Left Atrial Calcification

Review of Literature and Proposed Management

By J. Warren Harthorne, M.D., Ronald A. Seltzer, M.D., and W. Gerald Austen, M.D.

Increasing utilization of cardiac surgical techniques in the management of chronic rheumatic heart diseases has focused attention on calcification of the left atrial wall. Recent experience suggests that this is no longer the medical curiosity it was once considered. The present article reviews the available literature and presents data on 15 additional patients observed at the Massachusetts General Hospital during the past 6 years.

History and Review of Literature

In 1912, Oppenheimer1 presented the post-mortem findings on a 44-year-old male who died of congestive heart failure due to mitral stenosis. The entire left atrial wall was extensively calcified; only the atrial appendage and septum were spared. In 1924, MacCallum2 first described rheumatic endocarditis affecting the posterior left atrial wall. Stewart and Branch,3 in the same year, reported the post-mortem findings on a 16-year-old girl with chronic rheumatic pannicarditis and patchy ulceration and focal calcification of the left atrial endocardium. A similar case had been reported previously in the French literature4 although the relation to rheumatic fever had not been noted. The first x-ray of left atrial calcification, observed antemortem by Bedford, was published by Shanks and associates in 1938.5 Early clinical reports were those of Begg,6 Epstein,7 Young and Schwedel,8 Miller and associates,9 and Fainsinger10 who supported the concept of calcification due to underlying rheumatic endocarditis. Two cases were reported by Ruskin and Samuel11 in 1952 who observed that these patients seem to live unusually long despite severe valvular damage and prolonged chronic heart failure. Eight cases verified at surgery were reviewed by Curry and associates12 in 1953 out of 300 patients screened for mitral valvulotomy, but little clinical history was given. Hemley and co-workers,13 in 1953, cited two cases and stressed that most such patients have long-standing rheumatic heart disease and are usually more than age 40. Further cases were presented in 1954 by Hutton14 who stressed the female preponderance, and by Soloff and associates.15 Leonard and co-workers16 reviewed 38 previous cases and added one of their own. They stressed the frequency of multivalvular lesions and suggested this as a relative contraindication to surgery. Vickers and associates17 in 1959, added five cases. While most such patients had significant mitral regurgitation, four of their patients had predominant mitral stenosis leading to operation. They reviewed the surgical approaches available. Mahoney and O'Laughlin18 also in 1959, reviewed the significance of left atrial calcification and observed an average duration of disease of 29 years prior to the x-ray appearance of calcification with an average duration of symptoms of 17 years. Baeyens and associates,19 in 1960, estimated the frequency of such calcification at 1 to 2% of the rheumatic population. O'Farrell20 and LeDamany and associates21 reported attempted mitral valvulotomies which could not be completed because of heavy left atrial calcification. The largest series reported to date is that of Andersch and co-workers22 who, in 1961, observed 11 cases of calcified left atrium
recognized by x-ray. Shapiro and his colleagues in 1963 reviewed the roentgenographic findings in 12 cases, a number of which had been reported previously by other authors. Scattered case reports have appeared in recent years bringing the total number reported to date to 71 cases.

**Group Studied**

During the past 6-year period (1959 to 1965), 15 cases of left atrial calcification have been observed at the Massachusetts General Hospital. The clinical features of this group of patients are summarized in table 1. Selected cases are reviewed below in more detail. Thirteen of these were recognized roentgenographically. Two were seen retrospectively on preoperative films after surgery confirmed the presence of calcification. Of the 15 cases, 11 have been confirmed by surgery or postmortem examination or both while four were roentgenographic diagnoses alone.

