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SEPTAL DEFECTS

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INASMUCH as the clinical auricular septal defect is a static structural lesion, it would appear to be a type of disease which should be amenable to surgical repair. The malformation is apparently fairly common,1,2 and because of gradually progressive changes in hemodynamics, the life span of these patients is usually curtailed. Clinical attempt at closure of this defect has been reported.3 Lack of data on oxygen saturations in the right auricle before and after the attempted closure, however, make analysis of the effectiveness of the procedure impossible. This paper is a report of our experience with the creation and closure of auricular septal defects in the experimental animal.

THE CREATION OF THE DEFECT

For the purpose of studying methods of closure, it is desirable to create an experimental defect which lies as nearly as possible in the anatomic location of the common clinical lesion. Congenital septal defects occur in two separate areas having different embryologic origins as clearly described by Patten.4 In the first type, which occurs uncommonly, the defects are due to arrested development of the septum primum and lie in the extreme inferior portion of the septum directly overlaying the atrioventricular valves. In the second and by far the more common type, the defects result from arrest in the descent of the septum secundum coupled with abnormally extensive resorption of septum primum. This produces essentially a failure in the development of the superior margin of the foramen ovale and these defects thus extend from approximately mid-septum into the upper anterior quadrant. It is with the experimental creation of this second lesion that we are primarily concerned.

Our initial attempts to create a defect were made by inserting a narrow knife blade through the auricular wall and placing an elliptical incision blindly through the septum. We found it was quite possible to make the cut 1 or 2 cm. in length, but these incisions invariably healed rapidly, and three weeks later, inspection of the septa revealed them to have returned to an intact and essentially normal state. Recently, Martin and Essex5 have reported better success and an occasional lasting defect using this method.

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However, we concluded that it would be necessary to actually remove a segment of the septum if success were to be obtained. The initial approach was by means of a rotary punch applied blindly through the wall of the auricle using a purse-string suture. Because the septum is so flexible and because it is in constant motion, our success with this method was minimal. In one animal we managed to create a ragged defect about 1 cm. in diameter, which had begun to heal over when inspected at two weeks.

A rotating blade around the end of a cardioscope encountered the same difficulties. Although it was possible to identify visually the various landmarks in the auricle and on the septum, and thus the effort was directed more accurately at the proper anatomic area, the results were equally disappointing.

It appeared desirable, therefore, to attempt to create the lesion under direct vision. Early in 1948, we devised an instrument out of a standard type sponge forceps with which it was possible to approach the septum with a limited but bloodless field. By bending the blades of the forceps to form a circle one could compress the two walls of the auricle against the septum over an area of 1.5 cm. without embarrassing the action of the heart for periods of from four to as long as thirty minutes. Incision in the right auricular wall brought us directly down to the septum and a defect about 1 cm. in diameter, roughly circular in shape, could be made with the scissors or a small sharp knife. The animals tolerated this procedure well on the whole, but to our disappointment, we found that defects of this size, in spite of the fact that there had been loss of substance, uniformly tended to heal over spontaneously in a period of four to eight weeks. For this reason, we abandoned this method.

At about the same time Doderill in Detroit was developing quite independently a similar instrument. In his subsequent report, the illustrated defects, which originally were even larger than ours, appeared to be healing at one month and looked no bigger than the coronary sinus.

At about this time, August of 1948, Blalock and Hanlon published their ingenious method for the creation of a communication between the auricles. This shunt, however, is placed well posterior and superior on the wall of the right auricle and thus lies behind and above the usual anatomic site of the clinical defect. For our purposes, therefore, this procedure did not appear ideal; but if one desires to establish a permanent auricular shunt for experimental or therapeutic reasons, we believe this is the method of choice.

Accordingly, we turned to an attempt to excise the septum under direct vision with the auricle open and the blood flow temporarily occluded. This is a technique which was used by Templeton and Gibbons in their experiments on the tricuspid valve.

The chest is opened by an intercostal incision in the fourth interspace on the right side. The azygos vein is divided between ligatures near its entrance into the superior vena cava. The superior and inferior venae caveae are now mobilized and umbilical tapes are passed beneath them to serve as temporary ligatures. The pericardium is opened widely in a vertical direction anterior to the right phrenic nerve, and retracted by silk sutures. Two sutures are placed in the auricular wall to demarcate the ends of the proposed incision which is about 3 cm. in length, running vertically, with the lower end at
the point of junction of the inferior vena cava with the auricle and about 1 cm. anterior to the attachment of the pericardium. Two additional retraction sutures are placed about 2 mm. apart, straddling the midpoint of the proposed incision. One assistant now pulls up on the hemostatic umbilical tapes occluding the venae cavae. This is his only responsibility, and he maintains this control throughout the procedure until instructed to release by the operator. The two medial silk retention sutures are held by the first assistant. The operator makes the auricular incision with the scissors and then immediately sucks out the auricle with the sucker. There is a very small amount of blood entering the auricle from the coronary sinus. The landmarks are identified, particularly the coronary sinus. A point in the septum 1 cm. superior and 1 cm. posterior to the coronary sinus is grasped with the sharp arterial forceps, elevated, and excised with the scissors. A roughly circular piece of tissue about 2 cm. in diameter is removed. Surprisingly little blood emerges from the left auricle through this defect. It will be remembered that blood flow through the lesser circuit has been halted during the procedure and since the left auricle is dependent, the force acting to expel blood upward through the septal hole is minimal (Fig. 1).

