Complete heart block remains a prominent cause of death and disability following repair of septal defects.

There are several factors which may precipitate heart block during cardiac surgery. Damage to the conduction mechanism by sutures is the more prominent cause, but as has been noted previously, the state of the myocardium prior to surgery may well influence the supervision of this arrhythmia. Thus, in one published report, each of 11 patients with complete surgical heart block had severe pulmonary hypertension, the pulmonary artery pressure being 70 per cent - 80 per cent of the systemic pressure. In some of these cases, the authors observed heart block to occur before any sutures had been placed in the heart.1

We have no control of the patient's heart muscle before surgery, but a good perfusion during operation will prevent anoxic damage to the conduction mechanism.

A further cause of heart block arising during cardiac surgery is hypothermia, which is frequently used in association with extracorporeal techniques. The effect of cold on the heart consists of slowing of impulse formation and of conduction within the conducting system itself. This produces varying degrees of heart block, which terminate either in complete asystole or in ventricular fibrillation. With rewarming, these conduction disturbances reverse so that by the end of an operation, and in the absence of trauma to the bundle, normal rate and rhythm will return. However, should rewarming be incomplete atrioventricular dissociation may persist for several hours until normal body temperature is reached.

Although a number of patients with heart block may revert to sinus rhythm within the early postoperative period, the majority will remain in complete atrioventricular dissociation and of those who survive and leave the hospital, a further proportion will die during an attack of extreme bradycardia. Thus Kirklin reported in 1960 that of 298 patients having repair of ventricular septal defects, 48 had complete heart block at some time following operation. Eighteen of these died in the immediate postoperative period and a further 18 reverted to sinus rhythm. Of the 12 leaving the hospital, three died subsequently.4 Corresponding results are obtained in other large published series.

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Therefore, there apparently is a risk that about 10 per cent of these patients will incur permanent heart block, with a total mortality of about 5 per cent. This risk could be considered a barrier preventing repair of ventricular septal defect from taking its place in the group of relatively safe heart operations. It therefore behooves us to utilize a technic of repair which will avoid damage to the conduction mechanism.

Although a considerable amount of painstaking work has been undertaken to trace the main bundle in the normal human heart and in animals, less information is available concerning its morphology in hearts bearing congenital septal defects, for the good reason that successful reparative surgery is more and more successful. However, such work as has been done on the abnormal heart shows that there is little variation in the localization of that part of the main bundle which is at risk. Thus, Reemtsma and Copenhagen3 showed that in cases of ventricular septal defect and persistent atrio-ventricular canal, the bundle of His courses along the postero-inferior margin of the ventricular defect in a subendocardial position. Truex and Bishop4 showed that in a series of 15 abnormal hearts, 12 conduction mechanisms followed this course. In the remaining three, the course of the bundle was more irregular, but in these the defects were in the muscular septum and were complicated by additional defects. Lev's classical work indicates this postero-inferior course to be usual in ventricular septal defects, both isolated and associated with tetralogy of Fallot, and in endocardial cushion defects. He found that the main bundle often stays more to the left than the right side of the summit of the inferior wall of the defect.

Allen and his colleagues, having utilized a rather ingenious method of vital staining of the bundle during surgery, using 5 per cent iodine dissolved in 10 per cent sodium iodide. They demonstrated the bundle and were able to avoid it. However, there is experimental evidence demonstrating damage to the conducting cells by the dye and this method has not gained general favor.

A more promising approach to the problem of identification of the bundle at surgery is the use of an apparatus which measures electrical impedance of the bundle.10

Figure 2: Closure of ventricular septal defect using a Dacron patch.
Reemstma and his colleagues showed in three patients who died of surgical heart block following closure of septal defects, that suture tracks and hemorrhage in or around the bundle were evident in its course along the postero-inferior rim of the ventricular defect. 

Despite minor variations, it is clear that the bundle runs postero-inferior to the defect when viewed from the right side and in a subendocardial position.

