SUCCINYLCHOLINE-NITROUS OXIDE ANAESTHESIA FOR MITRAL COMMISSUROTOMY* **

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DURING the past decade, cardiac surgery has progressed from the realm of the occasional operation to an established position in the surgical armamentarium. While surgical interventions upon the heart are not commonplace, the performance of these procedures has become sufficiently widespread to necessitate an understanding of the inherent problems involved by all those engaged in the surgical care of these patients. These problems vary considerably in relation to the individual cardiac lesion for which surgical therapy is undertaken: in the instance of surgical attack upon mitral stenosis by commissurotomy (or valvulotomy), they impose unique demands upon not only the surgeon and the cardiologist, but also upon the anaesthesiologist.

An intelligent approach to the anaesthetic management of the patient undergoing mitral commissurotomy must be based upon a detailed knowledge of the pathologic physiology of mitral stenosis. A number of different anaesthetic techniques have been evolved and described (1-5). The anaesthetic agents chosen are of far less importance than a strict adherence to certain fundamental tenets of anaesthetic management which have followed from a rational understanding of the profound physiological aberrations associated with mitral valvular disease. It will be the purpose of this presentation to describe an anaesthetic technique which employs succinylcholine-nitrous oxide anaesthesia to overcome a number of the very real difficulties imposed by the operation of commissurotomy in these seriously ill patients.

THE PATHOLOGIC PHYSIOLOGY OF MITRAL STENOSIS

Mitral stenosis has its origins in the initial attack of acute rheumatic fever, which occurs, typically, during the first or second decade of life. There follows a relatively asymptomatic period of from several to twenty or more years during which the endocarditis produces a progressive fibrosis and contraction of the mitral valve. Mild dyspnoea on exertion occurs, associated with pulmonary congestion and dilatation of the left auricle. As the narrowing of the valve orifice increases and the pressures within the pulmonary capillaries and the left auricle mount, there are attacks of pulmonary oedema. Further stenosis limits the exercise tolerance progressively, and may be associated with hemoptysis. Myo-

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cardial dilatation and damage occur, causing arrhythmias. In the terminal stages of the disease, death results from cardiac failure.

Specific patho-physiological phenomena are responsible for this downward clinical course of the patient (Fig. 1). The normal mitral valve has a patent orifice that is 4–6 sq. cm. in area. As valve size is reduced by fibrosis and contraction, the pressure within the left auricle begins to rise, and there is an increased pulmonary capillary pressure. When the pressure within the pulmonary capillary bed is of the order of 20 mm. Hg., intimal proliferation and medial hypertrophy of the pulmonary arterioles occur and result in an increased pulmonary vascular resistance. Increased pulmonary vascular resistance produces a rise in pulmonary arteriolar pressure, which in turn leads to right ventricular

FIG 1 A schematic interpretation of the sequence of pathologic physiology in mitral stenosis.

dilatation and hypertrophy. The end result of this chain of physiological changes is, of course, cardiac failure. It should be emphasized that right ventricular hypertrophy and the cardiac failure which may follow occur only after pulmonary vascular resistance has increased, and is accompanied by intimal proliferation and medial hypertrophy of the pulmonary arterioles (6).

THE SELECTION OF PATIENTS FOR MITRAL COMMISSUROTOMY

In the natural history of relatively pure mitral stenosis, four distinct stages of the disease can be defined clinically (7) and are employed as a basis to facilitate the selection of patients for surgery (Fig. 2). Valve size cannot always be correlated with the severity of disease, and it is further obvious that there may be considerable overlap between stages of the disease in the individual patient. Nevertheless, these facts do not obviate the utility of the classification.

Patients in Stage I have a mitral valve area in excess of 1.2 sq cm., and their pulmonary vascular resistances are normal. Therefore, right ventricular dilatation and hypertrophy are not present. The left auricle is enlarged, however, and dyspnoea occurs on violent exertion. Surgery seldom has been performed on this group of patients in the past, and the feasibility of operation remains a most debatable subject. There is an increasing tendency on the part of certain cardiolo-

STAGE	SYMPTOMATOLOGY	VALVE AREA SQ. CM.	AURICLE ENLARGED (X-RAY)	PULMONARY VASCULAR RESISTANCE	RIGHT VENTRICULAR HYPERTROPHY (ECG)	SURGERY	MORTALITY
I	DYSPNEA ON VIOLENT EXERTION	> 1.2	YES	NORMAL	NO	DEBATABLE	VERY LOW
II	SEVERE—PULMONARY DYSPNEA ON ANY EFFORT, ORTHOPNEA PULMONARY EDEMA HEMOPTYSES	1.2	YES	LITTLE INCREASE	+ OR -	IDEAL	- 3-4 %
III	DYSPNEA, FATIGUE, CURTAILED ACTIVITY	1.0 OR LESS	YES	6-10 TIMES NORMAL	YES	INDICATED	-
IV	TERMINAL BED-RIDDEN, ALWAYS ON VERGE OF PULMONARY EDEMA	< 0.8	VERY	> 10 TIMES NORMAL	MARKED	TRANSIENT RELIEF	23 %

FIG. 2. Criteria employed for the selection of patients with mitral stenosis for commissurotomy.

gists, however, towards the belief that the most impressive results of commissurotomy have been obtained in patients subjected to operative intervention *prior* to the development of intimal proliferation and medial hypertrophy, and hence prior to the onset of an increased vascular resistance within the pulmonary circulation (8). Since the operative mortality in Stage I is extremely low, the future may witness an increased incidence of operative interventions with these patients.

