

# The Auscultatory Gap in Arteriosclerotic Heart Disease

By SIMON RODBARD, M.D., AND JACK MARGOLIS, M.D.

Auscultatory gaps, found in some patients with arteriosclerotic heart disease, can be elicited in others by reducing blood flow in the extremity. Reactive hyperemia eliminates the gap in every instance. The gap therefore appears to represent a vasospastic disturbance that limits the blood flow to the extremities, despite the presence of an enhanced pressure head.

**T**HE auscultatory gap, recorded commonly in patients with systolic hypertension or with aortic stenosis, probably represents an undefined disturbance in cardiovascular function. Aside from this, the gap may have practical importance, since significant errors in the estimation of the blood pressure may result when its presence is not recognized.<sup>1-5</sup> The present study of the gap extends previous investigations into mechanisms of sound production at the brachial artery during the indirect measurement of the blood pressure.<sup>6-10</sup> Technics for enhancing or eliminating the gap, and an analysis of its physiologic mechanisms and clinical significance are presented.

## METHODS

Twenty ambulatory male patients, 65 to 78 years of age, with systolic blood pressures of 170 mm. Hg or higher, were selected for study. The clinical diagnosis of arteriosclerotic heart disease was based on physical examination and electrocardiographic and roentgenologic study.

At least 10 blood pressure determinations were made on each patient. In some of these, a dynamic microphone placed in the antecubital fossa transmitted the arterial sounds to a Sanborn twin-beam instrument, which recorded them simultaneously with lead II of the electrocardiogram. Blood pressure determinations were made as the cuff pressure was deflated at about 3 mm. Hg per second.

From the University of Buffalo Chronic Disease Research Institute, Buffalo, N. Y., and the Medical Service, Veterans Administration Hospital, Big Spring, Tex.

Introductory phases of this study were begun at the Cardiovascular Department (Dr. L. N. Katz, Director), Medical Research Institute, Michael Reese Hospital, Chicago, Ill.

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## RESULTS

Of the twenty patients selected for the study only 3 had a gap while recumbent (table 1). In the sitting position, 2 of these continued to have a gap and 4 other patients also showed gaps. In the standing position, gaps were found in 7 of the subjects, but not always the ones who showed gaps in the recumbent or sitting positions. Eleven of the patients had a gap at least once during the testing periods. While there was no consistent effect of body position, the increased likelihood of gap in the sitting or standing position was apparent.

The gaps began 10 to 50 mm. Hg below the systolic pressure and had spans as large as 30 mm. Hg. In general, patients with higher systolic blood pressures had a greater tendency to have a gap.

*Comparison of Auscultation and Phonocardiography.* In some patients, gaps noted on direct auscultation of the arterial sounds were shown to be due to the subaudible amplitude of vibrations in the gap zone, which electronic amplification made clearly audible. Phonocardiographic records showed the vibrations in the gap range to have a markedly lower amplitude than the sounds in the other pressure ranges (fig. 1). These results indicated that the gap was due in part to a limitation in hearing, rather than to a complete absence of vibrations. This concept was supported by studies in which an induced reactive hyperemia enhanced the intensities of all the arterial sounds.

*Elimination of the Gap.* The gap was eliminated in every instance by producing a local reactive hyperemia. This was accomplished by increasing the cuff pressure to a level above systolic and then having the patient clench the fist 25 times; the increased blood flow following release of the tourniquet brought all the ar-