Applying this knowledge, a technic of closure has been utilized with excellent results at this center. All of these patients had their defects repaired using a pump oxygenator heart exchanger which we have previously described. The majority of ventricular septal defects were closed using a crimped Dacron patch, this patch being fixed with continuous and interrupted silk sutures. At the lower and posterior aspects of the defect, interrupted mattress sutures are placed several millimeters away from the defect and bite the right side of the muscular septum only, in this way avoiding strangulation of the bundle as it passes through this region. The insertion of the septal leaflet of the tricuspid valve is included in the sutures in this region in order to buttress this part of the repair. This maneuver is exaggerated in the repair of ostium primum defects, the patch being sutured more to the insertion of the septal leaflet of the tricuspid valve rather than to the ventricular septum at the lower edge.

At the remainder of the circumference of the defect, large bites of tissue are taken and a continuous suture is passed through the full thickness of the muscle. None of these sutures is drawn tighter than is necessary to oppose the muscle wall to the prosthesis, reliance being placed on numerous deep bites. The patch used is somewhat larger than the defect to be covered, and probably adds increased volume to the left ventricle. This may be of value in correction of tetralogy of Fallot, where left ventricular capacity is at a premium.

Ventricular septal defects are repaired under hypothermic arrest, with total body cooling to 26°C, and the heart being additionally cooled with frozen Ringer's lactate slush. The aorta is cross-clamped in order to diminish venous return to the heart, this clamp being released from time to time, usually every five minutes in order to perfuse the coronary arteries. Under these conditions, precise and deliberate placing of sutures is readily accomplished. Endocardial cushion defects are closed at 32°C with the heart beating. This is of importance in monitoring possible bundle damage as sutures are placed. Such damage is more likely to occur in repair of endocardial cushion defects owing to the more variable course of the bundle below and behind the defect. Furthermore, adequacy of the repair of the associated mitral incompetence is more readily assessed.

Since this technic was adopted, 112 patients in the most hazardous group with tetralogy of Fallot, ventricular septal defect, or endocardial cushion defect have been subjected to operation. Only two patients showed permanent complete heart block; one died after 24 hours with other complications, and the second patient, who had tetralogy, still has complete block, but is otherwise active and well.

References

RIFOMYCIN SV IN CLINICAL PRACTICE

The author reports the first results obtained with the therapeutic use of rifomycin SV, a derivative of Streptomyces mediterranei. The applications have been carried out through different modalities (parenteral, topical in fistulae and empyemas, endobronchial and endocavitary instillations.) While in the use by systemic administration the results seem uncertain and often partial, local applications give in most cases very good results and under certain conditions bring about complete healing of the lesions.


SOME PROBLEMS ON PASSIVE TRANSFER OF TUBERCULIN SENSITIVITY

Recently, the role of lymphocytes has come into notice in their ability to transfer tumor immunity or homotransplantation immunity from immunized to normal animals as reported by Mitchison (1953) and Billingham (1954). Do lymphocytes take part in the cellular transfer of the tuberculin sensitivity as well as in these immunities, as they are quite similar to each other in the absence of antibodies in sera of sensitized animals? Fukase et al. (1953) have already reported the possibility of cellular transfer of the tuberculin sensitivity by lymphocytes from lymph nodes or thoracic lymph of sensitized animals. We could confirm this result in the present experiments, but also recognized that the ability of the lymphocyte was not superior to the other cells, for instance, to splenic cells, peritoneal exudate cells or lung cells of sensitized animals. It may be unreasonable to restrict the ability of the cellular transfer of tuberculin sensitivity to one type cell.


SHEDDING OF MUCOSA IN ASTHMA

The sputa of asthmatics often contain large compact clusters of columnar epithelial cells (Creola bodies) which are a manifestation of detachment of the mucosa of the lower respiratory tract. This detachment, as shown by these peculiar columnar cell clusters, is seen almost exclusively in asthmatics and increases considerably during asthmatic attacks. This loss of ciliated columnar epithelium may be of great magnitude. There is evidence that it hinders recovery from asthmatic attacks, and may be of great magnitude.


TRANSPOSITION OF THE CARDIA IN SURGICAL TREATMENT OF CARDIOSPASM

The authors describe a new method of surgical treatment for cardiospasm. This mode of therapy was employed in 12 patients with cicatrictial cardiospasm which was due to protracted cardiospasm. A transthoracic excision is made of the cicatrictially changed area with transposition of the cardia into the fundal section of the stomach through the newly formed orifice in the diaphragm. Excellent results were obtained. Followup observations ranged from three to eight years.