Stage II is associated with a critical narrowing of the mitral valve to an area of 1.2 sq. cm., and is characterized by both left auricular hypertrophy and a slight increase in pulmonary vascular resistance. There may or may not be electrocardiographic or radiologic evidence of right ventricular dilatation and hypertrophy. Patients in this group have severe pulmonary symptoms on almost any exertion, since the output of the left ventricle is relatively fixed and any marked increase in output from the right ventricle must result in an increased pressure within the left auricle and the pulmonary bed, with the occurrence of orthopnoea, paroxysmal nocturnal dyspnoea, attacks of acute pulmonary oedema, and hemoptyses. These patients are ideal candidates for surgical intervention, and the mortality rate in this and the following group (Stage III) is generally only about 3-4 per cent.

The valve area in Stage III is 1.0 sq. cm. or less, and the pulmonary vascular resistance is six to ten times the normal. There is evidence of left auricular, right ventricular, and pulmonary artery hypertrophy and dilatation; and the electrocardiogram also demonstrates marked right ventricular hypertrophy. The patients in this group have marked curtailment of activity: they are constantly tired and fatigued, and may remain at total rest so constantly that dyspnoea as a symptom is not even evident. However, the slightest exertion will produce the entire gamut of pulmonary symptomatology. Surgery is most definitely indicated for these patients; the mortality rate of operation is about 3-4 per cent.

Patients in Stage IV have a valve area that is less than 0.8 sq. cm., and a pul-

monary vascular resistance that is more than ten times the normal. Cardiac enlargement of up to 50 per cent is found in these patients: there is a huge right ventricle, prominent pulmonary artery, increased pulmonary vascular markings, and a greatly enlarged left auricle, demonstrable on radiologic examination; and there is definite electrocardiographic evidence of right ventricular hypertrophy. These patients are in the terminal stages of the disease, and are frequently in right-sided heart failure: they are bed-ridden, cachectic, and completely incapacitated, being constantly on the verge of pulmonary oedema. Surgery for this group is extremely hazardous, the mortality rate running 23 per cent. The results are occasionally dramatic in those who survive the operative intervention, but the relief is apt to be transient owing to the irreversible cardiac and pulmonary damage.

THE CONTRA-INDICATIONS TO MITRAL COMMISSUROTOMY

The selection of those patients with mitral disease for whom surgical therapy is indicated is without doubt the most important single aspect of mitral commissurotomy, and while this decision must be a co-operative venture between cardiologist, surgeon, and anaesthesiologist, it is obviously the cardiologist who must bear the brunt of the burden. The difficulties are increased by the fact that there are certain complicating factors which must also be taken into consideration in the selection of candidates for surgery.

At least three of these conditions must be regarded as contra-indications to mitral commissurotomy (Table I). Activity of the rheumatic process is quite

TABLE I

THE CONTRA INDICATIONS TO MITRAL COMMISSUROTOMY

I *Absolute Contra-Indications*

- A Acute rheumatic activity
- B Acute or subacute bacterial endocarditis
- C Significant involvement of the left ventricle
 - 1. Aortic stenosis
 - 2. Aortic regurgitation
 - 3. Mitral regurgitation

II *Relative Contra-Indications*

- A. Calcification of mitral valve (disappointing result)
- B Pregnancy (abortion best; if operation necessary, between 16th and 28th week)
- C. Cardiac failure
- D Pulmonary infarction (anticoagulant therapy prior to commissurotomy)

uncommon at the late stage of the disease process characterized by a critical narrowing of the mitral orifice but when it is present it constitutes an absolute contra-indication to operation. Similarly, acute and subacute bacterial endocarditis are rare in patients being evaluated for possible surgical therapy, but also are an absolute contra-indication to operation when present. Finally, evidence of a significant involvement of the left ventricle, indicating the superimposition of the effects of other valvular lesions upon the mitral stenosis, must be considered a third absolute contra-indication to surgical intervention. This is

not to say that only "pure" mitral stenosis warrants surgery, but, rather, that mitral commissurotomy may enhance the undesirable effects of other valvular disease in certain mixed lesions and thus leave the patient more crippled than prior to the operation. The evaluation of the operability of multivalvular lesions is difficult, the decision being dependent upon whether or not the mitral stenosis is the limiting or dominant lesion physiologically. Lesions of the aortic valve, both stenosis and insufficiency, occur commonly in conjunction with mitral stenosis. If other lesions are of sufficient physiologic importance to produce left ventricular enlargement or damage, then surgical correction of a coexisting mitral stenosis is not feasible. Similarly, patients with severe mitral regurgitation, when it predominates the mitral stenosis, will not benefit from the operation of mitral commissurotomy.

