ANESTHESIA FOR MITRAL COMMISSUROTOMY *†

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Received for publication July 21, 1952

PATIENTS with severe mitral stenosis are the poorest surgical risks who have undergone regularly and successfully an elective surgical procedure as hazardous as mitral commissurotomy. The operation combines the hazards of general surgery and anesthesia with those special hazards associated with intrathoracic operations and operations on the heart itself. Perhaps mitral commissurotomy should not be considered an elective operation since medical management can offer these severely disabled patients little more than a few months of invalidism. Such a poor prognosis has justified the risk involved in the operation and, with experience, anesthesiologists have gained respect for the stamina remaining in many failing human hearts. However, the stamina of the heart cannot be given all the credit for the success of these operations. Some must go to the teamwork of groups of physicians of which the anesthesiologist is a member. Others included are the internist, pediatrician, cardiologist, physiologist and surgeon. The efforts of each must be coordinated exactly with those of all the others to insure the safest conduct of the poor-risk patient through a dangerous period.

PATHOLOGIC CONSIDERATIONS

To understand fully the problems involved in administration of anesthesia to these poor-risk patients it is necessary to know the disease and its sequelae. The opening through the mitral valve of the average adult measures about 4 sq. cm. Through this opening the cardiac output necessary at rest can flow at very low pressure. The left ventricle fills almost passively during the early part of diastole only. On demand great volumes of blood can flow through the normal mitral orifice with only small increase in pressure in the left auricle.

The scarring following endocarditis causes the mitral orifice to become smaller. As it constricts, increased resistance is offered to the flow of blood through it and if the normal flow is to be maintained the blood must be forced through at a higher pressure. The ratio of the pressure to the decrease in size of the orifice increases rapidly. Very early in the disease before symptoms of stenosis occur, the atrium may

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† Read at the meeting of the Illinois Society of Anesthesiologists, Chicago, Illinois, May 12 and 13, 1952.

be able to supply the necessary added pressure to the blood. However, being a thin-walled structure its powers of compensation are almost negligible and the atrium soon becomes only a dilated and frequently fibrillating tube which transmits the pressure supplied by the right ventricle through the pumonary vessels.

Lewis and associates (1) have shown that a mitral orifice of about 1 sq. cm. is of a critical size. The pressure necessary to force the resting cardiac output through a valve of this size is about equal to the osmotic pressure of blood, that is, 25 mm. of mercury; hence pulmonary edema and hemoptysis begin to occur. Little increase of flow is possible even though the pulmonary pressure is raised to a point much above 25 mm. of mercury; therefore, the cardiac output becomes fixed. At this point, compensatory hypertrophy of the pulmonary arterioles may occur to protect the pulmonary capillaries from edema-producing pressures. This increase in pulmonary resistance throws a greater burden on the right ventricle and when it cannot meet the demand, symptoms of right-sided heart failure occur: hepatomegaly, ascites and peripheral edema. Patients whose mitral area measures less than 1 sq. cm. are able to exist by maintaining a very delicate balance between pressure in the right ventricle and pulmonary resistance. Any slight alteration during anesthesia and operation may upset this delicate balance. The situation may be summed up by saying that these patients have practically no cardiac reserve. Even slight alterations in physiology for which compensation would go unnoticed in the average patient may mean the difference between life and death to the patient with mitral stenosis undergoing commissurotomy.

PREMEDICATION

Since a potent anesthetic agent is employed, it has been my practice to use only light premedication. Pentobarbital sodium in a dose of 100 mg. by mouth and morphine sulfate in a dose of 10 mg. subcutaneously are given to the average adult about an hour before the anesthesia is to be started. It is felt that this combination of drugs inhibits some of the patient's apprehension and the morphine may help to decrease the demand of the tissues for oxygen. As premedication for the first few operations, a half dose of atropine sulfate (0.2 mg. for the average adult) was combined with the morphine. The dose of atropine was restricted in an attempt to minimize the tachycardia which had been reported by others to be especially detrimental during mitral commissurotomy. After the first few operations the administration of atropine was eliminated entirely and, in spite of ethyl ether being used as the agent for maintenance of anesthesia, no disadvantages such as excessive secretion have been encountered as a result of the fact that no atropine or related drugs have been given.

