Transmission of audible praecordial gallop sounds to right supraclavicular fossa

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To evaluate the significance of audible gallop sounds in the right supraclavicular fossa we performed simultaneous external heart sound recordings at 50 and 100 Hz at the left ventricular apex, left sternal border, and right supraclavicular fossa in 50 patients with audible gallop sounds at the left ventricular apex. In each patient heart sounds were recorded with a simultaneous jugular phlebogram, apex cardiogram, and carotid pulse tracing. In 44 patients an apical fourth heart sound coincident with the ‘a’ wave of the apex cardiogram was recorded, and in 32 (73%) the fourth heart sound was audible and recordable in the right supraclavicular fossa. A left ventricular third heart sound, coincident with the rapid filling wave of the apex tracing, was present in 25 patients but was recorded in the right supraclavicular fossa in only 7 (28%). Intracardiac phonocardiography (high-fidelity catheter) was performed in six patients with left ventricular gallop sounds and in each instance arterial transmission of the third or fourth heart sound, or both, was present. Five additional patients had a prominent low-frequency event recorded in the right supraclavicular fossa preceding the peak of a prominent jugular venous ‘a’ wave, but only two had a soft parasternal fourth heart sound. Intracardiac phonocardiography in these five patients failed to reveal transmission of right ventricular gallop sounds to the superior vena cava.

We conclude that since left ventricular gallop sounds commonly are transmitted to the right supraclavicular fossa auscultation in this area is often helpful in their detection. In addition, a prominent jugular venous ‘a’ wave sometimes produces recordable presystolic vibrations that are occasionally audible as well.

Once the clinician has detected a gallop rhythm during routine auscultation of the praecordium he is faced with the problem of deciding whether these heart sounds emanate from the right or left ventricular chamber. A separate but related problem commonly faced in clinical practice is the detection of such gallop rhythm in patients with obesity or obstructive lung disease. Since it has been observed that gallop sounds can often be heard with the stethoscope in the supraclavicular fossae (W. P. Harvey and A. DeLeon, 1969, personal communication) we sought to determine the frequency with which this happens in respect of the right supraclavicular fossa. In addition, we sought to ascertain whether transmission of such sounds is specific for events which originate in the left ventricle.

Patients and methods

A total of 60 patients were studied. Fifty had organic heart disease of left ventricular origin except for five patients with a physiological third heart sound. Ten patients had organic cardiac disease involving only the right ventricle. In each patient praecordial gallop sounds were heard by more than one examiner. Auscultation in the supraclavicular fossa was performed using the bell of the stethoscope, which was placed in the right supraclavicular fossa with the patient's head turned slightly to the left. The examination was performed in held expiration with the patient instructed to avoid a Valsalva manoeuvre. Simultaneous phonocardiograms were recorded at the cardiac apex, left sternal border, and right supraclavicular fossa at 50 and 100 Hz. All recordings were made with the patient lying supine during held expiration using an Elema-Shonander Mingograph-75 with EMT-25b contact microphones. With each phonocardiogram a jugular phlebogram, apex cardiogram, and carotid pulse tracing were also obtained. Intracardiac phonocardiography was performed in 11 patients using a high-fidelity microman-
meter tipped catheter. Six of these patients had gallop sounds originating in the left ventricle, while five patients exhibited right ventricular gallop sounds.

Results

An apical fourth heart sound was audible in 44 patients. In 32 (73%) of these patients it was recorded in the right supraclavicular fossa (Fig. 1). In each instance the fourth heart sound was coincident with the ‘a’ wave of the simultaneously recorded apex cardiogram (Fig. 2). Twenty-eight patients had an apical third heart sound coincident with the rapid filling wave of the apex cardiogram. Only seven (28%) of these were recorded in the right supraclavicular fossa. In six patients gallop sounds which were recorded in the left ventricle displayed discrete arterial transmission (Fig. 3).

An additional group of five patients who had evidence of right ventricular disease also were studied by external phonocardiography. Two of these patients had primary pulmonary hypertension, one had chronic obstructive lung disease, one had an atrial septal defect (pulmonary to systemic flow ratio of 3:1), and one had pulmonary stenosis. Each patient exhibited a prominent but low-frequency sound in the neck which preceded the peak of a prominent jugular ‘a’ wave (Fig. 4) and in two of these patients a presystolic sound was recordable at the left sternal border. In one other patient the ‘v’ wave of the jugular venous pulse was

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**Figure 1** Incidence of radiation of praecordial gallop sounds to right supraclavicular fossa (SCF). $S_3$ = third heart sound. $S_4$ = fourth heart sound.