There are, in addition, several conditions which might be termed relative contra-indications to surgical intervention (Table I), but which are in reality only complicating factors that must be taken into consideration in the evaluation of patients for commissurotomy rather than contra-indications to operation. Calcification of the mitral valve, for instance, when extreme, may be associated with a disappointing surgical result owing to a failure of the mitral orifice to remain patent even after an adequate fracture of the commissures; but it is not a true contra-indication to operation. Pregnancy is a second example of a complicating factor that nevertheless does not constitute a true contra-indication to surgery: the greatly increased load which is placed upon the cardiovascular system, particularly during the final trimester, adds an extra risk to mitral disease (9), when the termination of pregnancy is unacceptable to the patient but parturition threatens catastrophe, mitral commissurotomy is performed between the 16th and 28th weeks. Frank cardiac failure cannot be regarded as a contra-indication to operation either, but its presence most certainly does increase the difficulties in an already complex situation. Finally, the occurrence of recent pulmonary infarction, while also not a contra-indication to commissurotomy, may warrant anticoagulant therapy prior to surgical intervention.

THE ANAESTHETIC REQUIREMENTS OF MITRAL COMMISSUROTOMY

Knowledge of the hemodynamic basis of the clinical syndromes and the physiological deviations encountered in patients with mitral stenosis permits an enumeration of the factors which must be considered in the development of any anaesthetic technique designed for the management of the patient undergoing mitral commissurotomy (Table II).

A major tenet of the anaesthetic management must be the avoidance of any physical effort by the patient. Slight exertion may precipitate the onset or progression of frank cardiac decompensation in the patient with mitral stenosis. Such exertion during anaesthesia may take several forms. a tumultuous second stage of anaesthesia during induction, "bucking" against the endotracheal catheter during intubation, when there is too light a plane of anaesthesia following intubation (particularly during certain surgical stimulations or during tracheo-bronchial suction), or at the time of extubation, straining during the course of operation itself, or a period of restlessness and even delirium during emergence

TABLE II

THE ANAESTHETIC REQUIREMENTS OF MITRAL COMMISSUROTOMY

1. Rapid, quiet induction
2. Adequate oxygenation—patent airway, high flows O_2 , positive pressure
3. Avoidance of exertion—straining or “bucking”
4. Maintenance of normotension—throughout operation
5. Lightest possible level of anaesthesia
6. Non-toxic anaesthetic agents—not irritable to myocardium
7. Non-explosive anaesthetic agents—if cautery to be employed
8. Control of cardiac arrhythmias
9. Rapid, quiet emergence

or in the immediate postoperative period. All these several forms of exertion must be avoided by the employment of certain appropriate anaesthetic manoeuvres.

A second major tenet is the maintenance of adequate oxygenation, particularly of the myocardium, throughout the operative and postoperative periods. This is accomplished by ensuring a completely patent airway, by employing high flows of oxygen, and by a sufficient use of positive pressure during respiration both to overcome the constant tendency towards pulmonary oedema and “drive” the oxygen across the alveolar membrane to the pulmonary vascular bed.

Normal systemic blood pressure must be preserved both during induction and throughout the operative intervention, inasmuch as oxygenation of the myocardium is dependent upon mean aortic pressure (10). Anaesthetic agents that would depress myocardial contractility and thus depress blood pressure must therefore not be employed. Normal peripheral resistance is particularly important, since the output of the left ventricle is relatively fixed and cannot increase to compensate for an enlarged capacity of the peripheral vascular bed. Therefore, the vasodilatation which follows upon depression of the medullary centres with even moderate depths of anaesthesia must be avoided by the employment of the lightest possible level of general anaesthesia. The hypotension that often accompanies actual fracture of the valve almost invariably reverts to normotension following the cessation of this particular surgical manipulation.

It is essential also that the diseased myocardium be protected from either physical or chemical stimulations that would tend to induce irritability and severe cardiac arrhythmias. Towards this end, non-toxic anaesthetic agents must be employed, and steps must be taken to decrease the irritability of the myocardium, or to control serious arrhythmias when they do occur, without at the same time producing a severe depression of that muscle.

Finally, it is important to employ non-explosive anaesthetic agents in order to permit the use of electro-coagulation, electrocardiographic recorder with a hot-stylus writing point, or other electrical equipment that might be a fire or explosion hazard.