The practice of elimination of atropine from the premedication has been difficult to evaluate. Suffice it to say that some nonatropinized patients have experienced mild tachycardia which might have been more marked had the vagus been inhibited. None have experienced tachycardia that was severe enough to necessitate postponement of the operation and no neostigmine has been used for its parasympathomimetic action. However, both theoretically and clinically tachycardia has been a serious complication. Stenosis of the mitral valve delays filling of the left ventricle with blood even though filling continues throughout diastole. As the heart rate increases and diastole is shortened, less time is allowed for filling to occur and stroke volume is decreased. The situation then arises whereby a reflex which would ordinarily increase cardiac output actually decreases the output when the mitral orifice is severely decreased in size. It has seemed that patients with auricular fibrillation have done better during operation than those with sinus rhythm because the former when digitalized have shown less tendency to experience tachycardia.

ANESTHESIA

The attitude and philosophy of the anesthesiologist would seem to be more important than the use of any particular agent or method. In preparation for mitral commissurotomy at the Mayo Clinic, the patient is given every drug and treatment which is beneficial. Everything constructive is done, the cardiac reserve is developed to the maximum and the most advantageous time for the operation to be done is carefully selected. There is little or no beneficial therapy left to be instituted in the operating room by the anesthesiologist. In contrast, up until the time that the valvulotomy is accomplished, the anesthesia and surgical procedure are necessarily detrimental to the well-being of the patient. If the anesthesiologist is prevented from using beneficial procedures, his general objective must be to do as little harm as possible. All his thinking and action in regard to questionable procedures must necessarily be of a negative nature. For these reasons my attitude has been one of conservatism. As few drugs in as small doses as possible are used. No agent or method is employed until I am convinced that there is a definite, clear-cut indication and until I am convinced that the agent or method will do more good than harm. This philosophy has already been expressed in the elimination of atropine from the preanesthetic medication. My objective is to keep everything as simple and uncomplicated as possible.

Some type of general anesthesia, in preference to local anesthesia, has been used in most reported cases of operations on the mitral valve. The production of relief of pain for the patient and the provision of good operating conditions for the surgeon are relatively minor prob-

lems for the anesthesiologist as compared with his problems of keeping the patient alive and safe.

Most general anesthetic agents are considered to cause vasodilation and decrease in over-all peripheral resistance to the flow of blood through the tissues. The amount of vasodilation is proportional to the dose of the general anesthetic agent and the depth of anesthesia produced. For the blood pressure to remain constant when the peripheral resistance is decreased, the cardiac output must be increased. Patients with severe mitral stenosis have a relatively fixed cardiac output and cannot adequately increase their cardiac output to maintain homeostasis. Therefore, their blood pressure theoretically should fall when they are deeply anesthetized and that is what happens clinically. The deeper the general anesthesia, the more the blood pressure falls. The heart rate may be increased in an attempt to maintain the blood pressure but beyond a certain rate the tachycardia becomes a disadvantage rather than an advantage.

At centers for cardiac surgery various agents have been used successfully to produce general anesthesia for operations on the mitral valve. No one agent seems to have outstanding advantages over all others. The depth of the anesthesia seems much more important than the agent employed. For all the operations performed on the mitral valve at the Mayo Clinic, the anesthesia has been induced with nitrous oxide and oxygen and maintained with ethyl ether and oxygen using circle absorption semiclosed technic. This combination was selected primarily because over a period of years the anesthesiology staff had come to consider ethyl ether more satisfactory than other agents for intrathoracic operations and for most patients with poor cardiac reserve. The use of the combination has been continued because it has met the demands of the operation as satisfactorily as the observed use of other agents in the hands of other anesthesiologists.

