. 1 and No. 23). Here the scoliosis is secondary to pulmonary or pleural fibrosis or to chronic muscle spasm on the affected side. Obesity per se may also initiate or aggravate the syndrome (Cases No. 2 and No. 9). Yet another cause is hysteria associated with severe spasm of the spinal muscles (Cases No. 6 and No. 7). Some cases are encountered that do not reveal any adequate overt cause despite careful investigation. (Case No. 16). One case (No. 21) only was due to "fibrositis" in the abdominal wall and responded to local procaine injections. Most cases are precipitated or aggravated by worry, overwork, fatigue and occasionally by an intercurrent respiratory infection.

Other well known causes of somatic pain include herpes zoster, tabes dorsalis, new growths in the spine or its vicinity (Case No. 22), spinal caries, prolapsed nucleus pulposus, spinal osteoarthritis and acute pleural affections.*

*It seems quite likely that the various pains suffered by tabetics are due to the inflammation and oedema of the nerve roots and ganglia where they lie in the intervertebral

The syndrome resulting from nerve root irritation is now termed "segmental neuralgia" (2) although in the past it has been called "radiculitis" or "intercostal neuralgia", if and when diagnosed. It consists of a painful tender area confined to one or more dermatomes. The whole dermatome is not always affected, the area involved depending on the location and extent of the nerve fibres involved. The pain varies from acute and stabbing or crampy in nature, to a dull ache or dragging or sometimes a burning sensation. The tenderness, which incidentally cannot be feigned, consists of hyperaesthesia of the skin to light pinching or pin-prick in most cases, but may only be elicited by poking or pressing firmly on the skin against the underlying muscle or bone. In some of the cases the tenderness was more marked when the patient's abdominal muscles were relaxed, probably indicating involvement of the nerve fibres supplying the deeper structures of the abdominal wall. The side affected is usually on the side of the concavity of the spinal column, but not always. The skin temperature is often decreased in the affected areas.

Clinical

Twenty-three cases of abdominal somatic pain (Table I) examined during the past year are summarized. These cases are unselected and consecutive, only two cases being omitted, one in whom the original diagnosis of segmental neuralgia was untenable, and, in fact, is still undiagnosed, and another recent case that is still under treatment. Many other cases were seen en passant as it were during the past year, but were not followed for one reason or another. It will be noticed that the females outnumber the males by twenty to three, and that two of the males were secondary to empyemata (Cases No. 1 and No. 23), the third male (Case No. 8), a boy of eleven years being due to trauma. Only the salient features of these cases are mentioned in the table. It was noted that many cases had abdominal distention and/or a feeling of bloating, at times, which was often confined to the painful quadrant of the abdomen but was occasionally generalized. Also, vomiting was severe and distressing in practically all the upper abdominal cases, but despite this their hydration and general condition remained fairly good in most cases. Four of the upper abdominal cases had had laparotomies with generally negative findings and little relief. One case (No. 2) in which the x-rays had revealed a persistent deformity of the pyloric antrum, tentatively diagnosed as gastritis, at operation, revealed a thickened slightly

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foramina. The girdle pains are certainly explicable on this basis and the vomiting and possibly some of the pain of "gastric crises" can be explained by the involvement of the sympathetic efferent and afferent nerves where they pass through the intervertebral foramina in the spinal nerves. The "lightning" pains that occur later in the disease could be accounted for by the pain impulses coming from the inflamed and oedematous nerve root being "blocked" by virtue of the destruction of many cells and fibres by the disease and only the most intense stimuli getting through. This interpretation (oedema of ganglia and nerve roots) of the pains of tabes is confirmed by the dramatic response that these patients show to I. V. procaine. The author had the opportunity to examine the posterior root ganglia of a tabetic recently. They were grossly enlarged and microscopic examination showed oedema, lymphocyte infiltration and nerve cell degeneration.