TABLE 1.—Auscultatory Gap in Twenty Patients

Case	Age	Diagnosis	Blood Pressure (mm. Hg)	Ranges of the auscultatory gap (mm. Hg)						
				Lying	Sitting	Standing	With tourniquet			After local exercise
							Lying	Sitting	Standing	
		Generalized arteriosclerosis plus:								
1	68	—	180/90	0	0	0	0	0	0	0
2	72	—	170/90	0	0	0	0	0	0	0
3	76	—	195/65	0	0	0	0	0	0	0
4	66	Chronic bronchitis	210/100	0	0	0	190-170	190-170	180-160	0
5	66	Osteoarthritis	190/100	0	0	0	160-150	160-150	170-160	0
6	66	Meningovascular syphilis	190/90	0	0	0	0	0	160-130	0
7	73	Meningovascular syphilis	180/90	0	0	0	160-130	160-140	160-140	0
8	76	—	175/65	0	0	0	150-140	140-120	140-120	0
9	77	Meningovascular syphilis	180/70	0	0	0	0	160-140	160-140	0
10	65	—	240/70	0	210-180	0	0	210-170	200-180	0
11	66	Chronic glomerulonephritis	250/110	0	0	210-200	230-200	220-200	230-200 170-160	0
12	68	Osteoarthritis	260/110	0	0	210-180	200-190	200-180	230-210	0
13	70	Chronic glomerulonephritis	230/110	0	0	210-190	0	210-180	210-180	0
14	70	Carcinoma of lung	190/60	0	170-140	0	170-140	160-120	0	0
15	71	Carcinoma of lung	230/90	0	200-170	0	200-180	210-200	0	0
16	71	Osteoarthritis	175/60	0	0	150-140	160-140	160-145	160-140	0
17	78	—	180/90	0	140-120	0	160-140	160-140	160-140	0
18	78	—	220/90	195-170	0	190-170	200-180	0	190-170	0
19	65	—	210/90	190-170	200-180	200-180	190-160	190-160	190-170 160-140	0
20	65	—	290/110	240-220	240-220	260-240 210-200	240-220	240-220	250-220	0

terial sounds above the hearing threshold. Such localized exercise has been shown by plethysmographic<sup>11</sup> and other technics to produce a marked increase in blood flow. This effect was enhanced when the blood flow to the extremity was restricted by tourniquet during the exercise period. Coincident with the increased blood flow, all the arterial sounds were markedly increased in intensity and duration, and a gap was no longer present.

It has been shown in previous studies that reactive hyperemia, enhancing the blood flow to the region, increases the intensity and duration of the arterial sounds.<sup>9</sup> The maneuver has no significant effects on the central blood pressure or on the pulse wave.

The results suggested, therefore, that the occurrence of the gap was associated with a reduced blood flow to the extremity. This concept was tested by the application of a tourniquet to the forearm for the purpose of reducing the blood flow to the arm.

*Induced Auscultatory Gaps.* To diminish the blood flow to the arm, a tourniquet, consisting of a sphygmomanometer cuff inflated to a pressure of 250 mm. Hg, was applied to the forearm. The blood pressure was then recorded according to conventional technics by increasing the pressure in the cuff on the upper arm to a level above systolic and permitting the cuff pressure to fall slowly.

Under these conditions all the arterial

sounds were markedly decreased in intensity and duration, and the likelihood of a gap was increased. A gap was now found in all but 3 of the 20 subjects. The effect of position was inconsistent in the occurrence of these gaps. Similar tourniquet studies carried out on 20 individuals without evidence of cardiovascular disease produced gaps in none of the subjects tested.

