ence of complement or toxic complexes and only a few membranes contained fibrin. Some of the membranes contained a few granulocytes, as well as granular pneumocytes and lymphocytes. Chronic interstitial pneumonitis in many areas had progressed to fibrosis (Fig 4). Postmortem cultures grew no pathogens.

**DISCUSSION**

Pulmonary injury due to allergy (hypersensitivity) or direct drug toxicity has been described in association with a number of drugs. Nitrofurantoin¹ and perhaps methotrexate¹ may cause a true hypersensitivity type of lung injury. Eosinophilia is often present and it has been postulated that antigen-antibody complexes are involved in triggering cellular damage. In contrast, interstitial pneumonitis and fibrosis has been described in association with the use of hexamethonium⁵ and busulfan.⁶ These drug effects are most likely due to direct pulmonary toxicity. Although sometimes characterized as "hypersensitivity" reactions, there is no direct evidence that immunologic mechanisms are involved.

The clinical and histologic findings in our patient lack the hallmarks usually associated with allergic pulmonary reactions and more closely resemble pulmonary toxicity of the direct type ascribed to hexamethonium and busulfan. Shimosato et al,⁷ in the Japanese literature have postulated that bleomycin acts directly upon pulmonary vascular endothelium, alveolar epithelium and ground substance causing increased vascular permeability which leads to intra-alveolar and interstitial edema as an intermediate step in pulmonary fibrosis. Our immunofluorescence observations tend to support this hypothesis since they can be interpreted as showing transudation and/or exudation of plasma proteins. They do not support the idea that this was a true "hypersensitivity" reaction which might be expected to show the presence of complement.

ACKNOWLEDGMENT: We would like to acknowledge the assistance of Dr. Teruo Masukawa for his translations from the original Japanese.

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**Ventricular Aneurysms and Blunt Chest Trauma**

Gary M. Silver, M.D.; Nicola Spampinato, M.D.; Rene G. Favaloro, M.D.; and Laurence K. Groves, M.D.

A patient with blunt chest trauma leading to a left ventricular aneurysm (LVA) is presented and the literature is reviewed. The common pathophyslogic factor in the formation of these aneurysms appears to be obstruction of the left anterior descending coronary artery (LAD). Awareness of this might, under ideal circumstances, lead to emergency coronary catheterization and saphenous vein grafting preventing the formation of a LVA in a relatively young person.

Blunt trauma to the chest can result in aortic insufficiency, transection of the thoracic aorta, rupture of ventricular and atrial walls, creation of a ventricular septal defect, and rupture of chordae tendineae and papillary muscles.¹ Another complication of blunt chest trauma is damage to the coronary arteries with resulting ventricular aneurysm. We have seen two such cases. Most recent of these patients (17 in the world literature) survived both transection of the aorta and formation of a left ventricular aneurysm (LVA). The other was previously reported.²

These cases correlated with our experience with LVA secondary to coronary artery disease (CAD) suggest a common etiology. Conceivably, emergency coronary angiography and coronary artery surgery under ideal circumstances could abort aneurysm formation.

**CASE REPORT**

A 33-year-old man was in an automobile accident on April 22, 1970. He was hospitalized at another hospital until May 7, 1970, with fractures of the right wrist, ankle, three left ribs, and probable hematoma of the left lung, the result of injury from the steering wheel. There was no history of heart disease. The patient returned to work on June 7, 1970. On July 22, 1970, he suddenly became short of breath, and had substernal pain and nausea. He was hospitalized again and treated for acute congestive heart failure; an ECG showed extensive old anterior wall damage. On August 3, 1970, cardiac catheterization revealed normal right and circumflex coronary arteries and total occlusion of the anterior descending coronary artery with a large akiotic aneurysmal area of the anterior wall of the left ventricle and a mural thrombus. He was transferred to the Cleveland Clinic Hospital on August 27, 1970, where the absence of femoral pulses was discovered and an aortogram revealed transection of the thoracic aorta just distal to the left subclavian artery. It was decided that the left ventricular aneurysm should be repaired at this time and, in 8 to 12 months, the patient would return for repair of the aortic transection. On September 1, 1970, he underwent median sternotomy for resection of the left ven-

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Ventricular aneurysms and removal of the mural thrombus. Recatheterization on February 12, 1971 (Fig 1A and B) showed poor left ventricular function with elevated end-diastolic pressure and mild mitral regurgitation. This was due to the original infarct which involved the septum and a portion of the papillary muscles. Presently, he is continuing medical treatment, and working at a desk job.

