Bacterial Endocarditis Following Cardiac Surgery

By Clarence Denton, M.D., Elias G. Pappas, M.D., Joseph F. Uricchio, M.D., Harry Goldberg, M.D., and William Likoff, M.D.

Intracardiac surgery for rheumatic and congenital heart disease entails direct trauma to both normal and abnormal endocardium. This communication inquires into the incidence and nature of the endocardial infections that develop subsequent to this injury. On the basis of an examination of 2,263 patients operated upon for acquired and congenital heart disease during a 5-year period terminating in November 1955, bacterial endocarditis appears to be an infrequent complication of surgery, is caused by organisms not so commonly encountered in unoperated patients, and is characterized by a clinical pattern quite different from that ordinarily associated with bacterial endocarditis. The rate of attrition in this group of patients is high, and unquestionably is related to the antibiotic resistance of the unusual organisms and the severity of the basic heart disease.

In the era before cardiac surgery, bacterial endocarditis generally involved endocardial or endothelial structures rendered abnormal by disease or congenital variation, although occasionally it arose on normal structures and pursued a more virulent course. Surgery on the interior of the heart produces a type of acute endocardial injury, both in normal portions of the endocardium and in the area of chronic disease to which the surgery is directed. It should, therefore, not be surprising to find instances of infection of the endocardium after cardiac operations. However, with the advance of cardiac surgery, the occurrence of bacterial endocarditis has not been frequently reported.

Nevertheless, it has been a tragic, if uncommon, event in a large series of surgical cases. It was, therefore, considered worthwhile to review our experience with bacterial endocarditis following such surgery, and to compare these cases with the much larger group undergoing similar operations in the last 5 years, but without this complication.

According to White's estimate, before the application of surgery to heart disease, subacute bacterial endocarditis made up 1 to 2 per cent of all types of cardiac disease. The incidence was noted to be quite high in the age group 20 to 30, and rheumatic valvular disease was stressed as the background for the development of bacterial endocarditis. While pure mitral stenosis is rather rarely the underlying lesion, a combination of mitral stenosis and insufficiency is quite frequent. In the series of 408 cases of Gates and Christie, the mitral valve alone was affected in 169 cases, the aortic valve solely in 49 instances, both aortic and mitral valves in 145 cases, while in 45 congenital heart disease was the basic lesion. The occurrence of tricuspid or pulmonary valve infection is rare. When the right side of the heart is affected, it is almost always in association with a left-sided endocarditis or with a congenital abnormality.

In the era prior to cardiac surgery, the importance of the Streptococcus viridans was evident. Christian reported 150 cases due to this organism, 4 to Staphylococcus albus, 2 to unidentified staphylococci, and 1 to a pleomorphic bacillus. Morrison and Middleton gave similar, if not quite so overwhelming figures.

With the advent of cardiac surgery, reports of bacterial endocarditis have appeared, first in patients with congenital defects, later in patients operated upon for acquired valvular heart disease. In a report of 1,000 patients operated upon for the tetralogy of Fallot, by systemic-pulmonary anastomosis, Taussig and associates stated that 17 of 844 who survived surgery developed bacterial endocarditis after surgery, and that 4 died of this complication. The onset of infection in 11 patients was soon after surgery; in 6 others, more than 2 months afterwards. They assumed that the infection occurred at the site of anastomosis, but autopsy was performed in only 2 late cases, and the infection was not proved in 1. In the other case,
nothing was stated about the site of the infection. They implied that the higher incidence in the early postoperative period was due to infection at the line of suture before it had healed, but stated that the risk of illness after 2 months was no greater than in unoperated patients. Two case reports9, 10 with autopsy findings, 2 and 2½ years, respectively, after Blalock operations, showed no infection of the artificial ductus, but involvement of the pulmonary and tricuspid valves and a mycotic aneurysm of the pulmonary artery.

Of 273 cases operated on for patent ductus arteriosus, reviewed by Scott,11 only 1 developed bacterial endocarditis, the febrile episodes appearing 6 months after surgery. Autopsy revealed an aneurysm of the ductus and recanalization with infection.

