Mid-Systolic Sound Associated with Aortic Insufficiency and Bisferiens Pulse*

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Systolic ejection clicks, often associated with cardiac disease, can be distinguished from mid-systolic clicks of mitral and tricuspid origin, which are frequently benign. A different mid-systolic sound has been recognized, with timing similar to the mid-systolic clicks, which is associated with aortic insufficiency. This report describes a case in which such a sound was identified prior to aortic valve replacement, and disappeared with surgery, thus adding evidence to previous concepts of the origin of this sound, and its relationship to the hemodynamic abnormality associated with aortic valvular regurgitation.

The recognition and identification of extra sounds during cardiac systole poses a significant challenge to the clinician and investigator alike. Although the click of mitral prolapse has received much attention in recent years, lesser known causes must still be considered.

This report describes a mid-systolic sound associated with aortic insufficiency (without stenosis) and pulsat bisferiens, which disappeared following valve replacement. It confirms a previous explanation of the origin of the so-called aortic systolic gallop described first by Potain.1

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Case Report

The patient is a 49-year-old white man, previously in excellent health, who was referred for the new onset of symptoms of congestive heart failure. He had experienced dyspnea for several weeks, but had otherwise been well. Blood cultures gave negative results, and his congestive failure had failed to respond to administration of digitals and diuretics. He had had a single episode of rheumatic fever as a child and a subsequently documented heart murmur, but he had been previously free of cardiac symptoms. His family history was negative for cardiac disease. Medications at time of admission were digitals and a diuretic.

Physical Examination

The patient appeared his stated age and was comfortable at rest. With the patient in the sitting position, blood pressure was 110/88 mm Hg in the right arm, the pulse rate was 120, and respirations 14. He was afebrile. The carotid contour was bisferiens with a rapid upstroke. The jugular venous pressure was not elevated. The thyroid was not palpable. The chest was clear to auscultation and percussion. The cardiac examination revealed a visible apical impulse, and a palpable left ventricular lift in the fifth intercostal space in the anterior axillary line. There was a grade 3/6 systolic ejection murmur heard best along the lower left sternal border, and a grade 2/4 presystolic murmur, as well as a grade 3/4 diastolic blowing murmur along the left sternal border. A mid-to-late systolic sound was heard at the base, along the left sternal border and at the apex. A third heart sound gallop was present over the apex with the patient placed in the left lateral downward position. Other peripheral signs of aortic insufficiency were found, including Trousseau pistol shot sounds over the femoral arteries, and the Duriezien murmur. There was no edema or cyanosis, and the abdominal examination was normal.

Data from Noninvasive Tests

The chest x-ray film disclosed marked cardiomegaly with normal pulmonary vasculature. The electrocardiogram revealed left bundle branch block. Initial M-mode and 2D echocardiograms demonstrated left ventricular hypertrophy and dilatation. The left ventricular diastolic dimension was 7.2 cm. The aortic valve was thickened,

![Figure 1. Preoperative phonocardiographic recordings at the aortic area (AA) and apex with simultaneous carotid pulse tracing reveal a mid-systolic sound (C) occurring in the trough of the bisferiens pulse. A diastolic murmur (DM) and systolic murmur (SM) are noted.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21331/ on 06/26/2017)
and the root dilated. There was fluttering of the mitral valve consistent with aortic insufficiency. There was no evidence of mitral valve prolapse on either initial echocardiogram or any of multiple subsequent tracings.

Phonocardiogram prior to surgery (Fig 1) confirmed the findings on physical examination, i.e., a systolic ejection murmur, the decrescendo blowing murmur of aortic insufficiency, and the Austin-Flint rumble. The carotid pulse revealed the characteristic biferiens contour. Recordings at the apex, the left sternal border, as well as the pulmonic area displayed the presence of a mid-systolic sound occurring at the notch between the peaks of the biferiens pulse. The sound was approximately .30 seconds following the onset of the Q wave. The timing of this systolic sound did not vary with respiration. However, with expiration, the sound was seen to increase in intensity. The jugular-venous pulse and the apex cardiogram were normal.

The patient underwent cardiac catheterization which revealed normal right-sided pressures, an elevated left ventricular end-diastolic pressure, (124/22) and a dilated, hypokinetic left ventricle. The coronary arteries were normal. Aortic root injection revealed severe aortic insufficiency.

Hospital Course

The patient underwent aortic valve replacement with a No. 29 Edwards porcine prosthesis. His intraoperative and postoperative course was uneventful. Three weeks following operation, he had a repeat phonocardiogram which revealed (Fig 2) the disappearance of both the previously described biferiens carotid pulse, as well as the mid-systolic sound.

**Discussion**

Wolferth and Margolies' published the first graphic recording of what they called the "aortic" gallop. They asserted that a distinction must be made between mid-systolic clicks that have a high pitched clicky quality heard best over the apex, and the lower pitched sound heard best over the aorta. Although the timing in systole is often similar, the former usually occurred in the "absence of heart disease." The latter, on the other hand, was usually seen in the presence of significant disease: typhoid or other intense fevers, severe atherosclerosis, and aortic insufficiency. They did not hear the sound in normal subjects. A further point of distinction was the variability of the timing of the mid-systolic clicks, while the timing of the aortic sound remained constant. In our patient, the systolic sound was heard and recorded all over the precordium, and the timing was constant, although the intensity of the sound increased with expiration.