**Report of Cases**

**Case 1, E.H. (MGH 1010468)**

This 52-year-old mother of three had suffered rheumatic fever at age 7 with several recurrences. Dyspnea on exertion appeared at age 38, and at age 43, she suffered left hemiplegia consequent to a cerebral embolus at which time atrial fibrillation was first noted. Increasing exercise intolerance led to a closed mitral valvulotomy at age 52 at which time murmurs of pure mitral stenosis and mild aortic regurgitation were heard. Preoperative chest x-rays revealed heavy calcification of the left atrial wall (fig. 1 left). No note of calcification of the wall was made in the surgical report but a large soft thrombus was encountered. The valve was fibrous and split well, but the patient suffered a saddle embolus to the aortic bifurcation which was removed at the same time. She died 48 hours later of uncontrolled retroperitoneal hemorrhage. Postmortem examination revealed a large, soft mural thrombus in the left atrium with underlying fibrosis of the wall and whitish plaque-like calcification covering the entire posterior atrial wall. A postmortem photograph (fig. 2) reveals that the atrial septum was spared from the calcification. There were multiple recent and old renal and cerebral infarcts.

**Case 4, E.S. (MGH 1325667)**

This 44-year-old physician's wife had developed a heart murmur at age 8 years following scarlet fever. Mild congestive heart failure complicated her third pregnancy at age 37, and atrial fibrillation developed at age 38. At age 40 a saddle embolus to the aortic bifurcation was treated conservatively. At age 44 right and left heart catheterization with left ventricular angiogram demonstrated severe mitral stenosis, slight mitral regurgitation, severe pulmonary hypertension (P.A. 100/40 mm Hg), and

![Figure 1](image)

*In left panel, Case 1. Calcification outlines the atrial wall with an opening anteriorly in the region of the mitral annulus. In right panel, case 5, note extension of calcification into a pulmonary vein.*

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## Clinical Features of Fifteen Cases of Left Atrial Calcification

<table>
<thead>
<tr>
<th>Case</th>
<th>Age &amp; sex</th>
<th>Diagnosis</th>
<th>Valve calcium</th>
<th>Rhythm</th>
<th>Surgical (Op.) or postmortem (P.M.) findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. (1010468)</td>
<td>52 F</td>
<td>MS</td>
<td>No</td>
<td>AF, 16 yr</td>
<td>Op 1 and PM: Large friable LA clot, diffuse calcification of wall, septum spared, valve fibrous</td>
</tr>
<tr>
<td>2. (863838)</td>
<td>48 F</td>
<td>MS, AR</td>
<td>No</td>
<td>Parox. AF</td>
<td>Op. 1: Appendage thrombosed, plaques of calcium in wall, large clot, tight fibrous valve</td>
</tr>
<tr>
<td>3. (187988)</td>
<td>56 F</td>
<td>MS, AR</td>
<td>No</td>
<td>AF, 13 yr</td>
<td>Op. 1: Routine closed valvulotomy, no calcium or clot noted (1951)</td>
</tr>
<tr>
<td>4. (1325667)</td>
<td>44 F</td>
<td>MS, TR</td>
<td>No</td>
<td>AF, 6 yr</td>
<td>Op. 1: Atrial wall diffusely calcified in plaques, septum spared, no clot felt, much difficulty entering atrium, valve split “closed” with production of moderate regurgitation</td>
</tr>
<tr>
<td>5. (0410305)</td>
<td>54 F</td>
<td>MS, AR</td>
<td>Yes</td>
<td>AF, 20 yr</td>
<td>Op. 1: Thrombosed appendage, calcified wall, organized LA clot, fibrous valve.</td>
</tr>
<tr>
<td>6. (1260455)</td>
<td>50 M</td>
<td>MS, TR</td>
<td>No</td>
<td>AF, 11 yr</td>
<td>Op. 2: Diffusely calcified wall</td>
</tr>
<tr>
<td>7. (158010)</td>
<td>56 F</td>
<td>MB, MS, AR, AS TR</td>
<td>No</td>
<td>AF, 20 yr</td>
<td>Op. and PM: Calcification of wall with LA clot</td>
</tr>
<tr>
<td>8. (260943)</td>
<td>50 F</td>
<td>MS, AS, TR, PS</td>
<td>?</td>
<td>AF, 12 yr</td>
<td>P.M.: Calcification of wall, extent not specified with emboli of spleen and kidneys</td>
</tr>
<tr>
<td>9. (331479)</td>
<td>56 F</td>
<td>MS</td>
<td>No</td>
<td>AF, 20 yr</td>
<td>Op. 1: “Great deal” of adherent clot, wall calcified but not appendage, valve fibrous</td>
</tr>
<tr>
<td>10. (756047)</td>
<td>62 F</td>
<td>MS, TR</td>
<td>No</td>
<td>AF, 14 yr</td>
<td>None</td>
</tr>
<tr>
<td>11. (1308591)</td>
<td>49 F</td>
<td>MS</td>
<td>No</td>
<td>AF, 30 yr</td>
<td>None</td>
</tr>
<tr>
<td>12. (0814298)</td>
<td>57 F</td>
<td>MB, MS TR</td>
<td>No</td>
<td>AF, 27 yr</td>
<td>PM: Giant left atrium; calcified appendage and posterior wall</td>
</tr>
<tr>
<td>13. (570819)</td>
<td>56 F</td>
<td>MS, AS</td>
<td>Yes</td>
<td>NSR</td>
<td>Op. 1: Age 49, closed mitral, open aortic valves, no calcium</td>
</tr>
<tr>
<td>14. (287518)</td>
<td>50 M</td>
<td>MS, AR</td>
<td>No</td>
<td>AF, 19 yr</td>
<td>Op. 2: Age 51, open mitral and aortic valves, no calcium</td>
</tr>
<tr>
<td>15. (321917)</td>
<td>61 F</td>
<td>MS, TR</td>
<td>Yes</td>
<td>AF, 17 yr</td>
<td>Op. 3: Age 56, aortic and mitral prostheses; appendage and wall calcified; no clot; patient died on table</td>
</tr>
</tbody>
</table>