A few case reports have appeared describing infection following mitral commissurotomy, and all of these, where the organism was recovered, were due to penicillin-resistant staphylococci,14-18 except 1 that was due to a pseudomonas.12 In 1 instance all cultures were sterile.13 The interval between surgery and the onset of the illness was reported as 7 to 21 days in most of these, but 1 case of Dalton, Williams, and Atkins16 occurred 2 months, another 3½ months after operation. These authors are the only ones reporting incidence in a large surgical group (3 cases in 150 operations). In the 7 cases reported by these various authors, only 2 recovered. Vegetations on the mitral valve were found at autopsy in 4 of the other 5 cases.

**Material**

Cases of valvular heart disease subjected to cardiac surgery in the 5 years prior to November 1,

### Table 1.—Clinical and Bacteriologic Data in Twenty Cases of Bacterial Endocarditis

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age range</th>
<th>Heart rhythm</th>
<th>Valve calcification</th>
<th>Valve lesion or congenital defect</th>
<th>Operation</th>
<th>Interval after surgery</th>
<th>Organism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>14</td>
<td>30-51</td>
<td>11 NSR* 3 AF</td>
<td>10 yes 1 not determined</td>
<td>6 TAAC+ 2 TAAC + NP NAAL 1 NY 1 MC 1 MC and TAAC 1 MC and TVAC 1 MSu and MC 1 TVPT 6 TAAC + 2 TAAC and NP</td>
<td>4 10 days or less 4 More than 10 days but less than 3 months 5 3 months or more 1 Not known 2 10 days or less 2 10 days but less than 3 months</td>
<td>7 CNS§ 1 CPS 2 SV 1 NRS 1 ? 2 CNS 2 CPS 2 SCND</td>
</tr>
<tr>
<td>Female</td>
<td>6</td>
<td>27-51</td>
<td>5 NSR 1 AF 4 no 1 not determined</td>
<td>2 MI (both with ms) 3 MS 1 ASD 3 MC 1 ASP</td>
<td>1 10 days or less 2 More than 10 days but less than 3 months 3 3 months or more</td>
<td>2 10 days or less 2 10 days but less than 3 months 3 3 months or more</td>
<td>2 CNS 2 CPS 2 SCND</td>
</tr>
</tbody>
</table>

* NSR—normal sinus rhythm; AF—atrial fibrillation.
† AS—major aortic stenosis; as—minor aortic stenosis; MS—major mitral stenosis; ms—minor mitral stenosis; AI—major aortic regurgitation; ai—minor aortic regurgitation; MI—major mitral regurgitation; mi—minor mitral regurgitation; ASD—atrial septal defect.
‡ TAAC—Transaortic commissurotomy; TVAC—Transventricular aortic commissurotomy; NAAL—Nylon aortic annulus ligature; NP—Nylon peduncule; MC—Mitral commissurotomy; MSu—Mitral suture; TVPT—Transventricular pericardial tamponade for MI; ASP—atrioseptoplasty.
§ SV—Streptococcus viridans; EC—Enterococcus; NHS—Nonhemolytic streptococcus; CNS—Coagulase negative staphylococcus; CPS—Coagulase positive staphylococcus; SCND—Staphylococcus coagulase not determined; ?—Not known, lesion found at autopsy.
1955, were reviewed. Of 1,889 cases, 1,159 were pure mitral stenosis, 155 pure aortic stenosis, 142 major mitral regurgitation, 33 major aortic regurgitation, and 119 combinations of aortic and mitral stenosis. The remaining 281 were varying combinations of stenosis and regurgitation at 1 or more valves. In addition, 374 cases of congenital heart disease were operated upon. The usual clinical and bacteriologic criteria for bacterial endocarditis were fulfilled by all cases included in this report, except 1 discovered at autopsy. The interval between surgery and the onset of infection was considered to be the time elapsed between the date of operation and the onset of the fever during which positive blood cultures were obtained.

RESULTS

The pertinent clinical and bacteriologic findings are summarized in table 1. Altogether, 20 cases of bacterial endocarditis occurred prior to November 1955. The aortic valve was the one most frequently the site of prior disease in the patients who developed this infection.