**Figure 2** Typical example of transmission of an apical fourth heart sound ($S_4$) to the right supraclavicular fossa (SCF). The $S_4$ is coincident with the ‘a’ wave of the simultaneously recorded apex cardiogram (ACG).
associated with an apparent third heart sound but a third heart sound was not recorded at the lower left sternal border. In an additional group of five patients with right ventricular disease intracardiac phonocardiograms demonstrated either a right ventricular third or fourth heart sound in each. These gallop sounds, however, could not be recorded in the superior vena cava.

Discussion

Auscultation in the supraclavicular and carotid regions is usually performed to detect carotid or subclavian bruits, cervical venous hums, or to trace the transmission of praecordial systolic murmurs. In the past little attention has been directed towards the appreciation of either the normal heart sounds or gallop sounds in these areas. Others have observed that gallop sounds can often be heard with the stethoscope in the supraclavicular fossae (W. P. Harvey and A. DeLeon, 1969, personal communication). Groedel and Miller (1944) were among the first to use modern methods to study the heart sounds over the great vessels of the neck. In each of their 24 cases the first and second heart sounds were recorded over the jugular and carotid regions. In one of their patients the heart sounds, including both third and a fourth heart sound, were recorded more easily over the neck vessels than over the cardiac apex. The authors emphasized that sounds and murmurs originating in the heart are transmitted in the direction of the flow of blood along the walls of the great vessels. They concluded, however, that the arteries did not seem important for the transmission of the heart sounds and murmurs with the exception of the audible events at the aortic valve region.

Our results show that left ventricular gallop sounds, especially the fourth sound, are often transmitted to the right supraclavicular fossa. Less frequently a third sound gallop can be recorded in the same region. Recordings were made over the right supraclavicular fossa since we found it difficult to obtain adequate recordings over the carotid vessels. It is our contention that these sounds, which are generated in the high pressure left ventricular chamber, are propagated antegrade by means of transverse vibrations along the walls of the great arteries, as has been reported experimentally by Farber and Purvis (1963). Not only may these sounds be recorded after a variable but short delay in the right supraclavicular fossa but when the bell of the stethoscope is placed lightly over the right supraclavicular fossa such gallop sounds are often easily audible. Why the third heart sound should be transmitted so much less frequently is not clear, but this phenomenon may be related to the active nature of fourth heart sound production— that is, to atrial contraction—compared with the essentially passive nature of third heart sound production. Whether the presumably greater kinetic energy imparted by the former accounts for this difference must remain speculative.

During the course of our studies it became evident that it was possible also in patients with right ventricular dysfunction to record apparently promi-
nent third and fourth heart sounds in the right supraclavicular fossa. However, these easily recordable events were less commonly audible in the right supraclavicular fossa, possibly because of their lower frequency (less than 50 Hz). Audible sounds corresponding to recordable events in the neck were not present upon precordial auscultation, nor could they be recorded along the left sternal border. We contend that prominent jugular venous pulsations may lead to the recording, and at times to the auscultation, of these vibrations. A similar explanation for the occurrence of presystolic clicks recorded over the jugular venous pulsation in patients with tricuspid regurgitation has been offered by Abinader (1973). By contrast, when right ventricular gallop sounds were recorded within that chamber transmission to the superior vena cava could not be demonstrated. This observation is consistent with the hypothesis that arterial sounds are transmitted through vessel walls. Presumably the arterial wall is more conducive to such sound transmission than are thin-walled veins.

The diagnostic and prognostic importance of precordial gallop sounds is well known. Both third and fourth heart sounds can be difficult to hear for a variety of reasons. The observation that the left ventricular fourth sound and, less frequently, the left ventricular third sound are transmitted to the supraclavicular fossae increases the likelihood that they will be more frequently recognized in patients in whom routine precordial auscultation is difficult, such as commonly occurs in patients with obesity or hyperinflation of the lungs.

References


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