*Figures in parentheses indicate the number of years between the original episode of rheumatic fever and the first recognition of atrial calcification.*
<table>
<thead>
<tr>
<th>Embolic phenomena</th>
<th>History of rheumatic fever*</th>
<th>Present status</th>
<th>Duration of symptoms prior to recognition of calcium</th>
<th>Means by which calcification was diagnosed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral at age 47; saddle embolus during operation</td>
<td>Yes (52)</td>
<td>Dead P.O.</td>
<td>21 yr</td>
<td>X-ray and postmortem</td>
</tr>
<tr>
<td>Pulmonary emboli</td>
<td>No</td>
<td>Living, improved following surgery</td>
<td>5 yr</td>
<td>X-ray and surgery</td>
</tr>
<tr>
<td>Cerebral embolus at age 43, renal ?; B.P. 200/104</td>
<td>Yes (41)</td>
<td>Living, CHF</td>
<td>20 yr</td>
<td>X-ray only; not felt at operation</td>
</tr>
<tr>
<td>Saddle embolus at age 40</td>
<td>Yes (38)</td>
<td>Living, improved P.O.</td>
<td>7 yr</td>
<td>X-ray and surgery</td>
</tr>
<tr>
<td>Cerebral, splenic, and pulmonary emboli at age 27</td>
<td>Yes (24)</td>
<td>Dead, P.O.</td>
<td>22 yr</td>
<td>X-ray, surgery, and postmortem</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>No</td>
<td>Living, improved P.O.</td>
<td>9 yr</td>
<td>Surgery and X-ray in retrospect</td>
</tr>
<tr>
<td>Pulmonary and both femorals</td>
<td>Yes (50)</td>
<td>Dead, P.O.</td>
<td>20 yr</td>
<td>X-ray, surgery, and postmortem</td>
</tr>
<tr>
<td>Splenic and renal</td>
<td>Yes (39)</td>
<td>Dead, CHF and pulmonary emboli</td>
<td>12 yr</td>
<td>X-ray and postmortem</td>
</tr>
<tr>
<td>Cerebral and renal ?; B.P. 170/100</td>
<td>Yes (41)</td>
<td>Living, improved P.O.</td>
<td>25 yr</td>
<td>X-ray and surgery</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Yes (27)</td>
<td>Living, chronic CHF</td>
<td>14 yr</td>
<td>X-ray only</td>
</tr>
<tr>
<td>Total occlusion of abdominal aorta</td>
<td>Yes (44)</td>
<td>Living, refuses operation</td>
<td>20 yr</td>
<td>X-ray only</td>
</tr>
<tr>
<td>Cerebral at age 48; several suspected pulmonary</td>
<td>Yes (43)</td>
<td>Dead, chronic CHF</td>
<td>27 yr</td>
<td>X-ray and postmortem</td>
</tr>
<tr>
<td>None</td>
<td>Yes (47)</td>
<td>Died during op. 3</td>
<td>11 yr</td>
<td>X-ray, surgery, and postmortem</td>
</tr>
<tr>
<td>Pulmonary emboli at ages 33 and 34, left femoral embolus at age 45, right femoral embolus at age 50</td>
<td>Yes (40)</td>
<td>Living, improved after op. 2</td>
<td>19 yr</td>
<td>X-ray and surgery</td>
</tr>
<tr>
<td>Left femoral embolus at age 49</td>
<td>Yes (46)</td>
<td>Chronic CHF</td>
<td>27 yr</td>
<td>X-ray only</td>
</tr>
</tbody>
</table>
tricuspid regurgitation. Because of the severe pulmonary hypertension, a “closed” mitral valvulotomy through the left chest using the transventricular dilator was performed. The left atrial wall was diffusely calcified as was the atrial appendage. Great difficulty was encountered in dissecting the left atrial wall and in achieving hemostasis. An adequate “split” was obtained with the production of moderate regurgitation. No left atrial clot could be felt. The atrial septum was not involved by the calcification. The postoperative course was uneventful with the exception of a transient postpericardiotomy syndrome.