Case	Age & Sex	Diagnosis Provisional	Diagnosis Final	Etiology	Symptoms	Signs	Treatment	Result
1	M 35	Duodenal ulcer	Segmental neuralgia	Healed empyema Kypho-scoliosis	Recurrent attacks epigastric pain and vomiting for 18 yrs.	Kypho-scoliosis. Tender area left epigastrium. Tender paravertebrally T VI to T XI. X-ray "duodenum irreg. (2nd part)"	I.V. Procaine X 7 Postural exercises	"Cured"
2	F 33	Cholecystitis	Segmental neuralgia	Obesity ?	Recurrent attacks pain right upper quadrant and back vomiting †††	Hyperaesthesia ††. Right upper quadrant and right chest posteriorly X-ray "antral gastritis"	Laparotomy, biopsy pylorus	Improved
3	F 58	Pancreatitis	Segmental neuralgia	Kypho-scoliosis Short leg—⅜"	Recurrent pain right upper quadrant. Vomiting †† —4 yrs.	Tender T IX paravertebrally. Osteoarthritis spine	I.V. Procaine X 5 Heel lift Postural exercises	Improved
4	F 19	Pancreatitis	Segmental neuralgia	Child birth (2nd) Lordosis	Pain upper abdomen †††. Vomiting for 6 months. Abdominal distention	Hyperaesthesia whole right upper abdomen and back. Lordosis X-ray "Delay barium upper small bowel"	Paravertebral Block X 1 I.V. Procaine X 5 Postural exercises	"Cured"
5	F 63	Anorexia nervosa	Segmental neuralgia	Short leg 1" ? other causes	Pain left upper quadrant †††† 2 yrs. Vomiting ††† 2 yrs. Constipation †† Distention † Difficult to void	Hyperaesthesia ††† left upper quadrant also left chest posteriorly. Emaciated 65 lbs. Nervous wreck	I.V. Procaine X 36 Paravertebral Block X 4 I.V. Xylocaine X 4 Splanchnic Block X 3 Subarachnoid Alcohol X 4 Lumbar sympathetic Blk. X 1 (alcohol)	Improved
6	F 20	Renal colic	Segmental neuralgia. Hysteria	Hysterical muscle spasm	Pain left upper quadrant 6 days. Vomiting 7 days	Hyperaesthesia T IX abdominally and para-vertebrally. Hysterical	I.V. Procaine X 3 Psychotherapy	Improved
7	F 18	?	Segmental neuralgia	Hysterical muscle spasm	Pain left chest and whole abdomen, very severe. Hurts to breath	Marked hyperaesthesia T VIII to L I (left) T VIII to T XII (right) Tender paravertebrally in these segments also.	Bed rest E.U.A. Change home environment.	"Cured"
8	M 11	Acute appendicitis	Segmental neuralgia	Trauma	Pain right lower quadrant recurrent 3 times in 6 months. Nausea slight.	Hyperaesthesia right lower quadrant. Tender T XII paravertebrally	Proctocaine T XII and L I Paravertebrally X 1	"Cured"
9	F 14	Cystic ovary	Segmental neuralgia	Obesity Postural	Pain right lower quadrant recurrent 2yrs.	Hyperaesthesia right lower quadrant	Laparotomy	Improved
10	F 7	Acute appendicitis	Segmental neuralgia	?	Pain right lower quadrant	Hyperaesthesia right lower quadrant and lumbar region	Appendectomy right. "Terminal small bowel (2"). Swollen and doughy" Appendix "slight lymphoid hyper-plasia"	
11	F 12	Acute appendicitis	Segmental neuralgia	?	Recurrent attacks of pain right lower quadrant for 2 years. Occasional nausea	Hyperaesthesia right lower quadrant. No tenderness in back W.B.C. 9,600	Appendectomy Appendix normal *"Terminal 3" of ilium edematous"*	*Improved*