Calcification was commonly present in the whole group, especially in the aortic valves. The operative procedures performed were mostly those designed to correct the aortic valvular lesion, either stenosis or regurgitation or both. The cases were about evenly divided, as to the postsurgical interval before the clinical appearance of the endocarditis, among an early period (less than 10 days), an intermediate period (10 days to 3 months), and a late period (3 to 10 months). The most important infecting organism was the staphylococcus, generally the coagulase-negative variety.

Information about the sensitivity of the organism to antibiotics was available in 15 of the 20 cases (table 2). In general the staphylococci were resistant to penicillin and showed the greatest sensitivity to carbomycin (Magnamycin) and chloramphenicol (Chloromycetin). The streptococcus viridans and the enteric

<table>
<thead>
<tr>
<th>Organism</th>
<th>No. tested</th>
<th>Penicillin</th>
<th>Streptomycin</th>
<th>Magnamycin</th>
<th>Chloromycetin</th>
<th>Terramycin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staphylococcus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coagulase positive</td>
<td>3</td>
<td>3 Resistant</td>
<td>2 Resistant</td>
<td>2 Resistant</td>
<td>2 Resistant</td>
<td>2 Resistant</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 Sensitive</td>
<td>1 Sensitive</td>
<td>1 Not</td>
<td>1 Sensitive</td>
<td>1 Sensitive</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>determined</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coagulase negative</td>
<td>9</td>
<td>6 Resistant</td>
<td>6 Resistant</td>
<td>1 Resistant</td>
<td>2 Resistant</td>
<td>2 Resistant</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 Moderatelysensitive</td>
<td>2 Moderately sensitive</td>
<td>1 Moderately sensitive</td>
<td>6 Sensitive</td>
<td>2 Resistant</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 Sensitive</td>
<td>1 Sensitive</td>
<td>1 Not</td>
<td>5 Sensitive</td>
<td>2 Resistant</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>determined</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coagulase not determined</td>
<td>1</td>
<td>1 Sensitive</td>
<td>1 Resistant</td>
<td>1 Not</td>
<td>1 Resistant</td>
<td>1 Resistant</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>determined</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Streptococcus</td>
<td>2</td>
<td>2 Resistant</td>
<td>2 Resistant</td>
<td>1 Not</td>
<td>1 Sensitive</td>
<td>2 Resistant</td>
</tr>
<tr>
<td>(enterococcus)</td>
<td></td>
<td></td>
<td></td>
<td>determined</td>
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<td></td>
</tr>
<tr>
<td>(S. viridans)</td>
<td></td>
<td></td>
<td></td>
<td>1 Sensitive</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Sensitivity was determined on blood agar plates with filter paper disks saturated in antibiotic solutions.

Penicillin, 50 U./ml.: Sensitive: Zone of inhibition greater than 20 mm. diameter
Moderately sensitive: Zone of inhibition 15 to 20 mm. diameter
Resistant: Zone of inhibition less than 10 mm. diameter

Streptomycin, Magnamycin, Chloromycetin, Terramycin, 500 U./ml. each: Sensitive: Zone of inhibition greater than 15 mm. diameter
Moderately sensitive: Zone of inhibition 10 to 15 mm. diameter
Resistant: Zone of inhibition less than 10 mm. diameter
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coccus showed the expected sensitivity and resistance respectively to penicillin.

Information concerning the therapy was available in 17 patients (table 3). It was not practical to analyze the dosage, duration, or method of administration in this small series, because of their great variation. Most of the patients were seriously ill, and changes were made in all aspects of therapy in accordance with the clinical response of the patient, rather than any arbitrary rules of management. For this same reason a number of patients were treated with a considerable variety of antibiotics.