THE ANAESTHETIC TECHNIQUE FOR MITRAL COMMISSUROTOMY

These multiple requirements have imposed stringent limitations upon the anaesthetic techniques employed for the surgical therapy of mitral stenosis. It is essential that the lightest possible level of general anaesthesia be maintained in

order to obviate the development of hypotension; yet, at the same time, there must be sufficient anaesthetic control over the patient to prevent any "bucking," straining, or exertion. This seeming paradox has been resolved in the present technique by the use of nitrous oxide-oxygen anaesthesia in combination with a dilute intravenous infusion of succinylcholine.

Nitrous oxide, when administered with an abundant concentration of oxygen, is perhaps the least toxic of all the general anaesthetic agents and at the same time the most analgesic. It has the single disadvantage for purposes of commissurotomy that it produces levels of anaesthesia that are so light that surgical manipulations within the thorax and the presence of an endotracheal tube within the trachea are but poorly tolerated without "bucking," straining, or other exertion. This disadvantage can be overcome by the use of succinylcholine, itself a non-toxic drug, to provide graded muscular relaxation that is controllable on a minute-to-minute basis. The combination of nitrous oxide-oxygen and succinylcholine produces an extremely light degree of general anaesthesia and yet prevents straining or exertion on the part of the patient. When, in addition, small doses of pentothal are employed for induction, intermittent doses of demerol are injected intravenously to maintain basal narcosis, and myocardial depressants are administered judiciously to control hyper-irritability of the myocardium, a form of modern balanced anaesthesia results which serves admirably for the anaesthetic management of the patient undergoing mitral commissurotomy (Table III).

TABLE III

ANAESTHETIC TECHNIQUE FOR MITRAL COMMISSUROTOMY

1. Premedication—little or no parasympatholytic drug
2. Induction—pentothal, to loss of eyelid reflex
3. Inhalation—N₂O:O₂ (6:2 litres), semi-closed technique
4. Intubation—apnoeic technique: succinylcholine 0.2 per cent infusion
5. Positioned carefully and slowly
6. Maintenance—
 - N₂O:O₂ (6:2 litres)
 - Succinylcholine 0.2 per cent infusion (assisted respirations)
 - Demerol 10 mgm. i.v. (p.r.n.)
7. Periodic reinflation of lung
8. Tracheal suction only when absolutely necessary
9. Extubation—apnoeic technique: succinylcholine 0.2 per cent infusion
10. Moved to bed slowly, carefully, and only after an interval for stabilization

Premedication. The premedication must differ in certain important respects from that employed in the ordinary surgical patient. Tachycardias are the most frequent and most serious disturbances of rhythm encountered during operation upon the patient with mitral stenosis: filling of the left ventricle is restricted by the stenotic mitral valve, and a superimposed tachycardia will reduce the amount of blood reaching the ventricle. The parasympatholytic drugs are therefore omitted from the preanaesthetic medication because of the danger that even moderate degrees of vagal blockade may precipitate this undesirable arrhythmia.

The usual sedative and hypnotic drugs are employed in light to moderate

dosage, for the patient is frequently so anxious to have the operation and the relief that it promises that apprehension is a minor or even an absent problem. This happy state of affairs does not exist on all occasions, however, and it is important to note that preoperative tachycardia and even pulmonary oedema may be precipitated by apprehension (11). A middle course must be maintained, therefore, and premedication consists of 100 mgm. of demerol administered hypodermically one hour prior to induction, with the addition of pentobarbital 100 mgm. by mouth two hours prior to induction for the very robust or excessively nervous patient.

Induction. Anaesthesia is induced by the intravenous administration of small (2-3 cc), repeated doses of 2½ per cent pentothal. The circulation time may be distinctly prolonged in the patient with mitral stenosis, and almost a minute must be allowed before the effects of the pentothal are judged and reinjection is performed. The depressant effects of pentothal upon the myocardium, with the subsequent production of hypotension, are very real. The great advantages of a rapid, quiet, smooth induction with pentothal must be achieved, therefore, with due consideration to slow injections of minimal amounts of this agent.

Inhalation of nitrous oxide-oxygen, 6 litres: 2 litres, is begun by the semi-closed circle carbon dioxide absorption technique following the loss of the eyelid reflex, which occurs generally after a total dosage of 100-250 mgm. of pentothal.

Endotracheal intubation. When the induction of anaesthesia has been completed, a dilute, 0.2 per cent concentration of succinylcholine in 5 per cent glucose water is started as an intravenous infusion and continued until total apnoea supervenes. During the onset of muscular relaxation, respirations are assisted; when total muscular relaxation and apnoea occur, respirations are controlled. With the onset of apnoea, the patient is hyperventilated with the nitrous oxide-oxygen mixture and then endotracheal intubation is performed, employing the largest, cuffed catheter that will fit through the glottis easily. Prior to intubation, the tip of the endotracheal catheter is lubricated with a water-soluble 1 per cent metycaine anaesthetic jelly, to provide anaesthesia of the vocal cords of sufficient duration to persist throughout operation, but short enough to permit an active cough reflex at the termination of operation or soon thereafter.