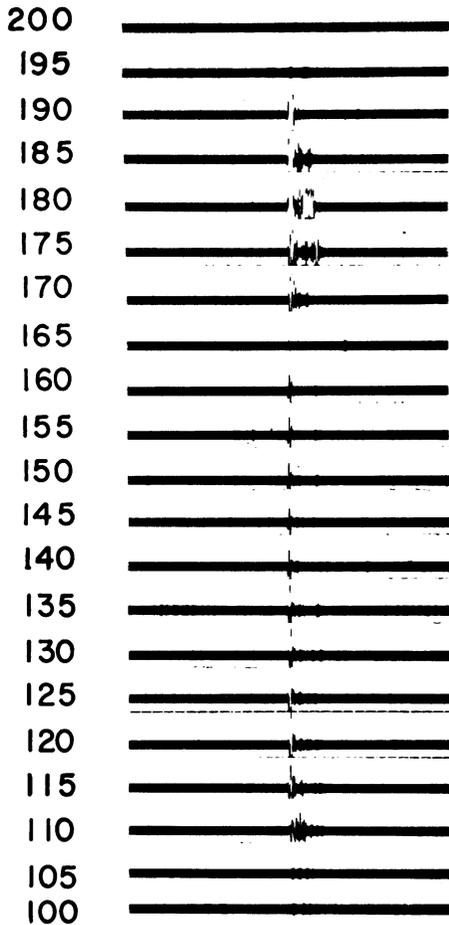


FIG. 1. Arterial sounds demonstrating the auscultatory gap. The figure comprises a set of arterial sounds obtained as the cuff pressure was lowered from above systolic to below diastolic levels in a single continuous recording in a patient. Deviations from the horizontal galvanometer trace represent sound vibrations. The arterial sounds at systolic pressure (190 mm. Hg) are seen to have a greater intensity than those in the gap range (165 mm. Hg) where they virtually disappear. The amplitude and duration of the vibrations then increase again as the cuff pressure falls until the diastolic pressure is reached (105 mm. Hg) at which time no further audible vibrations are noted.

These results, showing the increased tendency to gap in patients with arteriosclerotic heart disease during maneuvers that reduce blood flow to the extremity, indicate that the volume of blood flowing into the extremity is an important mechanism in the genesis of the gap. Since the systolic and mean blood pressures are high in such patients with arteriosclerosis, a vasoconstrictive tendency in the blood vessels of the extremity would appear to be suggested.

#### DISCUSSION

Our findings of spontaneous gaps in 3 of the 20 subjects examined suggest that this phenomenon is not uncommon in the aged patient with arteriosclerotic heart disease and systolic hypertension. The likelihood of a gap being present was increased in patients when the systolic blood pressure was excessively elevated, i.e., above 220 mm. Hg. It was found less commonly when the patient was recumbent than when he was sitting or standing. A double gap was elicited on 3 occasions.

The intensities and durations of the arterial sounds heard during blood pressure measurement are dependent, in part, on the relation of cuff pressure to blood pressure. These sounds are conventionally divided into 5 phases, but actually the pattern of the sounds may vary significantly from individual to individual, as well as changing with various cardiovascular conditions. The systolic level is usually characterized during the decompression of the cuff by a tapping sound (first phase) that increases in intensity and then sometimes changes to a softer sound (second phase). As the cuff is further deflated, an added murmur-like rumble increases the duration of the sound, ushering in the third phase. When the cuff pressure approaches the diastolic level, the sounds become muffled (fourth phase) and finally can no longer be heard (fifth phase).

The auscultatory gap in arteriosclerotic heart disease is most frequently noted within 40 mm. Hg of the systolic pressure when the arterial sounds become softer, during the second phase.

The present data are consonant with the point of view that the occurrence of the gap is

related to a reduction in the intensities of the vibrations in the gap range, rather than to their complete elimination. That vibrations are actually produced in the gap range is suggested by the consistent demonstration of sounds in this range with adequate electronic amplification. The amplitudes and durations of the sounds in the gap range, however, are significantly less than those recorded in other ranges of the blood pressure determination.

The magnitude and duration of the vibrations produced at the compressed artery are affected to a marked degree by the blood flow through the vessel, as indicated by both clinical and hydrodynamic studies. With increased flow the intensities and durations of the sounds are increased. This effect is seen strikingly after the induction of reactive hyperemia, which intensifies all the arterial sounds so that a gap can no longer be demonstrated.

By contrast, when the blood flow through the compressed artery is obstructed as by a tourniquet immediately beyond the stethoscope, the arterial sounds become much weaker and more difficult to hear. The likelihood of a gap is thereby enhanced. After placement of the tourniquet a gap could be elicited in patients in whom none was discerned during the previous blood pressure measurements.

The pulse is present at the radial artery during the gap phase even though the sounds at the brachial artery fade. The pulsations may be very forceful even though only a limited blood flow is taking place through the palpated artery.<sup>8</sup> Thus, the strength of the pulse, often used as an index of extremity blood flow, may be marked, despite evidence that the blood flow is restricted.

