Endocarditis after Intracardiac Surgery

DONALD R. CARTER, M.D., J. A. BARNEY, M.D., JERRY LEU, M.D., WILLIAM R. HANNA, M.D., and G. RAINNEY WILLIAMS, M.D., Oklahoma City, Oklahoma

From the Department of Surgery at the University of Oklahoma Medical Center, Oklahoma City, Oklahoma. This investigation was supported in part by U. S. Public Health Service Grant H-4293 and in part by the John and Mary R. Markle Foundation.

Bacterial endocarditis is a significant complication of intracardiac surgical procedures [1-3]. The obvious opportunities for contamination during operations involving cardiopulmonary bypass, the common occurrence of fever in the postoperative period, and the high mortality associated with proved infections are reasons for interest in this complication. The following experiments were designed to supply information about the duration of increased susceptibility to infection after intracardiac operations.

MATERIAL AND METHODS

Mongrel dogs of both sexes weighing 10 to 20 kg. were immunized against canine hepatitis and distemper, and treated with anthelmintics. All animals were subjected to a ten day control period prior to operation, during which time daily weights and temperatures were recorded. Blood cultures were taken and white blood cell counts determined on alternate days. Animals appearing ill and those with positive cultures of the blood or leukocytosis were excluded. No antibiotics were used at any time during the experiments.

A pure strain of Streptococcus mitis No. 6249, obtained from the American Type Culture Collection, was used in all experiments. Each inoculum contained approximately 5 billion cells obtained by a standard method.

Operations were performed under sterile operating room conditions using sodium pentobarbital anesthesia, endotracheal intubation, and a piston type respirator.

The experiment was divided into three groups. Five dogs comprised group I; these animals served as controls and after the standard control period were injected with the standard Strep. mitis inoculum.

The dogs in group II received the standard bacterial inoculum one hour after completion of an intracardiac procedure; group III is referred to as the acute group in reference to the time of bacterial injection as related to the operative procedure.

The dogs in group III underwent a six week period between operation and injection of the bacterial inoculum; they also underwent the standard preoperative control period. In addition, for ten days prior to injection of the bacterial inoculum, these dogs had determinations of weight, temperature, and white blood cell count as well as blood cultures taken on alternate days. The dogs in group III are referred to as the chronic group in reference to the time of bacterial injection as related to the operative procedure.

After bacterial injection, dogs in all three groups were followed up with determinations of weight, temperature, white blood cell counts, and blood cultures daily for one week, and similar determinations on alternate days during the second week post injection.

Two weeks after bacterial injection, each animal was sacrificed. At autopsy, gross and microscopic examinations of the heart, lungs, liver, spleen, and kidneys were carried out and cultures were taken from all operative sites as well as from any suspicious lesions. Tissue sections of intracardiac lesions were stained with Brown-Brenn bacterial stain and hematoxylin and eosin stain.

Table I summarizes the operative procedures performed and the time of bacterial injection.

The aortic valve injury was produced by passing a semirigid plastic rod through a carotid artery and forcibly tearing or perforating an aortic cusp. Arterial pressure in the opposite carotid artery was recorded and a definite increase in pulse pressure was considered to indicate aortic valve injury.

Pulmonic valve injury was produced by excising a pulmonic valve cusp under direct vision via the pulmonary artery under inflow occlusion.

* Presented at the Nineteenth Annual Meeting of the Southwestern Surgical Congress, Phoenix, Arizona, April 10-13, 1967.
TABLE I
RESULTS OF EXPERIMENTS IN THE PRODUCTION OF BACTERIAL ENDOCARDITIS

<table>
<thead>
<tr>
<th>Group</th>
<th>Operation</th>
<th>No. of Dogs</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>I (Control)</td>
<td>Injury to aortic valve</td>
<td>5</td>
<td>No evidence of infection</td>
</tr>
<tr>
<td>II (Acute)*</td>
<td>Right ventriculotomy using continuous silk suture for closure</td>
<td>3</td>
<td>3 Infections</td>
</tr>
<tr>
<td></td>
<td>Placement of compressed Ivalon patch in right ventricular outflow tract using silk suture</td>
<td>5</td>
<td>1 Infection</td>
</tr>
<tr>
<td></td>
<td>Placement of an autologous pericardial patch in right ventricular outflow tract using chronic catgut</td>
<td>7</td>
<td>7 Infections</td>
</tr>
<tr>
<td></td>
<td>Injury to the pulmonic valve</td>
<td>4</td>
<td>3 Infections</td>
</tr>
<tr>
<td>III (Chronic)†</td>
<td>Placement of compressed Ivalon patch in right ventricular outflow tract using silk suture</td>
<td>4</td>
<td>4 Infections</td>
</tr>
<tr>
<td></td>
<td>Placement of autologous pericardial patch in right ventricular outflow tract using chronic catgut</td>
<td>4</td>
<td>1 Infection</td>
</tr>
<tr>
<td></td>
<td>Injury to the pulmonic valve</td>
<td>5</td>
<td>No infections</td>
</tr>
</tbody>
</table>

* Injection of 5 billion cells of Strep. mitis one hour after operation.
† Injection of 5 billion cells of Strep. mitis six weeks after operation.

The compressed Ivalon® and autologous pericardial patches were sutured into right ventriculotomy incisions using cardiopulmonary bypass. Bypass was accomplished using a rotating disc oxygenator primed with 5 per cent dextrose in water or blood. Body temperature was lowered with a Harrison-Brown heat exchanger prior to total bypass.

The right ventriculotomy was performed under inflow occlusion.

RESULTS

The results of all three experiments are shown in Table I.