Case 12, F.H. (MGH 0814298)

This 57-year-old woman had suffered rheumatic fever at age 14 and had developed mild congestive failure during her only pregnancy at age 18. Atrial fibrillation and dyspnea on exertion appeared at age 30 and became progressively more severe thereafter culminating in hospitalization in acute pulmonary edema at age 43. A left cerebral embolus occurred at age 48 and several suspected pulmonary emboli resulted in repeated admissions. Her terminal years were spent in chronic congestive heart failure despite a “tight” medical program. She persistently refused surgery.

Physical examination revealed evidence of pre-dominant mitral stenosis with mild mitral regurgitation and mild aortic regurgitation. X-rays showed a giant left atrium filling most of the thorax and a rim of calcium in the region of the atrial appendage. The electrocardiogram showed atrial fibrillation. The final admission was precipitated by an epileptic seizure. Death in congestive failure occurred several days later. Postmortem examination revealed predominant mitral stenosis, evidence of mild mitral regurgitation, a huge left atrium, and calcification of the atrial appendage and posterior left atrial wall. The atrial septum was spared by the calcification. There was no clot found in the atrium, but the heart had been perfused prior to opening.

Case 14, W.E. (MGH 287518)

This 49-year-old unemployed male had a history of “growing pains” as a child without definite rheumatic fever. At age 26, a murmur of mitral stenosis was heard with hemoptysis and exertional dyspnea appearing at age 29. Several episodes of pulmonary embolism occurred at ages 33 and 34 and led to ligation of the femoral veins and later of the inferior vena cava with brief treatment with anticoagulants. Increasing symptoms led to closed mitral valvulotomy at age 39. No clots or calcium were noted but the appendage was very small. The postoperative result was excellent. At age 45, he suffered a femoral embolus which was removed by embolotomy, but he was otherwise well until age 48 when increasing dyspnea and fatigue led to right heart catheterization which confirmed mitral restenosis. X-rays showed a ring-like calcification of the entire left atrial wall (fig. 3). Physical findings were those of mitral stenosis and mild aortic regurgitation. While awaiting reoperation, he suffered a right femoral embolus which was treated conservatively.