Case	Age & Sex	Diagnosis Provisional	Diagnosis Final	Etiology	Symptoms	Signs	Treatment	Result
12	F 19	Pelvic inflammatory disease	Psoas muscle strain	Lordosis and scoliosis	Almost constant dull ache right lower quadrant for 4 years.	Lordosis and scoliosis	I.V. Procaine X 4 Postural exercises	"Cured"
13	F 25	Segmental neuralgia	Segmental neuralgia	Obesity, posture (lordosis) child birth	Pain both lower quadrants for 4 yrs. (since birth of child)	Hyperaesthesia T IX X II and L I Lordosis	Paravertebral Block X 1 I.V. Procaine X 3 Postural exercises	Improved †
14	F 66	Diverticulitis	Segmental neuralgic diverticulosis	Child birth Lordosis	Pain left lower quadrant 40 yrs. Pain upper abdomen 18 mos. Nausea 18 mos. Diarrhoea recurrent	Hyperaesthesia (slight) left lower quadrant	I.V. Procaine X 5 Paravertebral Block Proctocaine X 1	"Cured" of pain. Diarrhoea recurs at times
15	F 35	Hydronephrosis	Hydronephrosis segmental neuralgia	Trauma Lordosis Neurotic	Pain right lower quadrant 6 months following plastic operation on kidney	Hyperaesthesia right lower quadrant and lumbar regions Lordosis ††	Postural exercises I.V. Xylocaine X 1. I.V. Procaine X 6 Caudal X 1 Paravertebral proctocaine X 1	Improved
16	F 43	?	Psychoneurosis Segmental neuralgia	Child birth Psychoneurosis	Pain right lower quadrant and left lower quadrant 20 yrs. Pain upper abdomen 7 yrs. Abdominal distention	Tender practically whole abdomen— No hyperaesthesia	I.V. Xylocaine X 1 I.V. Procaine X 5 Paravertebral Block X 1	Improved †
17	F 35	Spastic colon	Segmental neuralgia	"Trigger area" buttock	Pain left lower quadrant 2 yrs. slightly	Slightly tender left lower quadrant. No hyperaesthesia. Also a tender spot in left buttock	Paravertebral Block X 1 Inject trigger area with proctocaine X 1	"Cured"
18	F 27	N.Y.D.	Psoas Muscle strain	Childbirth and scoliosis	Pain left lower quadrant 1 yr.	Tender posterior abdominal wall anteriorly in region L 3–4 vertebrae	I.V. Procaine X 3 Postural exercises	No change
19	F 42	N.Y.D. Hydronephrosis	Segmental neuralgia. Hydronephrosis	Short leg. ? other cause.	Pain right lower quadrant and loin 5 yrs. Pain left lower quadrant 6 months. Large bowel dysfunction	Tender whole abdomen but especially both lower quadrants. Tender T XII paravertebrally	I.V. Procaine X 6 Paravertebral Block X 1 I.V. Xylocaine X 2 Lumbar sympathetic	Pain less Bowel habits now normal
20	F 30	Pelvic inflammatory disease	Segmental neuralgia	Childbirth Lordosis	Pain lower abdomen 11 years	Tender left lower quadrant. Tender left buttock	I.V. Procaine X 2 Bed rest	"Cured"
21	F 21	Appendicitis	"Fibrositis"	?	Pain and tenderness right lower quadrant. Worse on movement. Vomiting at times	Tender spot just medial to McBurney's point	Local Procaine X 5	"Cured"
22	F 19	Hepatitis	Intrathoracic sarcoma	?	Recurrent pain right upper quadrant.		Paravertebral Block X 1 I.V. Procaine X 3	No benefit Died
23	M 45	Acute appendicitis	Healed empyema Segmental neuralgia	Healed empyema Congenital spastic paralysis	Pain right lower quadrant	Tenderness and hyperaesthesia (†††) right lower quadrant and right loin and chest posteriorly	Bed rest	Improved

"Cured" indicates that the patient is leading a normal life and is not having any pain worth mentioning at present.

oedematous pyloric antrum. This was biopsied and reported to show "diffuse plasma cell and some lymphocyte infiltration in the mucosa, separating the gland spaces. There is hyperplasia of lymphoid follicles in a deeper portion of the mucous membrane. The submucosa shows moderate infiltration by eosinophils and lymphocytes". These x-ray and pathological findings could be explained on the basis of persistent sympathetic nerve impulses, from the irritated nerves in the intervertebral formina, producing dyskinesia, and chronic vasospasm of the vessels supplying the bowel with ensuing oedema. Certainly no other explanation accounts for the otherwise mysterious syndrome. Cases No. 1 and No. 4 also had x-ray findings that are otherwise inexplicable (Table I). Case No. 1 had an exploratory laparotomy at another centre with negative findings.