**DISCUSSION**

An analysis of reported cases, and experience with this

<table>
<thead>
<tr>
<th>Case</th>
<th>Author</th>
<th>Year</th>
<th>Age, sex</th>
<th>Type of trauma</th>
<th>Time until diagnosis</th>
<th>How diagnosed and findings</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Potain</td>
<td>1892</td>
<td>40 M</td>
<td>Hit by wagon shaft over left chest</td>
<td>13 months</td>
<td>Autopsy—apex aneurysm, no mention LAD</td>
<td>Dead—congestive heart failure</td>
</tr>
<tr>
<td>2</td>
<td>Groom</td>
<td>1897</td>
<td>16 M</td>
<td>Thrown from horse</td>
<td>1 month</td>
<td>Autopsy—LVA, no mention LAD</td>
<td>Dead—rupture</td>
</tr>
<tr>
<td>3</td>
<td>French</td>
<td>1912</td>
<td>3 F</td>
<td>Fell out of third floor window</td>
<td>20 days</td>
<td>Autopsy—anterior lateral LVA, no mention LAD</td>
<td>Dead—rupture</td>
</tr>
<tr>
<td>4</td>
<td>Fraschke</td>
<td>1917</td>
<td>20 M</td>
<td>Blast injury</td>
<td>6 months</td>
<td>Autopsy—anterior lateral LVA—thrombus occluding LAD</td>
<td>Dead—CHF</td>
</tr>
<tr>
<td>5</td>
<td>Schminke</td>
<td>1925</td>
<td>45 M</td>
<td>Struck by wagon</td>
<td>6 months</td>
<td>Autopsy—LVA—thrombus in LAD</td>
<td>Dead—arrhythmia</td>
</tr>
<tr>
<td>6</td>
<td>Joachim and Mays</td>
<td>1927</td>
<td>12 M</td>
<td>Run over by wagon</td>
<td>13 years</td>
<td>Autopsy—“LAD stopped abruptly at upper edge of aneurysm,” anterior LVA</td>
<td>Dead—arrhythmia</td>
</tr>
<tr>
<td>7</td>
<td>Hawkes</td>
<td>1935</td>
<td>6 M</td>
<td>Hit by truck</td>
<td>3 months</td>
<td>Autopsy—LVA, no mention of LAD</td>
<td>Dead—rupture</td>
</tr>
<tr>
<td>8</td>
<td>Hildebrandt</td>
<td>1938</td>
<td>27 M</td>
<td>“Blunt chest trauma”</td>
<td>18 years</td>
<td>Autopsy—old LVA with mural thrombus</td>
<td>Dead—embolus</td>
</tr>
<tr>
<td>9</td>
<td>Pitts and Purvis</td>
<td>1947</td>
<td>10 M</td>
<td>Hit by truck</td>
<td>2.5 mos.</td>
<td>Autopsy—anterior lateral LVA, no mention of LAD</td>
<td>Dead—rupture</td>
</tr>
<tr>
<td>10</td>
<td>Cavassuti and Forattini</td>
<td>1952</td>
<td>No information</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Green et al</td>
<td>1965</td>
<td>10 M</td>
<td>Hit by car</td>
<td>8 weeks</td>
<td>X-ray; surgery; then ciné; LVA—no mention of LAD</td>
<td>Alive and well after surgery</td>
</tr>
<tr>
<td>12</td>
<td>Waldhausen et al</td>
<td>1966</td>
<td>5 M</td>
<td>Kicked by horse</td>
<td>15 months</td>
<td>Murmur; left and right heart catheterization</td>
<td>Alive and well after surgery</td>
</tr>
<tr>
<td>13</td>
<td>Cuendet et al</td>
<td>1966</td>
<td>No information</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>Glancey et al</td>
<td>1967</td>
<td>20 M</td>
<td>Playing football</td>
<td>32 months</td>
<td>EKG and x-ray—ciné, large apical and anterior lateral LVA</td>
<td>Alive and well after operation, then died of embolus</td>
</tr>
<tr>
<td>15</td>
<td>Killen et al</td>
<td>1969</td>
<td>36 M</td>
<td>Auto accident</td>
<td>2 years</td>
<td>X-ray, explore mediastinum; then ciné, anterior lateral LVA, no mention LAD</td>
<td>Alive and well after operation</td>
</tr>
<tr>
<td>16</td>
<td>Pupello et al</td>
<td>1969</td>
<td>17 M</td>
<td>Auto accident</td>
<td>3 weeks</td>
<td>X-ray chest, angiogram; no mention LAD</td>
<td>Alive and well after operation</td>
</tr>
<tr>
<td>17</td>
<td>Present report</td>
<td>1973</td>
<td>33 M</td>
<td>Auto accident</td>
<td>3 months</td>
<td>Total obstruction LAD; RCA Cx good; clots LVA</td>
<td>Alive and well after operation</td>
</tr>
</tbody>
</table>