Table 3 also shows the over-all mortality in relation to the infecting organism. It is not always possible to be sure that any given patient died as the result of his infection. One patient (case 7), who was free of fever for nearly 18 months and died of heart failure, might be included in the recovered category, particularly since it is believed that he could well have died of heart failure at this time even if he had never had bacteremia. One patient (case 19), who appeared clinically cured when last seen and who died 6 months later of an unknown cause, is listed under those dead of the disease. One of those listed as dead (case 13) was reported at autopsy in another hospital to have miliary tuberculosis, with tubercle bacilli and staphylococci clustered together on the strip of pericardium used to suture the mitral valve; it is doubtful that she died of bacterial endocarditis as such. A fourth patient (case 3) appeared to recover, but had a febrile relapse 2 months after the end of therapy to the time of his death. He succumbed while undergoing surgery for an infected retroperitoneal hematoma, diagnosed clinically as a mycotic aneurysm of the left iliac artery.

In general, it is apparent that the mortality rate is high for the coagulase-negative staphylococci, less so for the other organisms.

Five of these patients came to autopsy at our institution. The aortic valve was the only site of the endocarditis in 3 cases (2, 5, and 18) and the mitral valve in 1 case (13). Both the aortic and tricuspid valves were involved in case 20.

The cardiac rhythm at the time the infection appeared was normal in 16 cases and atrial fibrillation in 4. This incidence is undoubtedly partly the result of the preponderance of aortic lesions in this group of 20 cases. It may also be a reflection of the old dictum that bacterial endocarditis is uncommon in atrial fibrillation.

**DISCUSSION**

The well-known tendency of bacterial endocarditis to seek out the aortic valve in nonsurgical cases is also manifest in the surgical

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**Table 3.—Therapy and Results**

<table>
<thead>
<tr>
<th>Organism</th>
<th>Penicillin</th>
<th>Sulfadiazine</th>
<th>Chloramphenicol</th>
<th>Aureomycin</th>
<th>Ther. not known</th>
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<tr>
<td>Staphylococcus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coagulase positive (3 cases)</td>
<td>20</td>
<td>15</td>
<td>16</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Coagulase negative (9 cases)</td>
<td>2*</td>
<td>2</td>
<td>2</td>
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</tr>
<tr>
<td></td>
<td>3#</td>
<td>3</td>
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<td>5</td>
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<td>7**</td>
<td>7</td>
<td>7</td>
<td>7</td>
<td>6</td>
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<td>10</td>
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<td>14</td>
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<td></td>
<td>18</td>
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<td></td>
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<tr>
<td></td>
<td>19†‡</td>
<td>19</td>
<td>19</td>
<td>19</td>
<td>13‡</td>
</tr>
<tr>
<td>Staphylococcus</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Streptococcus</td>
<td>8</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(5 cases)</td>
<td>11</td>
<td>11</td>
<td></td>
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<td></td>
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<tr>
<td></td>
<td>17</td>
<td>17</td>
<td></td>
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<tr>
<td></td>
<td>17</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Organism not known (1 case)</td>
<td>12§</td>
<td>12</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Numbers refer to the case number. Those in **bold type** represent survivors.

* Also Bacitracin.
† Also Erythromycin and Achromycin.
‡ Also sulfadiazine and Erythromycin.
§ Autopsy showed endocarditis.
∥ Autopsy also showed miliary tuberculosis.
* Apparent recovery; death from relapse with mycotic iliac aneurysm.
° Recovery from endocarditis; died 18 months later of heart failure.
group here reported. Thus, of the 150 cases of pure or major aortic stenosis, 6 developed this disease. Even more striking is the incidence in surgically treated cases of aortic regurgitation, 4 of 33 developing infection. This incidence may be related to the use of foreign material (especially Nylon) at operation. On the other hand, of 1,139 cases of pure mitral stenosis, only 4 are known to have become infected after surgery. There is an intermediate incidence of the disease in the other 2 large groups that came to surgery, 3 in 400 cases of combined aortic and mitral stenosis or varying combinations of such stenosis with minor regurgitation, and 2 in 142 cases of major mitral regurgitation.

It cannot be stated that these results represent a higher incidence of this disease than could be expected if surgery had not been performed. One would have to know what the expected occurrence of the disease would be in a 3-month or 6-month period of the life of a group of similar patients (similar in age, sex, location and degree of valvular disease) not undergoing cardiac surgery, or even a group of similar patients undergoing some other form of surgery. Such data are not at hand. The only information now available is from autopsy material that tells us the rate of occurrence in the lifetime of a group of patients, now dead, not in an isolated 3 to 6-month segment of that lifetime, as we learn from our surgical material.