The present findings indicate that the arterial sounds have value in the appraisal of the clinical status of the patient. Thus, when the sounds are faint, a reduced blood flow to the extremity may be presumed. This reduced peripheral flow might be expected in older patients with systolic hypertension and arteriosclerotic heart disease in whom cardiac reserve and output are limited. It is likely that active vasoregulatory mechanisms<sup>12</sup> ration the major portion of the restricted cardiac output to vascular beds in the central nervous system,

the heart and other vital structures, while the extremities receive only a limited flow. This effect is probably enhanced in the sitting or standing position. Clinical evidence of a reduced blood flow to the arms and legs in arteriosclerotic heart disease is seen in the cold extremities, intermittent claudication, and peripheral cyanosis so common in this age period. The presence of diminished arterial sounds and especially of a gap may thus be considered as a sign of peripheral vasospastic activity.

#### SUMMARY

Evidence is presented that auscultatory gaps heard in patients with arteriosclerotic heart disease may be associated with a reduced blood flow to the extremity. Further reduction of such flow, as by application of a tourniquet, increased the likelihood of a gap. Contrariwise, increase in extremity blood flow eliminated the gap in every instance. Vibrations are present in the gap range, but these are subaudible; electronic amplification may bring these sounds to audible levels. The clinical implications of these findings are discussed.

#### SUMMARIO IN INTERLINGUA

Es documentate le these que le lacunas auscultatori notate in patientes con arteriosclerotic morbo cardiac es associate con un reduction del fluxu sanguinee verso le extremitates. Reductiones additional de ille fluxu—per exemplo per le application de un tourniquet—augmenta le probabilitate del occurrentia de lacunas. Del altere latere, le augmento del fluxu sanguinee verso le extremitates eliminava le lacunas in omne casos. Il existe vibrationes durante le lacunas, sed iste vibrationes es infra le limines del audibilitate. Per medio de un amplification electronic il es possibile render tal sonos audible. Es discutate le implicationes clinic de iste constatationes.

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**Rifkin, H.:** *The Kimmelstiel-Wilson Syndrome: Clinical Aspects.* *J. Mt. Sinai Hosp.* **23**: 674 (Sept.-Oct.), 1956.

Although there is little doubt that a clinical syndrome characterized by diabetes, edema, hypertension, proteinuria, azotemia, and diabetic retinopathy is found in about 10 to 15 per cent of patients with the specific Kimmelstiel-Wilson nodular lesion of the glomerulus, similar symptoms and signs may be found in patients without this specific lesion. The diagnosis of this syndrome is further helped by the finding of doubly refractile lipid cells and casts in the urinary sediment and may be confirmed during life by renal biopsy. It is also well known that anatomically proved cases of this condition may not be identified during life because of the paucity or lack of clinical findings. The earliest clinical signs of the disease may be detected in diabetic patients by repeated ophthalmoscopic examinations for the retinal micro-aneurysms that coexist with the glomerular involvement. Doubly refractile bodies in the urine may also possibly be an early finding.

In regard to pathogenesis, controversy exists as to the effects of control of diabetes on the development of the renal and retinal lesions. In the author's experience no real differences in the insulin requirements or frequency of episodes of acidosis are apparent between diabetic patients with and without the specific renal lesion. It has been established that the duration of diabetes is an important factor in pathogenesis, but the time factor may vary from as little as 6 years to as long as 25 years. Some interesting biochemical alterations regarding tissue concentrations of mucopolysaccharides, lipids, and lipoproteins have been reported in this condition, but the present evidence is too fragmentary to allow a definite causal relationship of these substances to the renal and glomerular lesions. Recent studies have also indicated a possible relationship between increased pituitary-adrenal cortical activity and the development of the specific vascular lesions, but it is much too early to come to any definite conclusions. As yet no real therapeutic regimen is available for these patients.

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