Positioning patient for operation. The patient is then positioned carefully and slowly for left thoracotomy. It is essential that this be done in such a manner that sudden shifts of posture, which can lead to profound falls in the blood pressure of the anaesthetized patient, are avoided. Apnoea is maintained by a continuation of the succinylcholine infusion throughout this manoeuvre of posturing in order to prevent "bucking" upon the endotracheal catheter.

Maintenance. Maintenance anaesthesia is established after the patient has been properly postured for operation, and differs from that employed during induction and intubation in two minor, but very important, particulars.

First, the infusion rate of succinylcholine is decreased very gradually to permit the return of spontaneous respiratory activity and the substitution of assisted respirations for controlled respirations. The use of positive pressure during late inspiration is desirable both to offset the possibility of pulmonary oedema and to aid in driving oxygen across the pulmonary membrane. It is important to avoid

the use of continuous controlled respirations in the patient with mitral stenosis, however, since a constant intrapulmonary positive pressure will introduce an added resistance to the flow of blood through the pulmonary circulation and will place a further burden upon the overworked right ventricle. Furthermore, in the technique of continuous succinylcholine infusion, apnoea must be avoided assiduously, for a slight degree of respiratory activity provides the only evidence as to the required rate of flow of the succinylcholine infusion. If apnoea and controlled respirations are maintained throughout operation by the infusion of succinylcholine, it is quite possible to administer larger amounts of succinylcholine than the body can metabolize, and a prolonged period of postoperative apnoea may result. The great majority of the instances of prolonged apnoea which have been reported following the uses of succinylcholine have undoubtedly occurred on this basis (12). It is also important to effect the transition from controlled to assisted respirations very gradually, for a sudden diminution in the rate of flow of succinylcholine may allow a sudden resumption of a marked degree of muscular activity, since the patient is being maintained in an extremely light plane of nitrous oxide-oxygen (6 liters : 2 liters) anaesthesia, the return of marked muscular activity may be associated with dangerous "bucking" or straining.

Second, small (10-20 mgm) intravenous doses of demerol are administered at intervals to supplement the degree of basal narcosis (Fig. 3). The addition of demerol to the combination of succinylcholine-nitrous oxide produces slightly more extensive anaesthesia, without at the same time inducing deep levels of general anaesthesia, than can be maintained by nitrous oxide alone. This extra degree of anaesthesia aids both in preventing the occurrence of "bucking" and, perhaps, in obtunding autonomic activity in these lightly anaesthetized patients. The use of demerol has the added advantages of exerting some quinidine-like action upon the heart and of producing a certain amount of postoperative analgesia and sedation. The total dosage of demerol employed throughout operation is not large, generally less than 200 mgm depending upon the individual patient and the duration of the surgical intervention. The indications for the administration of these small, intermittent doses of demerol are somewhat nebulous and difficult of definition: one, two, or even three doses of 20 mgm. each are administered soon after the patient is postured and maintenance anaesthesia is begun, subsequent 10 mgm doses are injected in response to a slight urgency of inspiration as detected by the anaesthesiologist's hand on the rebreathing bag.

Tracheal suction and reinflation of lung. The problem of whether or not tracheal suction should be employed, and if so, how often, remains an enigma. The tracheo-bronchial tree must certainly be cleared if adequate oxygenation is compromised by the presence of accumulated fluids. On the other hand, pulmonary oedema is a constant and a dreaded threat, and its occurrence may be prompted by the production of strong negative intrapulmonary pressure. A middle course must be steered, and tracheal suction is employed only when urgently necessary. Reinflation of the collapsed lung is carried out periodically, every 20 to 30 minutes, in the same manner as during any other type of thoracotomy.

Endotracheal extubation. Endotracheal extubation is performed with the same anaesthetic technique as that employed for intubation, with the patient in the

apnoeic state, in order to prevent the occurrence of "bucking" or straining. At the termination of operation, and following the application of dressings to the wound, the rate of flow of the succinylcholine infusion is increased to reproduce apnoea and controlled respirations replace the assisted respirations that were employed throughout the maintenance of anaesthesia. The pharynx is suctioned thoroughly, the lungs inflated several times with 100 per cent oxygen, the cuff on the endotracheal tube is deflated, and the endotracheal catheter is removed. Both the succinylcholine infusion and the inhalation of nitrous oxide are discontinued, and controlled respirations with 100 per cent oxygen are employed until spontaneous respiratory activity resumes. Assisted respirations with 100 per cent oxygen are continued further until *full* respiratory activity is present. This generally occurs within a matter of 3-9 minutes, but it is absolutely imperative that assisted respirations with 100 per cent oxygen be continued until there is no doubt as to the total efficiency of the respiratory exchange.