Group I (Control). The five dogs not operated upon showed few ill effects after bacterial injection. Anorexia persisted for one to two days and low grade fever and mild leukocytosis were universal. All animals appeared well after the third day and the latest a positive blood culture was obtained was at twenty-four hours in a single animal; the remaining cultures became negative after one day. At autopsy there was no gross or microscopic evidence of infection. The responses in the control dogs were typical of the response in all dogs in the chronic group.

Group II (Acute). Clinical and laboratory evidence of endocarditis was found in all three animals surviving injury to the aortic valve. A murmur was heard in the immediate postoperative period and increased in intensity during the fourteen day observation period. The animals quickly became severely ill clinically. At autopsy vegetative lesions were present on the aortic valve in all animals, involving a single cusp in one animal and all three cusps in the other two animals. In addition, in one animal there was a vegetative lesion on the aortic cusp of the mitral valve. Cultures were consistently positive for Strep. mitis. No lesions other than mild splenomegaly were recognized at autopsy.

In four animals undergoing right ventriculotomy and closure a smooth endocardial surface with a well healed suture line was noted at autopsy. In the fifth a small thrombus was attached to the endocardial surface which demonstrated gram-positive cocci on microscopic examination and from which Strep. mitis was cultured. No other lesions were noted.

Of seven animals with insertion of a right ventricular Ivalon patch, only four survived as long as two weeks. Two animals died of pneumonia during the first postoperative week, and in one of these dogs Strep. mitis appeared to be the etiologic organism. One dog died suddenly on the tenth postoperative day, and at autopsy was found to have bleeding from a disrupted, grossly infected suture line around the ventricular patch. The remaining four animals were sacrificed at the end of two weeks and all had gross and microscopic evidence of infection; cultures were positive for Strep. mitis in all seven animals.

In three of four dogs with pericardial patches sutured with chronic catgut into the right ventricular outflow tract bacterial endocarditis developed positive, time of at the in the re healed with.

In all after inj et endocard fourth posently inj in the r seen at a typical of Strep. m Group excision to the pos inj enter als in the pul and fibre were pr were ste.

In for patches endocarditis of the in three se the the ep area of mitis.

Neit terial with a ventri was q area o the to be.

The chron

American Journal of Surgery

Vol. 1
Endocarditis after Intracardiac Surgery

Results

No evidence of infection
Infections
Infections
Infections
No infections
No infections

were present on the aortic cusps in the aortic cusps were consistently less lesions other than those at autopsy. The right ventricularg on the epicardial surface of the patch appeared smoothly endothelialized in three instances. In the fourth animal, a small abscess containing purulent material which cultured Strep. mitis was encountered between the epicardium and adherent pericardium and an area of granulation tissue on the inner surface of the patch also was positive for Strep. mitis. No other lesions were encountered. Neither clinical nor autopsy evidence of bacterial endocarditis developed in four animals with autologous pericardial patches in the right ventricular wall. The shrinkage of these patches was quite striking and frequently the entire area of the ventriculotomy and patch appeared to be a contracted scar.

The total results in the control, acute, and chronic groups are summarized in Table 1.

COMMENTS

The intact cardiovascular system is quite resistant to the establishment of bacterial endocarditis. Alterations in the cardiovascular system have a marked influence on susceptibility to infection. The presence of foreign body, valve injury, and assorted conditions which give rise to changes in flow (septal defects, arteriovenous fistulas, and the like) have been shown both clinically and experimentally to be associated with an increased susceptibility to bacterial infection [4-7]. Since any intracardiac operation of necessity leaves some intracardiac defect, it seems important to determine how long the increase in susceptibility to bacterial endocarditis, which has been demonstrated to be present in the immediate postoperative period, may persist [8]. Available clinical reports would suggest that although chronic disturbance of valve function predisposes to bacterial endocarditis, this susceptibility is particularly marked in the immediate postoperative period.

Of interest is the possibility that open heart bypass and whole body perfusion may produce a temporary blockade of the reticuloendothelial system or alter the production or concentration of heat stable or heat labile serum factors (opsonins) that are necessary for phagocytosis at the site of injury. This could predispose the local area of injury, the heart valves, to sepsis during the early postoperative course, and not during the later postoperative period with chronic valvular damage [9].

There is little clinical experience indicating that bacterial endocarditis is a late complication after closure of septal defects or ventriculotomy, when no permanent valve dysfunction is left. Because the effects of dosage and virulence of bacteria cannot be controlled in the clinical situation, the experiments described herein were carried out. The results appear to indicate that there is lessening of the susceptibility to infection previously demonstrated to be present in the immediate postoperative period. The Ivalon patches in the right ventricle were found to be completely healed at the end of four weeks, and it seems likely that these no longer behave as a foreign body after this period of time. The same is true of the pericardial patches. An additional interesting observation is the marked shrinkage observed in the pericardial patches. It seems likely that, if the pericardial patch had been used initially to increase ventricular outflow, the immediate result would have been negated by shrinkage. The failure to produce bacterial endocarditis in the dogs with chronic injury to the pulmonic valve is perhaps surprising. It may be that the virulence and dosage of the bacteria were not sufficient to produce endocarditis in the presence of the defect with this degree of influence on local susceptibility. It should also be stated that there is no evidence of real insufficiency other than the...
presence of murmur and the grossly demonstrable injury at the time of autopsy.

SUMMARY

Intravenous injection of 5 billion viable bacteria of the strain Strep. mitis six weeks after intracardiac operations failed to result in development of bacterial endocarditis, whereas injection in the immediate postoperative period resulted in a high incidence of bacterial endocarditis.

Autologous pericardial patches utilized in this study demonstrated marked scarring and contracture during the six week period of observation.

REFERENCES