Fig. 1.—Operative view of freshly created auricular septal defect. The venae cavae are temporarily occluded and the wall of the right auricle is open. The defect is superior and posterior to coronary sinus.

The chest cavity is now immediately flooded with saline solution. The operator pulls upward on all four auricular retention sutures controlling the extremities of the incision and the first assistant applies the curved spring clamp. The upward pull on the umbilical tapes is now released, and blood flow from the venae cavae, which has been obstructed between one and two minutes, is at once resumed. The incision in the auricle is now closed at leisure with evertting mattress sutures of 000 silk which effects an endocardium-to-endocardium closure. The spring clamp is removed, the pericardium closed, and the chest wound repaired. The animal's recovery is uneventful.
It is remarkable that the disturbance in cardiac rhythm is so slight. In no instance did either auricular or ventricular fibrillation supervene. Neither did any evidence of interference with the sinoauricular pacemaker occur. One pitfall of the operation is the possibility of exceeding the margins of the septum and cutting through the wall of the right auricle. Very accurate placement of the septal excision is essential.

We have observed two complications. In one animal death occurred two hours postoperatively. Autopsy revealed air emboli of the left coronary arteries. This is the only animal which had any symptoms of air embolus. In the experience of Martin and Essex with this technique, air embolism was common. It is possible that our method of immediately flooding the chest cavity with saline after the defect was cut was helpful in preventing this complication. In another, upon second operation for the purpose of repairing the defect, a firm intraluminal thrombosis was found in the right auricle adherent to the incision in the auricular wall.

The fate of septal defects created in this fashion is most extraordinary. In spite of the removal of the major portion of the septum, healing commonly occurs in a period of from two to three months, often with complete restoration of a functional septum. It would appear that the auricular septum has great powers to repair a traumatic defect, that careful endocardium-to-endocardium

Fig. 2.—View through right auricle of healed septal defect three months after creation. Arrow points to scar in septum; small pledget is in coronary sinus.
Coaptation is essential if the defect is to persist (as in the Blalock-Hanlon procedure), and that blind or open surgical incisions or excisions of septum will not consistently remain patent (Fig. 2).

However, for the purposes of experimental closure a preparation was accomplished which simulated the clinical lesion, if closure was attempted within a month of the creation of the defect.

**Closure of the Defect**

In 1947, Cohn described an experimental method for the closure of small auricular septal defects in the dog. The lateral wall of the right auricle was pushed against the septum and two sutures were placed to hold it in this position. A wire snare was threaded into the auricle around this portion of wall, and the button-shaped section attached to the septum was cut free, allowing the rest of the wall to revert to normal position. The hole in the auricle left by this maneuver was sutured closed.

This ingenious operation seemed to us to have two potential dangers. During the period of initial invagination of the wall, blood flow from the venae cavae was disturbed and cardiac irregularities were commonly encountered. Second, after excision of the segment of wall, the surface exposed to the internal blood flow in the right auricle was the external surface of the myocardium; it was not lined with a smooth endocardial layer. It seems likely that the factors most suitable for the initiation of intra-auricular thrombosis were thus established. If a method utilizing auricle wall could be devised which did not interfere even temporarily with blood flow and which left exposed to the blood stream only endothelialized surfaces these potential dangers might be eliminated. Such a method is here presented.

The fundamental concept of this technique is to invaginate both auricular appendages and to fasten their tips firmly together through the septal defect. This converts the lumen of the auricle to a hollow space eccentrically "doughnut" in shape, entirely lined with endothelium. No interference with blood flow is encountered. The procedure is as follows:

Under intravenous nembutal anesthesia, using intermittent positive pressure endotracheal oxygen, the animal is placed supine on the operating table and both sides of the chest are prepared and draped. Bilateral anterior intercostal thoracotomies are placed in the fourth interspace, and rib retractors inserted. Eight cubic centimeters of 2 per cent procaine are injected into the pericardium. Attention is first directed to the left side. A T-shaped incision is made in the pericardium over the tip of the left auricle lateral and posterior to the emergence of pulmonary artery. Left auricular tip is identified. A moist drape is now placed over the wound and activity is shifted to the right side. A vertical incision is made in the pericardium the length of the auricle, just anterior to the phrenic nerve. The right auricular tip is seized with the toothed forceps and a purse-string suture of 000 silk is placed at the very apex. The eye end of a standard surgical probe is thrust through the loop into the auricle and the purse string is tightened to effect hemostasis. The bent probe is advanced by feel into the inferior vena cava. It is then drawn slowly upward feeling with the tip along the septum seeking the defect. The coronary sinus may be entered, in which case the probe can be felt in the atrioventricular groove posteriorly. From here the defect lies superior and posterior. If the tricuspid valve is entered, the probe is too far anterior. Usually only one or two tries are sufficient to pass
the probe through the defect into the left auricle. Care must be taken that it does not progress out one of the left pulmonary veins. The left incision is now inspected and by feel and direct vision the tip of the probe is manipulated until it lies in the very apex of the left appendage. It is pushed through the muscle at that point (Fig. 3).

The first assistant now rapidly threads the eye of the probe with the two ends of a strand of No. 1 silk on which has previously been threaded a circular button of stiff Polythene, 1½ to 2 cm. in diameter. A piece of Gelfoam lines the inner side of the button. The probe is now entirely withdrawn carrying with it the silk strand. This is pulled up to snug the Gelfoam against the left tip and to start the inversion of the left appendage. A similar piece of Gelfoam and Polythene button is now threaded onto the ends of the silk, the first throw of a surgeon’s knot is placed, and the throw gradually but firmly tightened until it is quite snug. This maneuver inexorably draws the inverted tips of the auricles toward each other until they abut through the septal defect. The two buttons can be identified by feel as having been brought into close apposition in the midline. The knot is finished and the ends cut.

This entire maneuver can be accomplished with almost no loss of blood, and has not been accompanied by any severe disturbance in cardiac rhythm. The heart does not need to be displaced, and no interference with blood flow is encountered at any time. It remains only to repair the pericardium, expand the lungs, and close the incisions in the usual fashion.

A series of ten dogs with artificially created defects have been closed using this technique. The condensed operative protocols of these animals are seen in Table I.
TABLE I. CONDENSED PROTOCOLS OF CLOSURE OF AURICULAR SEPTAL DEFECT

<table>
<thead>
<tr>
<th>DOG NUMBER</th>
<th>DATE DEFECT CREATED (1949)</th>
<th>DATE DEFECT CLOSED (1949)</th>
<th>REMARKS</th>
</tr>
</thead>
<tbody>
<tr>
<td>125</td>
<td>March 15</td>
<td>April 8</td>
<td>Killed Jan. 18, 1950. A 2 cm. defect approximately one-half closed by repair. Remainder of defect closed by small organized thrombus.</td>
</tr>
<tr>
<td>141</td>
<td>April 15</td>
<td>May 11</td>
<td>Sacrificed Sept. 27. An 8 mm. defect was approximately two-thirds closed by repair.</td>
</tr>
<tr>
<td>145</td>
<td>April 22</td>
<td>May 9</td>
<td>Dog still alive and healthy. Normal ECG, and normal cardiac catheterization.</td>
</tr>
<tr>
<td>147</td>
<td>April 25</td>
<td>May 25</td>
<td>Sacrificed Dec. 20. A 1 cm. defect was two-thirds closed by repair.</td>
</tr>
<tr>
<td>148</td>
<td>April 27</td>
<td>May 31</td>
<td>Died 8 hrs. postrepair of bilateral atelectasis. A 6 mm. defect was totally closed by repair.</td>
</tr>
<tr>
<td>149</td>
<td>May 2</td>
<td>June 1</td>
<td>At second operation, a firm clot felt in right atrium. Dog died June 10, of empyema. The 8 mm. defect was totally closed. An organized thrombosis was found in right atrium.</td>
</tr>
<tr>
<td>161</td>
<td>June 6</td>
<td>June 28</td>
<td>Sacrificed Dec. 20. A 1 cm. defect was completely closed by repair. Heart appeared normal in size.</td>
</tr>
<tr>
<td>164</td>
<td>June 8</td>
<td>June 30</td>
<td>Dog still alive and healthy.</td>
</tr>
<tr>
<td>185</td>
<td>July 8</td>
<td>July 27</td>
<td>Sacrificed Sept. 24. The 9 mm. defect was completely closed by repair.</td>
</tr>
<tr>
<td>211</td>
<td>Aug. 3</td>
<td>Aug. 25</td>
<td>Dog died on table during skin closure. Cause of death not identified. A 6 mm. defect was well closed by the repair.</td>
</tr>
</tbody>
</table>

DISCUSSION

The effect on the cardiovascular hemodynamics of the creation of these septal defects is usually quite minimal within the period of time which they have been observed. The dogs remain quite active and apparently healthy. Occasionally, at second operation the right auricle appears moderately enlarged. The left auricle appears normal.