Post-operative care. Following the resumption of total respiratory activity and an interval of time for stabilization, the patient is moved slowly and carefully into bed. There must be no hurry to remove the patient from the operating table;

a few minutes should suffice for adequate stabilization following the resumption of full respiratory exchange, but a longer period may be required and should certainly be allowed when necessary. Postoperative care in the recovery room includes oxygen therapy, pharyngeal or tracheo-bronchial suction as indicated, moderate sedation, and meticulous and ever vigilant nursing care. Medical therapy during this phase is supervised by the cardiologist.

ADJUNCTIVE MEASURES DURING MITRAL COMMISSUROTOMY

Certain adjunctive measures to the management of the patient are of utmost importance during mitral commissurotomy, and are carried out by the surgeon, cardiologist, and anaesthesiologist working in concert (Table IV).

Cardiac activity is followed on an appropriate lead of the electrocardiograph, and is monitored by the cardiologist in person. Certain experienced surgeons feel that changes in cardiac tone, rate, rhythm, and irritability can be appreciated most readily by the surgeon's fingers. While there is no doubt that this is true in many instances, the use of electrocardiographic tracings is advisable as a further check to ensure the correct diagnosis of changes in cardiac action. Procaine amide in 50 mgm. doses is administered intravenously in repeated doses up to a total dosage of 50-300 mgm. when the myocardium is hyper-irritable during the initial exposure of the left auricle. Sinus tachycardia, a frequent occurrence during commissurotomy, may be slowed by the use of intravenous prostigmine in 0.5-3.0 mgm. doses. Multiple arrhythmias occur at the time of actual fracture of the valve, including ventricular extrasystoles, ventricular tachycardia, and bizarre patterns; these, however, require no other therapy than the removal of the surgeon's finger from the valve. Atropine may be employed in doses of 0.3-1.2 mgm. intravenously in the event of severe bradycardia, moderate bradycardia frequently occurs during the closure of the pericardium, presumably on the basis of vagal stimulation, but seldom requires the institution of active therapeutic measures.

The use of vasopressor agents to combat hypotension has been a moot question. Moderately profound hypotension frequently attends upon the actual fracture

TABLE IV

ADJUNCTIVE MEASURES DURING MITRAL COMMISSUROTOMY

1. ECG monitor of cardiac events.
2. Control of cardiac arrhythmias
 - A. Procaine amide. 50-300 mgm. i.v.
 - B. Prostigmine: 0.5-3.0 mgm. i.v.
 - C. Atropine sulphate: 0.3-1.2 mgm i.v.
3. Vasopressor agents—if prolonged or persistent hypotension
4. Blood and fluid replacement—little necessary or desirable, but must be prepared for possible surgical exigency
 - A. 15 gauge cannula *in situ*, and slow intravenous drip
 - B. Compatible blood for transfusion at hand
 - C. Blood pump available
 - D. Intra-arterial transfusion set-up ready
5. Compression of carotids at time of valve fracture—if history of auricular fibrillation or previous embolism

of the mitral valve, since all flow of blood to the left ventricle is terminated temporarily at this point in the operation, normotension, or at least lesser degrees of hypotension, generally follows promptly upon the removal of the finger from the valve orifice. A potent vasopressor agent, such as neosynephrine or levophed, however, should be kept available to combat persistent, severe hypotension.

Blood transfusions and fluid replacement are seldom either necessary or desirable. Indeed, the greatest danger is that unnecessary transfusions or over-zealous fluid therapy may induce congestive failure or pulmonary oedema. On the other hand, it is essential to be prepared for a possible surgical exigency, for a gross haemorrhage of considerable magnitude can occur in a matter of seconds should the auricular opening escape the surgeon's control. A large intravenous cannula (15 gauge), therefore, should be inserted prior to operation and its patency maintained during operation by a slow infusion of 5 per cent glucose and water; compatible whole blood is kept available for transfusion in the operating room. It is advisable to have a mechanical device for pumping blood intravenously, or even an intra-arterial transfusion set-up (2), in readiness.

Finally, if there has been a history of auricular fibrillation or of embolism, bilateral carotid compression is performed at the time of the actual fracture of the valve. While the anaesthesiologist can perform this manoeuvre, it is preferable to have the duty devolve upon some other available medical personnel.

SUMMARY

The anaesthetic management of the patient undergoing commissurotomy must be based upon a detailed knowledge of the fundamental pathologic physiology of mitral stenosis. As increasing fibrosis and constriction reduce the valve area towards the critical size of 1.2 sq. cm., left auricular pressure rises and produces an increased pulmonary capillary pressure. The latter induces intimal proliferation and medial hypertrophy of the pulmonary arterioles, causing increased pulmonary vascular resistance. Right ventricular dilatation and hypertrophy follow, setting the stage for cardiac failure.