He subsequently underwent open mitral valvulotomy which was purposely carried out through the right side of the chest anticipating approach to the left atrium through the atrial septum or posterior interatrial groove. A narrow strip of the posterior left atrial wall adjacent to the septum was free of calcium and allowed easy access to the mitral valve. A large amount of endocardial calcium and adherent thrombus was excised, and the valve was opened under direct vision. The atrial septum was entirely free of calcium. The postoperative result has been excellent.

Discussion

Conclusions are difficult to draw from much of the published literature on calcification of the left atrium because of the sparsity of clinical details given. However, certain character-
istic features are apparent (table 2). Atrial fibrillation of many years duration is almost universal; it averaged 10 years in other series. Mitral valve involvement, usually of a severe degree, is present in all patients although the incidence of valvular calcification seems unrelated. Combined or multivalvular lesions are common. The predominance of women is striking (74%) and follows the higher female incidence of rheumatic involvement of the mitral valve. Symptoms referable to the cardiovascular system had been present for an average of 19.7 years prior to recognition of left atrial calcification. The diagnosis was confirmed by postmortem examination or surgical intervention in 31 of the 71 cases reported in the world literature. The incidence

![Figure 3](image_url)

*Case 14. Right anterior oblique and lateral x-rays of chest. The left atrial wall was diffusely calcified at the time of surgery. The atrial septum was uninvolved.*

<table>
<thead>
<tr>
<th>Source</th>
<th>Total patients &amp; sex</th>
<th>Average age, yr</th>
<th>Rhythm</th>
<th>Emboli</th>
<th>Duration of symptoms</th>
<th>Diagnosis confirmed</th>
</tr>
</thead>
<tbody>
<tr>
<td>World literature</td>
<td>53 Females 18 Males 71 Total</td>
<td>52 (53)*</td>
<td>52 AF 1 NSR</td>
<td>9 Systemic or pulmonary 2 (?)Renal 11 No emboli</td>
<td>(22)* Av 19.7 yr</td>
<td>31 of 71</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M.G.H. series</td>
<td>13 F 2 M 15 Total</td>
<td>54</td>
<td>13 AF 1 NSR 1 Paroxysmal AF</td>
<td>11 Systemic 8 Pulmonary 1 No emboli</td>
<td>Av 17.2 yr 11 of 15</td>
<td></td>
</tr>
</tbody>
</table>

*Figures in parentheses indicate the number of cases in which information was available.

**Table 2**

*Characteristic Features of Calification of Atrium in World Literature and Present Series*

*Abbreviations: AF = atrial fibrillation; NSR = normal sinus rhythm.*
of systemic or pulmonary embolism is difficult to estimate but they were present in 11 of the 22 cases in which a specific note was made.

Of the present series of 15 cases, the diagnosis in four remains unconfirmed by surgery or postmortem examination or both. Thirteen (87%) had a history of preceding rheumatic fever; the average duration from rheumatic fever to recognition of atrial calcification was 41 years; the average age at the time of recognition of calcium was 54 years; and the average duration of symptoms was 17.3 years. Atrial fibrillation was present in 13 patients; one patient had paroxysmal atrial fibrillation and one was in normal sinus rhythm. The average duration of atrial fibrillation was 17 years. Electrocardiograms were nonspecific except for atrial fibrillation and varied according to the underlying valve lesion. Three patients had relatively pure mitral stenosis while the remaining 12 had combined lesions present. A history of previous embolization, systemic (11 of 15 cases) or pulmonary (eight of 15 cases), was found in 93% of the present series. This observation places such cases in a high risk category both for medical as well as surgical treatment. Only one patient was free of a history of previous embolism.