The mere fact that ether is a potent anesthetic agent does not mean that only deep stages of anesthesia may be produced with it. To one not experienced in administering anesthesia for operations on the mitral valve it is almost unbelievable how little anesthetic agent is necessary for these patients. After the thorax is opened under light ether anesthesia many of the poorest-risk patients may go for as long as an hour without the need for any additional anesthetic agent. Fortunately the poorest-risk patients who suffer most from deep anesthesia are the ones who demand the least anesthesia. Since anesthesia is harmful to patients with severe mitral stenosis in proportion to the depth of the anesthesia, my policy has been to give just as little anesthetic agent as possible and to keep the patient in the very lightest stage of anesthesia that is compatible with conduction of the operation. The deepest anesthesia required during the entire operation is for intratracheal intubation—deep to prevent breath-holding with resulting cyanosis. After intubation the anesthesia is kept just deep enough to

prevent coughing. The one thing of which I am most convinced about the care of patients during operations on the mitral valve is that they cannot be treated like ordinary patients or even like debilitated patients who are undergoing intrathoracic operations. They require less anesthesia and, if they are given an average amount of anesthesia with any agent, they frequently will not survive the operation.

Although it is used for routine intrathoracic operations, thiopental sodium for induction of general anesthesia has not been employed for operations on the mitral valve. There is no particular reason why small doses of this agent should be contraindicated for patients with severe mitral stenosis but its use is not absolutely essential. These patients are usually quite anxious to have the operation performed and do not make demands about the anesthesia and its manner of induction.

SUPPORTIVE THERAPY

Since failure of the damaged and overburdened heart is the most likely serious complication during mitral commissurotomy, the efforts of the surgical team are directed toward maintaining adequate cardiac function. No treatment other than relief of the obstruction is going to make the heart function any more efficiently; hence any supportive measures must be directed toward preserving what efficiency is present at the beginning of the operation. Again the philosophy is one of prevention and avoidance of adverse factors and circumstances. Nothing is more dangerous than hypoxia of the myocardium and every possible means must be used to reduce its occurrence to a minimum.

The use of a potent anesthetic agent, such as ethyl ether, allows the administration of high tensions of oxygen. Assisted respiration promotes more nearly adequate pulmonary ventilation when the pleura is open. A cuffed intratracheal tube facilitates assisted respiration, and when the tube is attached directly to the anesthetic machine dead space is reduced. Any obstruction to the respiratory passages must be removed as soon as detected. These are routine procedures for most anesthesiologists during all intrathoracic surgical operations but during mitral commissurotomy they are even more important than for other operations. Anesthetic apparatus must be checked thoroughly before anesthesia is induced and constantly inspected thereafter. A technical failure the effects of which could be tolerated easily by the average patient until repairs could be accomplished might mean the difference between cardiac compensation and decompensation in the patient with mitral stenosis.

Since coronary blood flow is proportionate to the pressure in the proximal aorta, adequate blood pressure must be maintained if at all possible. The danger of deep anesthesia with resulting vasodilatation and fall in blood pressure cannot be overemphasized.

It has been suggested that blood pressure be maintained by the ad-

ministration of vasopressor drugs which have only slight pharmacologic effect on the heart itself. Theoretically this would seem to be a logical measure to diminish the vasodilation caused by the anesthetic agent. To date I have not had to administer vasopressor drugs during the operation. However, the continuous intravenous administration of such drugs to maintain blood pressure during vascular operations for conditions other than mitral stenosis has been disappointing. Neither have intermittent emergency doses of stronger concentrations of vasopressor drugs been found necessary since manipulation of the pericardium or the heart itself has not resulted in reflex depression of blood pressure. Again, the policy has been not to use any drug or method without some specific or demanding indication for its use.