Interest in the systolic click itself was greatly stimulated by the publication by Barlow and Bosman of the first description of the mitral valve prolapse syndrome, and the recognition that most systolic clicks originated from the mitral (or tricuspid) valve apparatus and chordae and were generally benign in nature. The mid-systolic sound of aortic origin did not receive attention again until Fukuda et al re-examined the origin of the systolic sounds in aortic insufficiency. They identified two sounds of aortic origin. One sound (AK1) was early systolic in origin and did not correspond to previously described sounds. The second sound (AK2) occurred only in association with pulsus biferiens, was found in the presence of moderate-to-severe aortic insufficiency, and occurred at the notch between the peaks of the carotid pulse. This sound corresponds in all respects to the sound recorded in our patient. From associated flow data derived from the same study, the authors concluded that the origin of the aortic sound (AK2) was a result of the observed biphasic flow in which the outgoing secondary wave was thought to clash with the reflected primary wave with the resultant generation of a sound. Our demonstration that the sound disappears with the abolition of the biferiens pulse is entirely consistent with this hypothesis.

Our case offers evidence that the hemodynamic substrate created in significant aortic insufficiency results in the biferiens pulse and a mid- to late-systolic sound. Both phenomena disappear with correction of the primary valve lesion. It is difficult to draw conclusions as to the specific pathophysiology of sound origin, but it does advance the observations of Wolferth and Margolies, as well as Fukuda et al, in establishing the close relationship between the presence of the systolic sound and the biferiens pulse. It further emphasizes that having made the distinction between ejection sounds and mid-late systolic sound, one must further define the latter sound. In addition to the usually benign clicks originating from the mitral valve, there are similarly timed sounds that may be the harbinger of more severe pathology.

**Acknowledgment**

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Osteomyelitis of Both Clavicles as a Complication of Subclavian Venipuncture*

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Complications of subclavian vein catheterization are common and include pneumothorax, hemothorax, and sepsis. Osteomyelitis is a rare complication. The present report describes a patient with osteomyelitis of both clavicles due to subclavian vein venipuncture, in whom fever and chills were absent and the sole clinical finding was local pain and tenderness in the involved area.

Percutaneous subclavian venipuncture has become a usual route of insertion of a central venous catheter. It is used for monitoring central venous pressure, and for long-term intravenous therapy. Complications of this procedure include subcutaneous hematoma, pneumothorax, hemothorax, and sepsis.

In the present communication, a patient is described with osteomyelitis of both clavicles, caused by *Pseudomonas aeruginosa*, after subclavian venipuncture.

**CASE REPORT**

A 69-year-old woman was admitted to another department because of severe shortness of breath. She has been known to suffer from rheumatic valvular disease and congestive heart failure. On examination, she appeared acutely ill, dyspneic, and cyanotic. Several hours later her condition deteriorated, the Po2 was 40 mm Hg, and she was intubated. A subclavian venipuncture at the right side was unsuccessfully attempted, followed by insertion of the catheter into the left subclavian vein. The catheter was in place for four days. The patient's condition improved, and the catheter was drawn out.

On the fifth day, she began to complain of pain in the left sternoclavicular joint. The area was red, warm, and tender. X-ray examination of the left sternoclavicular joint was without pathologic findings. The patient was discharged with analgesic therapy.

One month later, the patient was readmitted because of purulent discharge from the site of the left venipuncture accompanied by severe pain. The ESR was 60/100 mm/h (Westergren). A tomogram of the clavicles showed clearly demonstrated borders of the head of the right clavicle, and rarefaction of the head and medial portion of the left clavicle, with sclerosis in the border between the involved and the healthy bone (Fig 1).

Cultures from the purulent discharge yielded *P aeruginosa*. The patient underwent surgical exploration with removal of the necrotic bone. Parenteral antibiotic therapy with tobramycin, 50 mg x 3, was instituted, and the pain subsided. Three weeks later, the patient was admitted to our department because of pains in her right sternoclavicular joint. Tomogram of the right clavicle showed rarefaction of the head of the right clavicle as well as sclerosis with considerable irregularity and thickening of the left clavicle (Fig 2). The patient was treated with antibiotics, and her condition improved.

**DISCUSSION**

Infection is one of the complications associated with vein catheterization. However, sepsis is a rare condition and depends on the time the catheter was in place and on the type of fluids infused.

In the present case, the catheter was inserted for four days without signs of sepsis or local infective foci. In three cases of clavicle osteomyelitis after subclavian venipuncture reported in the literature, the patients underwent abdominal surgery, and in two cases, fever and sepsis appeared before insertion of the catheter. It is conceivable to assume that in our patient the causative organisms were inoculated directly into the clavicular periosteum and did not propagate from distant foci. Moreover, the infective process involved both clavicles, a complication that, to the best of our knowledge, has not been described in the English literature.

Since there was no clinical evidence for the existence of

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**FIGURE 1.** Tomogram of the clavicles. Normal architecture of the right clavicle, and rarefaction of head and medial border of left clavicle.