It should be remarked that the incidence of bacterial endocarditis is relatively low in cases of atrial septal defect, and yet we have seen 1 case in 82 operated patients.

Perhaps the influence of calcific masses in the valvular structure is similar in some ways to the influence of foreign material on the course of the disease. Such areas of calcification are largely avascular, and at times the calcium erodes through the endocardial surface of the valve, producing a rough surface on which it is possible for a fibrin nidi to form and for bacteria to grow. This development may be a factor in the greater incidence of the disease at the aortic valve, where calcification is greater in frequency and amount than at the mitral valve.

The onset of the infection occurred fairly quickly in one third of the cases, moderately quickly in one-third, and slowly in another one-third. Yet, the possible causal relationship to surgery cannot be denied even in the last group, except perhaps in 2 cases that appeared 7½ months and 10 months after surgery. There can be no arbitrary time limit beyond which the disease can be said to be unrelated to surgery. Rather, one can say only that the longer the interval between surgery and clinical evidence of infection, the less likely is there a causal relationship. The insidious onset of the disease makes any other conclusion subject to considerable error.

The etiologic agent was not the usual streptococcus viridans, but rather the staphylococcus, most frequently coagulase-negative. Certainly the clinical picture was not characteristic of bacterial endocarditis of a subacute nature, since petechiae were never seen, nor was clubbing of the fingers, Osler's nodes, or a café-au-lait color. A palpable spleen was most uncommon. All in all, one is forced to conclude that these infections differ from the commonly accepted pattern of subacute bacterial endocarditis in nonsurgical cases. Embolism was seen in only 1 case.

In 1 case with a staphylococcus cultured at autopsy, tubercle bacilli were observed on the strip of pericardium used to obstruct the mitral valve orifice. In addition, there were healed vegetations on this same tissue, presumably due to another organism. Finally, there was generalized miliary tuberculosis of the lung, pericardium, liver, spleen, kidney, and peri-aortic nodes, suggesting the possibility of tuberculous endocarditis.

In accordance with recent experience, we have observed penicillin resistance in some staphylococci. Eight of 12 organisms were distinctly resistant to this antibiotic, and also to streptomycin and oxytetracycline (Terramycin). In a general way these organisms were found somewhat sensitive to Chloromycetin and erythromycin. Sensitivity studies on streptococci are too meager to permit any conclusions. Most patients received large doses of antibiotics prior to and in the 10-day period after surgery. This antibiotic program
always included penicillin, so that one could expect to grow in mouth, bowel, bladder, and bloodstream only those organisms that were able to survive such therapy and become established. If this theory is correct, the need for a "light hand" in the handling of antibiotics is emphasized for this group of patients. Consistent with this thought is the observation of a number of patients with pseudomonas bactere mia after surgery. This organism may be one of a group that would be released for growth when its potential antagonists had been abolished by vigorous antibiotic therapy.

If care not to use too much is important in prophylaxis against the development of resistance, care to use enough or more than enough, is important in the management of the case with a positive blood culture. Initially we thought it wiser to begin therapy with the first report of a positive culture, rather than to wait for sensitivity tests. For this reason, with the staphylococci we used very large doses of penicillin intravenously (5,000,000 to 30,000,000 units daily) and streptomycin intramuscularly. When the sensitivity tests became available, other antibiotics were added if there had been no clinical response.

At present only 8 of the 20 patients are alive. One of those dead recovered from his infection, but died of congestive heart failure 18 months later (case 7). One apparently recovered, but died of a relapse 6 months later, during surgery for a mycotic femoral aneurysm, the aneurysm probably resulting from an infected embolus (case 3). Another died of undetermined cause 6 months after completing therapy of her infection (case 19). In 1 case the patient had been afebrile for 48 hours, and died of heart failure, with evidence over some days of an increasingly dynamic aortic regurgitation (case 5). Finally, 2 cases can hardly be said to have been treated, since the diagnosis was not made until the last few days of life (cases 12 and 18), 1 case having been mistaken for rheumatic fever (case 18). Six cases remain in which presumably adequate therapy was truly a failure, 8 in which it was successful.