The lower abdominal cases very frequently suffered from constipation and/or diarrhoea and/or pain with defaecation. One of these cases was diagnosed as spastic colon and it seems likely that similar cases have been dubbed "irritable colon", or "spastic colon" in the past. They also often have urinary disturbances, such as, dysuria, frequency, etc. Two cases (one not included in the table) had had operations performed for hydronephrosis and another case is about to have an operation for the same condition. Two of these cases now have pain on the opposite side, for which they are being treated. It seems at least possible that the hydronephrosis could have been initiated in these cases by the ptosis of the kidney along with the ptosis of the other viscera, due to poor posture and poor abdominal muscle tone. The bowel and urinary symptoms have all improved or cleared with nerve block therapy, sometimes before the pain had been completely eradicated.

Diagnosis

Like other diseases, this condition is much more likely to be diagnosed if it is kept in mind. It should be suspected in any patient with long standing, persistent, or repeatedly recurring abdominal pain, especially if the pain is unilateral and associated with skin hyperaesthesia of a dermatomal distribution and accompanied by paravertebral tenderness of the nerve roots involved. A history of onset following childbirth and an obviously poor body posture, especially lordosis, are helpful pointers. If on top of this there have been one or more essentially negative laparotomies, the diagnosis becomes quite likely. Visceral symptoms are quite common and do not rule out the diagnosis. It seems that there may be equivocal radiological evidence of upper gastro-intestinal tract changes. It is, of course, often necessary to rule out other causes for the pain by x-rays of the spine and viscera, as well as by special examinations such as sigmoidoscopy, cystoscopy, etc. Finally, the response to differential nerve block therapy is often conclusive evidence, e.g., paravertebral root block, I. V. procaine, splanchnic block. As stated in a previous paper (1), I. V. procaine is very useful in differentiating somatic root pain from visceral pain, the latter being unaffected by it.

Treatment

This varies with the cause, of course, but comes generally under four headings: physiotherapy, nerve blocks, heel lift for short leg if present, and explanation of the nature of the trouble to the patient. Explaining the condition to the patient is important for it dispels the fear of internal disorders, especially cancer, and makes for a more relaxed state of mind and musculature. It also makes the patient understand the importance of good body posture without which permanent relief is unlikely. Attaining this good posture after years of slouching takes considerable time and effort.

The short leg is easily remedied by a heel lift. If the discrepancy is more than 5/8″, the sole should be raised any amounts in excess of the 5/8″.

Physiotherapy involves infra-red or short-wave diathermy directed at the spine where the nerves are affected and the para-spinal muscles are in spasm, and *not* at the areas where the pain is felt anteriorly. Massage of the spinal muscles is helpful also. Most important of all, however, are the postural exercises and the proper carriage taught by the physiotherapist. This will straighten the spine and strengthen the muscles, especially of the abdomen. These exercises should not be done while the pain is severe or to the point where they aggravate it.

The object of the *block* therapy is to break the vicious circle of pain–muscle spasm–pain mentioned earlier. There are several methods and drugs that may be used. The drugs include procaine, xylocaine, proctocaine, dolamine (.75% ammonium sulphate, benzyl alcohol .75% with .48% sodium chloride), and absolute alcohol. All, except possibly the alcohol, may be used for paravertebral injection and/or epidural injection. Paravertebral injections, although very useful in treatment and often essential are painful to perform, take moderate skill, and occasionally give rise to a severe neuritis. Also, if the painful area is extensive, a great number of injections may be needed. It was thus a great satisfaction to find that repeated intravenous procaine not infrequently had a dramatic curative effect on these patients (1), especially if occasionally fortified by a paravertebral block. It is thought that the intravenous procaine acts partly by relaxing the arteriolar spasm (vide supra) and thus improving the blood supply to the nerve and posterior root ganglia where they lie in the intervertebral foramen and partly by seeping through the damaged capillary walls and exerting an analgesic effect. By virtue of this action the pain is often *increased* following the first treatment (although there is relief during and for a few hours following the treatment) as the dilated vessels take up more space; however, this is soon compensated for by reduction of the oedema that follows the improved blood supply. Definite improvement may not be manifested until after the second or third treatment.