**Diagnosis**

Roentgenographic recognition of this entity has received much comment in radiological literature. Mural calcification appears as a thin, curvilinear density tracing the outline of the left atrium in part or completely. In the frontal projection, a round or oval shell of calcium 8 to 10 cm in diameter is usually seen lying in the center of the cardiac silhouette beneath the carina and the main stem bronchi (fig. 4). In the lateral or oblique projections, a completely calcified wall appears as a C-shaped curvilinear density with the opening of the C lying anteriorly in the region of the mitral annulus (fig. 1). Lesser degrees of calcification may be confined to streaks along one or more margins or may outline the entire posterior wall (fig. 5). Occasionally the calcification extends into the pulmonary veins (fig. 1 right). If the calcification closely approaches or forms the left heart border in the frontal projection (fig. 6), the atrial appendage is probably calcified, and this may occur as an isolated finding. It must be remembered, how-

![Figure 4](http://circ.ahajournals.org/content/XXXIV/3/204/F2.large.jpg)

**Figure 4**

Case 2. Posteroanterior and lateral views of chest. The left atrium is outlined by calcium in both projections.
ever, that the calcification is frequently more extensive than revealed by x-ray.

Careful roentgenographic techniques are necessary to identify left atrial calcification. High kilovoltage and well-penetrated films are essential. The lateral or oblique projections are most useful. Laminography may be helpful for better definition of the extent of the calcification (fig. 7). Fluoroscopy, because of movement of the deposits, often reveals calcification not apparent on routine films. Thus, the bright image produced by image intensifiers and cineradiography now offers perhaps the best means for the location and identification of intracardiac calcium.

Left atrial calcification must be differentiated from calcification occurring in the cardiac valves, pericardium, coronary arteries, ventricular aneurysms or infarctions, intracardiac tumors, mediastinal cysts, hilar nodes, and costal cartilages. All of these have characteristic locations and motion, and present little difficulty to the experienced observer.

Figure 5

Case 3. Chest fluoroscopy, posteroanterior, lateral, and both oblique views. The barium-filled esophagus is deviated posteriorly by the enlarged left atrium. Calcification incompletely traces the outline of the left atrial wall.
Figure 6

Case 7. Posteroanterior and lateral views of chest. Calcification appears in the area of the atrial appendage. However, at surgery, calcification involved the posterior, superior, and lateral atrial walls.

Figure 7

Case 15. Posteroanterior x-ray of chest and anteroposterior laminogram. Superior calcification lies in the left atrial wall. Inferior deposits lie in the mitral annulus.
CARDIAC PATHOLOGY

Cardiac catheterization was performed 9 times in seven patients. The data are summarized in table 3. No characteristic findings were present other than those associated with the primary valve lesion. Of six patients in whom adequate information was available, four showed evidence of pulmonary vascular disease as manifested by an elevation of the pulmonary arteriolar or pulmonary vascular resistances. Three of these had had documented pulmonary emboli. The cardiac index was reduced in all six patients in whom it was determined and averaged 2.3 L/min/m² of body surface area. Phasic pulmonary capillary pressure tracings did not reveal any characteristic changes in the wave forms.

Pathology

The consensus of most authors is that left atrial calcification is the end-result of repeated and extensive rheumatic auriculitis beginning with focal patches of calcification in areas of rheumatic endocardial ulceration and progressing to chronic fibrotic changes with plaques of subendocardial calcium of varying extent. The incidence of atrial calcification appears to be related to the severity of the original rheumatic attack and the associated valvular damage. As noted in the present series, adherent mural thrombi are a common finding. Accurate description of the atrial calcification is missing in most published reports. In two cases in the literature and five cases in the present series the region of the atrial septum was specifically noted as free of calcium (fig. 2). Associated valvular calcification bears no predictable relation to atrial calcification.

Surgical Approach

Eleven patients of the present series underwent a total of 16 operations with four operative deaths (25%). Of these, 12 were closed valvulotomies carried out via a standard left thoracotomy or median sternotomy. One of the patients undergoing closed valvulotomy died of massive retroperitoneal hemorrhage consequent to a saddle embolus incurred at the time of surgery. Another died
of electrolyte imbalance and congestive failure 3 weeks postoperatively. Good results followed the remaining 10 closed valvulotomies with no immediate embolic consequences. Considerable difficulty was encountered in most of these cases in entering the left atrium because of marked thickening and calcification of the left atrial wall. In general, entry into the atrial cavity was accomplished by blunt dissection in the region of the atrial appendage through which the index finger was then forced. In five of the 12 cases, an extensive mural thrombus was encountered at the time of surgery. Hemorrhage at the site of the atriotomy was frequent, and in one patient, the calcified atrial wall fractured outside of the atrial purse string requiring patching with a piece of pericardium.