**Summary**

Twenty cases of bacterial endocarditis occurring after cardiac surgery, in a total of 2,263 cases operated between 1950 and 1955, are reviewed. There was 1 instance among 374 cases of congenital heart disease, 19 among 1,889 cases with acquired valvular heart disease. Aortic valvular disease predominated among the cases of rheumatic heart disease (10 out of 19); mitral disease was the background in 6 cases; combined aortic and mitral disease in 3. Among the 19, fairly heavy valvular calcification was present in 11.

Aortic commissurotomy was the most frequently performed operation. In 4 cases a nylon foreign-body was introduced above the aortic valve to try to correct aortic insufficiency. Eight cases had a mitral commissurotomy, but 3 of these were in association with aortic surgery.

About one third of the cases occurred early (within 10 days after surgery), one-third late (more than 3 months after surgery), and the remaining one-third were in the intermediate group. The most common infecting organism (14 cases) was the staphylococcus, the coagulase-negative variety far more frequently than any other. These organisms were generally quite resistant to penicillin, both in the bacteriologic laboratory and clinically. With a variety of therapy, which was generally fairly massive, 8 patients are living and well. Of the 12 dead, 6 must be considered true failures of treatment of the infection.

Clinically and bacteriologically, bacterial endocarditis occurring after cardiac surgery seems to be a more malignant lesion than the common subacute variety seen in nonsurgical cases. This increased severity is probably a reflection of the trauma to the valve, the nature of the organism causing the infection, and the severe stress to which the patient has been subjected.

**Sumario in Interlingua**

Es presentate un revista del 20 casos de endocarditis bacterial occurrente postchirurgicamente in un total de 2,263 operationes cardiac execute inter 1950 e 1955. Un del 20 casos representava un gruppo de 374 operationes in casos de congenite morbo cardiac; le alte 19 representava le 1,889 casos de acquirite morbo de valvula cardiac. Morbo del valvula aortic predominava inter le casos
de rheumatic morbo cardiac (10 ex 19). Morbo mitral eseva al fundo de 6 casos. Le combinación de morbo mitral e aortic caracterisava 3 casos. Inter le 19 casos de rheumatic morbo cardiac, 11 revelava le presentia de satis sever grados de calcification valvular.

Commissurotomia aortic eseva le operation executate le plus frequemente. In 4 casos un corpore alien de nylon eseva introducute supra le valvula aortic in le effortio de corrigre le insufficientia aortic. Commissurotomia mitral eseva le operation in 8 casos, sed in 3 de illos le commissurotomia mitral eseva associate con chirurgia aortic.

Circa un tertio del casos occurreva promptemente (intra 10 dies post le operation); un tertio tardivamente (plus que 3 menses post le operation); e le ultime tertio constitueva un grupo intermediari. Le plus commun or- ganismo infective eseva le staphylococco (14 casos). Le varietate coagulase-negative eseva plus frequente que omne le altere varietates. Iste organismos se mostrava generalmente satis resistente a penicillina, tanto in le laboratorio bacteriologic como etiam clinicamente. Un varietate de terapias de character generalmente massive eseva usate. Octo del patientes vive e se trova ben. Ex le total de 12 mortes, 6 debe esser considerate como claramente causate per non-successo del trattamento del infection.

Ab le punctos de vista clinico e bacteriologic, endocarditis bacterial post chirurgia cardiac es apparentemente un lesion plus maligne que le subacute varietate common que es incontrate in casos nonchirurgic. Iste plus alte grado de severitate es probablemente un reflexion del trauma valvular, del natura del organism que causa le infection, e del severitate del stress que le paciente ha absorbite.

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Bacterial Endocarditis Following Cardiac Surgery
CLARENCE DENTON, ELIAS G. PAPPAS, JOSEPH F. URICCHIO, HARRY GOLDBERG and WILLIAM LIKOFF

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