Mitral disease can be classified into four clinical syndromes, or stages, which serve as a basis for the selection of patients for surgery. Stage I is characterized by an uncritical narrowing of the mitral valve, left auricular dilatation and hypertrophy, and dyspnoea on exertion: these patients have not usually been regarded as candidates for commissurotomy in the past. Stage II is associated with a mitral valve size of about 1.2 sq. cm., both left auricular and right ventricular hypertrophy, and severe pulmonary symptomatology upon almost any exertion: these patients are ideal candidates for the operation. Stage III is accompanied by a valve size of less than 1.0 sq. cm., greatly increased pulmonary vascular resistance and marked right ventricular hypertrophy, and symptoms of constant fatigue even at total rest: surgical intervention is definitely indicated in these pulmonary cripples, and its results are often dramatic. Patients in Stage IV have a mitral valve area less than 0.8 sq. cm., cardiac enlargement, and are terminal, bed-ridden and cachectic: surgery is extremely hazardous, and while the results are occasionally miraculous, the relief is often transient.

Subacute bacterial endocarditis, rheumatic activity, and predominant mitral regurgitation, aortic stenosis or aortic regurgitation in mixed lesions, must be considered absolute contra-indications to surgery. Marked calcification of the mitral valve, pregnancy, frank cardiac failure, and recent embolism are not true contra-indications, but are complicating factors necessitating special considerations.

The anaesthetic requirements of mitral commissurotomy include a rapid, quiet induction; adequate oxygenation at all times, avoidance of any exertion, straining, or "bucking"; maintenance of normotension throughout operation, the lightest possible level of general anaesthesia; the use of non-toxic and non-explosive anaesthetic agents, the control of cardiac arrhythmias, and a rapid, quiet emergence from the anaesthetic state.

The anaesthetic technique is based upon the use of succinylcholine-nitrous oxide anaesthesia for the fulfilment of these varied requirements. Premedication is light or moderate, and parasympatholytic drugs are avoided. Small, repeated doses of pentothal are employed for induction, following which nitrous oxide-oxygen, 6 liters : 2 liters, is administered by the semi-closed circle carbon dioxide absorption technique. Succinylcholine, 0.2 per cent concentration in 5 per cent glucose in water, is begun as a constant intravenous infusion and continued until apnoea occurs, at this time, oral intubation is performed with a large, cuffed endotracheal catheter. After slow and careful posturing of the patient, the succinylcholine infusion is slowed until spontaneous respiratory activity is just evident. Anaesthesia is then continued with the nitrous oxide-oxygen mixture and assisted respirations throughout, the flow rate of the succinylcholine infusion being adjusted constantly to maintain minimal, but spontaneous, respiratory activity. Small (10-20 mgm) doses of demerol are administered intravenously at intervals to supplement the basal narcosis. The lungs are reinflated periodically, but tracheal suction is avoided except when the gross accumulation of fluids in the tracheo-bronchial tree make its use mandatory. At the termination of operation, the succinylcholine infusion rate is again increased to produce apnoea for the removal of the endotracheal catheter. An interval for stabilization is permitted after the cessation of the succinylcholine infusion and the resumption of *complete* respiratory activity before the patient is moved, slowly and carefully, into bed.

Cardiac activity is monitored continuously by the electrocardiograph. Procaine amide, in 50-300 mgm. doses, is injected intravenously to control any hyper-irritability of the myocardium during the exposure of the left auricle. Sinus tachycardia may be slowed by the use of intravenous prostigmine in 0.5-3.0 mgm. doses. Bradycardia rarely becomes a problem, but may be an indication for the intravenous administration of 0.3-1.2 mgm. of atropine sulphate. Prolonged and profound hypotension may necessitate the use of vasopressor agents. Fluid therapy is restricted at all times, in order to prevent overloading the circulation; but compatible blood and a pressure transfusion set-up should be available in the event of a surgical exigency leading to gross haemorrhage. Bilateral carotid compression is employed if there has been auricular fibrillation or previous embolism.

RÉSUMÉ

Le traitement anesthésique du malade qui subit une commissurotomie doit reposer sur une connaissance détaillée de la physiologie pathologique fondamentale de la sténose mitrale. La fibrose et la constriction, quand elles augmentent, réduisent d'autant la surface de la valvule jusqu'à atteindre la dimension critique de $1,2 \text{ cm}^2$, la pression monte dans l'oreillette gauche et provoque une augmentation de la pression capillaire pulmonaire. Cette dernière entraîne une prolifération de l'intima et une hypertrophie de la média des artérioles pulmonaires, produisant ainsi un accroissement de la résistance vasculaire pulmonaire. Une dilatation et une hypertrophie du ventricule droit s'ensuivent, préparant le terrain à l'insuffisance cardiaque.