Proctocaine was used frequently, usually 5 c.c. being deposited around each affected nerve root. This was considered to be fairly effective.

Subarachnoid alcohol was used in one case only (No. 5), on four occasions and with considerable success. This patient was also given a lumbar sympathetic

block with 5 c.c. of absolute alcohol for severe burning pain in the region of the left hip with a good result.

Dolamine was thought to be a useful drug and was used paravertebrally and extra-durally as well as locally. There were no side effects.

Fifty per cent nitrous oxide analgesia was often used while doing blocks and seemed to be appreciated by the patients.

Discussion

Someone once described a woman as "a charming person with a pain in her side and a headache", with the inference that was woman's lot and nothing could be done about it. Incidentally, many headaches, especially occipital ones, can be explained on a postural basis, but that is another story. Twenty of the twenty-three cases were women. Although women undoubtedly suffer from abdominal and pelvic troubles due to their special organs, it is also true that they suffer more from postural disabilities, because of their relatively poor musculature and also on account of child bearing. Six of the cases described here developed their pain syndrome shortly after the birth of their first or second child. The extra work involved by children, carrying the child for example (pre and post partum), the extra nervous strain, as well as the general debility and lack of muscle tone post partum, constitute an ideal setup for the development of postural distortions with resultant pain.

It was noteworthy also that all the cases in the child bearing age group had exacerbations of their pain just before, during, or following their menstrual period; or when it was due in those who had had hysterectomies. This would fit in with the nerve root oedema theory, sodium retention being an accompaniment of menstruation.

It should be stressed that the painful areas may spread to involve greater areas of the body or they may migrate up or down or to the other side of the body. With an S type scoliosis the patient may have pain in the left lower quadrant and the right upper chest for example (2).

The commonest site for the pain is in the area of distribution T $\overline{\overline{XII}}$ and L $\dot{\overline{I}}$, because this is where the mobile lumbar meets the relatively immobile thoracic spine, and is thus subject to more strain. High heels aggravate the pain in some cases. Two cases in this series volunteered this information. This is to be expected as they tilt the pelvis forward, thus increasing any lordosis that is present.

One hears much these days about "psychosomatic medicine". The body and the brain are in indissoluble unity, of course, and what happens to one always affects the other. In two cases in this series (No. 6 and No. 7) it was felt that the segmental neuralgia, which was real enough, was secondary to severe muscle spasm brought on by hysteria. Many of the other cases, however, were undoubtedly examples of somatopsychic origin, in which the constant pain affected the patient's personality adversely. This could often only be fully appreciated in retrospect when one saw the remarkable difference in appearance and outlook that the patient, relieved of persistent pain, presented.

Conclusion

An important factor in these cases is that the pain therapist be persevering, and not be discouraged by temporary setbacks or disappointing results. Persistent efforts, sometimes, with different drugs or different methods will often be rewarded by ultimate success. The patients are sometimes "difficult", no doubt due to their prolonged suffering and frequent disappointments, and it is necessary to be "en rapport" with them at all times if one is to avoid unnecessary failures due to too early discontinuance of treatment.

It is well to remember that the pain therapist is often the court of last resort, the patient having usually been treated and investigated by all possible means and by many other specialists. This entails considerable responsibility, which cannot be lightly cast aside.

REFERENCES

1. Walton, F. A., "Coeliac Ganglion Syndrome". (To be published in CMAJ)
2. Judovich and Bates, "Pain Syndromes". (F. A. Davis Co., Philadelphia 1950)