Replacement of blood loss by transfusion presents many problems. Although incision of the atrium is not followed by as sudden loss of blood as is incision of the ventricle during operations for stenosis of the pulmonary valve, preparation must be made for rapid replacement of blood before the operation is started. The opening in the auricle may be split by passage of the surgeon's finger and sudden loss of blood could occur at the relatively high atrial pressure. My practice is to put a 15-gauge needle in a flick-walled vein in the right leg and another in a vein in the left forearm, a left posterolateral incision being preferred by the surgeon. A simple apparatus is prepared for administering the blood under pressure. Intra-arterial transfusion has not been found necessary but facilities for administration of blood into the exposed aorta are kept constantly available. Such facilities must always be prepared but should seldom be used. It is my belief that more serious results will follow administration of too much blood than too little blood to these patients with potential if not actual cardiac decompensation. In the excitement of entering the heart of a patient whose blood pressure is perhaps decreasing, the loss of blood is very apt to be overestimated. Sponges are weighed and blood aspirated from the thorax is collected in a calibrated trap to gain objective evidence of the actual loss of blood. I prefer to keep blood replacement 100 or 200 cc. less than the objectively estimated loss. None of my patients have experienced acute pulmonary edema during the operation but it is believed that many of them might have done so if a little too much blood had been administered too rapidly.

Once the anesthesia has been induced no operation for mitral commissurotomy has had to be postponed or discontinued. In spite of this, what was considered an optimal blood pressure could not be maintained in all patients. In spite of all our preventive efforts the systolic blood pressure has remained as low as 60 to 70 mm. of mercury systolic for as long as an hour in a few patients but all have recovered promptly from the anesthesia and none have had any symptoms of cerebral hypoxia. It seems that either these patients have developed a tolerance

to low blood flow or our past opinion about what constitutes a safe minimal blood pressure during anesthesia has been too high. The advent of hypotensive anesthesia would indicate that the latter is more likely correct.

Cardiac arrhythmia is a complication which must be considered during any operation on the heart. Adequate oxygenation of the heart by the measures mentioned is probably the best means of preventing arrhythmia. Once it has occurred its significance can be properly evaluated and scientific treatment instituted only after an accurate electrocardiographic diagnosis has been established. Palpation of the pulse alone is inadequate and a direct-writing electrocardiograph is almost essential equipment for determining the actual ventricular rate during auricular fibrillation and for determining the site of origin of premature contractions.

Sinus tachycardia has been the most frequent and most serious arrhythmia encountered. The nodal rhythm that often occurs during ether anesthesia does not seem to alter the efficiency of the heart. The occasional and isolated premature ventricular contractions have not been treated but are a warning to watch the electrocardiogram for the occurrence of more serious arrhythmias. During the time that the valve is being cut or palpated, ventricular extrasystoles, ventricular tachycardia and bizarre patterns are expected. To date in my experience the use of procaine-like drugs has not been necessary during any operation on the mitral valve either as treatment or as prophylaxis. When the arrhythmia seems persistent the practice has been for the surgeon to withdraw his finger into the lumen of the atrium away from the valve area and give the heart a period of rest. In all patients the rhythm has returned to normal or as near normal as existed early in the operation. It is my opinion that there is a place for the use of procaine-like drugs in the armamentarium of the anesthesiologist but it just happens that I have not seen any clear indication for the use of such drugs during an operation on the mitral valve. The reason for not using such drugs prophylactically is that they may cause myocardial depression. While this may not be an adverse effect in the average patient, it is assumed that the myocardium of some of the patients with mitral stenosis undergoing operation is just barely able to maintain an efficient circulation. The depression by procaine might be the factor which would precipitate cardiac failure.

Cardiac asystole occurred during two of the first thirty mitral commissurotomies at the Mayo Clinic. In one instance the arrest was secondary to fatal bilateral embolism of the anterior cerebral vessels from a thrombus in the right atrium. The other arrest occurred just after incision of the valve; there was prompt response to cardiac massage so that no further therapy was necessary. This second patient had an uneventful convalescence without any evidence of cerebral hypoxia.

SUMMARY

Patients with severe mitral stenosis have a relatively fixed cardiac output and do not tolerate deep stages of general anesthesia. Tachycardia has been the most serious cardiac arrhythmia encountered and atropine has been eliminated as a premedication agent in an effort to reduce the incidence and severity of tachycardia. Neither the prophylactic nor therapeutic use of procaine-like drugs has been found necessary.

REFERENCE

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