Cardiac catheterization studies were performed on the animals to evaluate changes in hemodynamics occurring after creation of the defect and after the subsequent repair. An increase of 1.0 volume per cent or more in the oxygen content between the superior vena cava and the right ventricle was chosen as the minimal criteria necessary to suggest flow of oxygenated blood through the septal defect. Table II is a summary of the catheterization studies. It will be seen that in most instances, the status of the auricular septum was accurately reflected by the oxygen saturation studies; but in four animals (Group 3) the

TABLE II. CARDIAC CATHETERIZATION STUDIES

A. Creation of Defect

1. Abnormal blood flow; defect No. 125, 145, 147, 148, 161
   - Catheter revealed status
2. No abnormal blood flow; no defect present
   - Catheter failed to reveal status
3. No abnormal blood flow; defect No. 117, 149, 185, 211
   - Catheter revealed status

B. Repair of Defect

4. Returned to normal blood flow No. 125, 145, 147, 161
5. Did not return to normal flow None
catheterization failed to reveal a defect which was subsequently proved to exist. Apparently, at the time of the study, there was no significant left-to-right flow of blood through the defect. It is probable that in the early stage of this abnormality, blood flow may be variable.

What is the functional efficiency of the method of closure? In the four animals which had evidence of abnormal blood flow before repair, catheter studies following repair revealed a return to normal hemodynamics. This data is presented in Table III. We think the evidence is suggestive that abnormal blood flow through the experimental defect is effectively prevented by the operation.

Fig. 4.—Seen from above, the inverted auricular tips abut through the septal defect. Autopsy specimen obtained from Dog No. 161, six months following repair, shows smooth endothelial surfaces, lack of thrombosis, and effective closure of defect.

The bilateral thoracotomy was well tolerated by the animals. In only one (No. 148) was atelectasis a significant complication, and it is probable that we were careless in re-expanding the lungs in this animal at the close of the operation.

Intra-auricular thrombosis occurred in only one instance, and this was apparently a result of the creation of the defect rather than of the repair, since it was felt to be present at the second operation. Thrombosis, however, might well occur if auricular rhythm were disordered. This complication did not occur in any of the animals in this series. It is possible that the enlarged, dis-
Table III. Effect of Closure of Septal Defect on Catheterization Findings
(Group 4, Table I)

<table>
<thead>
<tr>
<th>DOG NUMBER</th>
<th>VOL. PER CENT O₂ SATURATION (BEFORE CLOSURE)</th>
<th>VOL. PER CENT O₂ SATURATION (AFTER CLOSURE)</th>
<th>O₂ SAT. DIFF. S.V.C. TO R.V.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SUPERIOR VENA CAVA (1)</td>
<td>RIGHT AURICLE (2)</td>
<td>RIGHT VEN-TRICLE</td>
</tr>
<tr>
<td>125</td>
<td>8.5</td>
<td>8.1</td>
<td>9.1</td>
</tr>
<tr>
<td>145</td>
<td>7.3</td>
<td>8.0</td>
<td>7.3</td>
</tr>
<tr>
<td>147</td>
<td>9.5</td>
<td>11.2</td>
<td>10.9</td>
</tr>
<tr>
<td>161</td>
<td>7.1</td>
<td>7.0</td>
<td>8.0</td>
</tr>
</tbody>
</table>

tended auricles of the clinical patient would not react so kindly to this manipulation as did the auricles of these essentially normal dogs.

There was no evidence of any interference with blood flow or with auricular function by the inverted auricular tips. This is apparently because anatomically the inversion occurs in the upper anterior quadrant of the lumen of the auricle, while the blood flow occurs primarily in the posterior and inferior quadrants. In addition, the intraluminal auricular pressure apparently compresses the inverted appendage so that its diameter, as it traverses the auricle, is usually quite small, much less than the size of the button at its tip (Fig. 4).

In some of the specimens, the edge of the defect had healed firmly to the auricular appendage passing through it either totally or in part. It is possible that this is related to the traumatic origin of the defect and would not occur in defects which were congenital.

SUMMARY

1. A method for the creation under direct vision of an auricular septal defect in dogs, which simulates anatomically the congenital clinical defect, is described.

2. These defects have a strong tendency to spontaneous closure by healing.

3. An operation for the anatomical and functional obliteration of these experimental defects by inversion of the auricular appendages has been devised which is well tolerated by the animals.

4. The effectiveness of this procedure in reverting abnormal blood flow to normal is suggested by cardiac catheterization studies.

REFERENCES