LAD: Left Anterior Descending  
LVA: Left Ventricular Aneurysm

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case allow for some interesting observations regarding the pathogenesis, diagnosis and treatment of ventricular aneurysms resulting from blunt chest trauma (Table 1).

Most cases involved relatively young persons in whom the lesions were caused by direct trauma on the anterior chest wall. Recent cases were related to car accidents, with sudden deceleration and impact of the chest against the steering wheel (Fig 2). Under these conditions direct trauma to the chest can cause rupture of the atrial or ventricular wall, from a sudden increase of intrachamber pressure, particularly in the late diastolic or early systolic phase. Rib fractures can cause perforation of the ventricle with consequent cardiac tamponade or even formation of a false aneurysm communicating with the ventricular cavity through a very narrow neck.

The formation of a true ventricular aneurysm is a relatively late manifestation of the trauma, in many instances discovered only after lethal complications: rupture, emboli, arrhythmias or congestive heart failure. The pathogenesis has been related to direct myocardial trauma with resulting contusion, hemorrhagic infiltration, necrosis and scar with progressive dilatation of this area forming a ventricular aneurysm.

In arteriosclerotic heart disease, the majority of ventricular aneurysms seen at surgery are associated with occlusion of the anterior descending branch of the left coronary artery, infarction of the anterolateral wall of the left ventricle, and subsequent aneurysmatic dilatation of the fibrotic area.

Furthermore, observations based on our experience in indirect myocardial revascularization (mammary implant procedures) demonstrate the improbability of trauma resulting in an intramyocardial hematoma, particularly in a normal myocardium with its unique anatomic and physiologic structure. Experimental studies confirm the fact that there is difficulty in creating an infarct from direct contusion of the heart unless
VENTRICULAR ANEURYSMS AND BLUNT CHEST TRAUMA

the trauma involves the coronary circulation.

Clinical evidence and an analysis of the reviewed cases suggest that coronary artery obstruction rather than direct myocardial trauma is the cause of aneurysm in patients surviving blunt chest trauma. The anterior and superficial position of the anterior descending coronary artery exposes it to possible damage from either direct compression between the sternum and underlying tissue or from sudden impact during rapid deceleration that pushes the heart against the anterior chest wall (Fig 3). In such cases, complete transection of the artery, severe intimal contusion with subsequent thrombosis or the formation of a subepicardial perivascular hematoma with compression and narrowing of the lumen are alternative injuries which might occur (Fig 4). Possible prolonged spasm of the artery9 due to the direct injury associated with the hemocoagulation, hypotension, hypovolemia and shock might also lead to occlusion of the vessel, first functional and later organic. In five of the cases reviewed, a definite lesion of the anterior descending coronary artery was shown angiographically or found at autopsy. In the other cases there was no mention of the status of the coronary arteries.

Failure to diagnose the infarct at the time of the accident is probably because other traumatic lesions (fractured ribs, pulmonary contusion, hemothorax, pneumothorax, transection of aorta, et cetera) are responsible for the pain and state of shock masking the symptoms of the infarct. In the cases reviewed, there is no report of an ECG done at the time of the accident.

Once the aneurysm has formed, it is not very different from arteriosclerotic aneurysms. The most frequent complications, especially rupture and emboli, probably are related to the youth of the patients who have a tendency to greater physical activity with consequent increase of cardiac activity.

The prognosis in these cases is poor. In the cases reviewed, all patients died except the six who underwent emergency surgical treatment of acute coronary occlusion is now being performed.

Electrocardiograms should be performed in the emergency room on all patients who have sustained chest trauma. If acute ECG changes are present, the possibility of direct trauma of the coronary arteries should be considered. If coronary cineangiography and saphenous vein bypass surgery can be performed within six to ten hours after trauma, perhaps a myocardial infarct would be averted.9

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