Four open mitral valvuloplasties were done. Two of these were done by way of the left chest in the same patient at the time of open aortic valvuloplasty through a median sternotomy incision. At the second operation, the extensively calcified atrium and mitral annulus became torn during the removal of the diseased mitral valve and prevented adequate insertion of the prosthesis; the patient died on the operating table. The other two open valvuloplasties were performed through the right chest. In one of these cases (case 5), this route was used to visualize the tricuspid valve. The left atrium was entered through the posterior interatrial groove. The mitral valve was excised and replaced by a Starr-Edwards prosthesis. The entire left atrial endocardium was lined with loose calcium plaques although involvement of the septum was not commented on in the operative summary. The patient never regained consciousness and died on the twelfth postoperative day.

Of particular interest is case 14 in whom atrial calcification had been recognized well in advance of mitral valve surgery utilizing the techniques mentioned earlier (fig. 3). On the basis of the previous pathology reports, it was anticipated that the atrial septum would be spared from the process of calcification. This patient's operation was done as an open procedure and the approach was through a right thoracotomy and the posterior interatrial groove. The mitral valve was easily approached through a noncalcified portion of the wall adjacent to the septum which was, itself, free of calcification despite extensive involvement of the remainder of the chamber. Loose plaques of calcium and adherent thrombus were debrided and the mitral valve repaired under direct vision. Repair of the atriotomy was uneventful and the postoperative result has been excellent.

The surgical approach to the patient with calcification of the left atrium must be individualized according to the overall clinical, hemodynamic, and radiological situation. When unrecognized prior to surgery, atriotomy must be accomplished by whatever route is most readily available; for example, the atrial appendage if it is not involved in the calcification, a pulmonary vein, or the free left atrial wall. When recognized preoperatively, several choices are available. For the relatively good-risk patient without complicating pulmonary hypertension, an open approach by way of the posterior interatrial groove or atrial septum through a median sternotomy or right thoracotomy incision seems preferable not only from the standpoint of providing readier access to the mitral valve but also for the removal of associated thrombus or loose calcium plaques. The presence of associated valvular lesions will dictate which incision is preferred. The risk of cardiopulmonary bypass is considerably higher in patients with pulmonary hypertension. Consequently, those patients with an noncalcified, purely stenotic mitral valve with significant elevation of the pulmonary arteriolar resistance may best be handled by a "closed" procedure through the left chest recognizing the risk of associated embolism and hemorrhage.

**Summary**

Calcification of the left atrial wall or appendage or both constitutes a major complication and risk to mitral valve surgery due to difficulty in entering the left atrium, potential embolization, and impaired hemostasis. This condition can be diagnosed preoperatively by
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a variety of radiological techniques. Surgery must be tailored to the individual patient and in the patient with isolated, noncalcific mitral stenosis with complicating pulmonary hypertension is probably best carried out as a closed procedure recognizing the attendant hazards. For the patient without pulmonary hypertension or with mixed or combined lesions, an approach through a right thoracotomy or median sternotomy and open valvuloplasty through the posterior interatrial groove or atrial septum seems preferable. The high incidence of embolism, systemic or pulmonary, renders such patients suitable candidates for long term anticoagulation treatment.

Addendum

Since completion of the above manuscript, a sixteen patient with calcification of the left atrium has been observed. This was a 64-year-old female with a 22-year history of congestive failure and previous renal, splenic, and cerebral emboli who died of complications following a thoracentesis. At postmortem, the entire left atrium was heavily calcified with the exception of the atrial septum which was soft and pliable. A large organized thrombus occupied much of the chamber. There was severe calcific mitral stenosis without other valvular lesions.

References

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J. WARREN HARTHORNE, RONALD A. SELTZER and W. GERALD AUSTEN

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