Les affections mitrales peuvent se classer en quatre syndrômes, ou stades, cliniques, qui servent de base à la sélection des malades à opérer. Le premier stade est caractérisé par un rétrécissement non-critique de la valvule mitrale, une dilatation et hypertrophie de l'oreillette gauche, de la dyspnée d'effort. d'habitude, ces malades n'ont pas été dans le passé considérés comme candidats à la commissurotomie. Le deuxième stade se caractérise par une valvule mitrale d'environ $1,2 \text{ cm}^2$ de surface, une hypertrophie à la fois auriculaire gauche et ventriculaire droite, et une symptomatologie pulmonaire sérieuse après le plus petit effort: ces malades sont les candidats parfaits à l'opération. Le troisième stade s'accompagne d'une valvule mitrale de moins de 1 cm^2 de surface, d'une augmentation considérable de la résistance vasculaire pulmonaire, d'une hypertrophie ventriculaire droite marquée et de symptômes constants de fatigue, même au repos absolu: l'intervention chirurgicale est toute indiquée dans ces conditions pulmonaires critiques et les résultats sont souvent spectaculaires. Les malades du quatrième stade ont une surface de la valvule mitrale inférieure à $0,8 \text{ cm}^2$, un grossissement cardiaque et sont dans la phase finale de la maladie, cloués au lit et cachectiques: la chirurgie est extrêmement hasardeuse, et bien que les résultats tiennent quelquefois du miracle, le soulagement est souvent passager.

L'endocardite bactérielle subaigue, l'activité rhumatismale, ainsi que l'insuffisance mitrale prédominante, la sténose aortique ou l'insuffisance aortique compliquées de lésions, doivent être considérées comme contre-indications absolues de la chirurgie. Les calcifications nettes de la valvule mitrale, la grossesse, l'insuffisance cardiaque marquée, et les embolies récentes ne sont pas exactement des contre-indications mais des facteurs de complication qui exigent des considérations spéciales.

L'anesthésie pour la commissurotomie mitrale exige: une induction rapide et douce, une oxygénation constamment adéquate; l'absence de tout effort, tension ou "cabrage", maintien de la tension normale pendant toute l'opération; le niveau le plus léger possible d'anesthésie générale; l'emploi d'agents anesthésiques non-toxiques et non-explosifs; le contrôle des arythmies cardiaques; et une sortie rapide et douce de l'état anesthésique.

La technique anesthésique est basée sur l'emploi de l'anesthésie au protoxyde d'azote-succinylcholine, en vue de satisfaire à ces diverses exigences. Le prémédication est légère ou modérée, et on évite les médications parasympatholytiques.

Pour provoquer le sommeil, on emploie de petites doses répétées de pentothal, en suite de quoi on administre du protoxyde d'azote-oxygène dans la proportion 6 litres/2 litres par la technique de l'absorption de l'anhydride carbonique en circuit demi-fermé. On commence l'injection intra-veineuse continue, de la succinylcholine, sous la forme d'une concentration à 0,2% dans 5% de glucose dans l'eau et on continue jusqu'à apparition de l'apnée, à ce moment, on pratique une intubation orale à l'aide d'une large sonde endotrachéale munie d'un brassard pneumatique. Après avoir mis le malade en position, lentement et avec soin, on ralentit l'injection de succinylcholine jusqu'à ce que l'activité respiratoire spontanée soit tout juste évidente. On continue alors l'anesthésie avec le mélange protoxyde d'azote-oxygène et avec des respirations assistées tout au long, le débit de l'injection de succinylcholine étant constamment réglé de manière à maintenir une activité respiratoire minime, mais spontanée. On administre en intra-veineuses de petites (10-20 mg) doses de démerol de temps à autre pour compléter l'anesthésie de base. On insuffle périodiquement les poumons, mais on évite les succions trachéales sauf quand l'accumulation de mucosités dans l'arbre trachéo-bronchique en rend l'emploi impératif. A l'issue de l'opération, le débit de l'injection de succinylcholine est à nouveau accru pour produire l'apnée pendant l'extraction de la sonde endotrachéale. On ménage un intervalle après la cessation de l'injection de succinylcholine et la reprise *complète* de l'activité respiratoire avant de remettre, lentement et avec soin, le malade au lit.

L'activité cardiaque est suivie en permanence sur l'électrocardiographe. On injecte, en intra-veineuse, de la procaine amide, en doses de 50 à 300 mg pour pallier à toute hyper-irritabilité du myocarde pendant la mise à nu de l'oreillette gauche. La tachycardie des sinus peut être ralentie par l'emploi de prostigmine en doses de 0,5 à 3 mg intraveineuses. La bradycardie devient rarement un problème mais peut être une indication de l'administration intraveineuse de 0,3 à 1,2 mg de sulfate d'atropine. Une hypotension profonde et prolongée peut exiger l'emploi d'agents vasoconstricteurs. On évite à tout moment la rehydratation thérapeutique, pour éviter de surcharger la circulation, mais il est bon d'avoir sous la main du sang compatible et un appareil de transfusion sous-pression pour prévenir tout besoin chirurgical entraînant une grosse hémorragie. On emploie la compression bilatérale de la carotide s'il y a eu fibrillation auriculaire ou embolie antérieure.

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