Characterization of Auscultatory Gaps With Wideband External Pulse Recording

Seymour G. Blank, James E. West, Franco B. Müller, Mark S. Pecker, John H. Laragh, and Thomas G. Pickering

Three types of auscultatory gaps, called G1, G2, and G3, that occur during blood pressure measurement have been identified by using wideband external pulse recording. We have previously shown that the wideband external pulse recorded during cuff deflation can be separated into three components (K1, K2, and K3), one of which (K2) is closely related to the Korotkoff sound. G1 occurs with cuff pressure just below systolic and is characterized by the presence of K1 and K2 with intermittent disappearance of K2. G1 gaps are related to a phasic decrease of arterial (systolic) pressure and were exhibited by 13 of 60 hypertensive patients. G2 gaps are related to a phasic increase of arterial (diastolic) pressure, occur when cuff pressure is just above diastolic, and are characterized by the presence of K1, K2, and K3 with intermittent disappearance of K2. Seven of 60 hypertensive patients exhibited a G2 gap. G3 gaps occur with cuff pressure between systolic and diastolic and are characterized by an underdeveloped or blunted K2 signal. Three of 60 hypertensive patients exhibited a G3 gap. The identification of auscultatory gaps in relation to the wideband external pulse provides a qualitative measure of their existence, can be of significant value in better understanding aspects of the auscultatory blood pressure measurement technique, and provides an objective basis with which to better understand the mechanisms that cause them. (Hypertension 1991;17:225–233)

The auscultatory gap (i.e., loss and reappearance of Korotkoff sounds during blood pressure cuff deflation in the absence of cardiac arrhythmias) was first recognized by Krylov in 1906,1 1 year after N.S. Korotkoff described the auscultatory blood pressure measurement technique. Cook and Taussig4 are generally credited as the first to recognize the clinical importance of these gaps. When present, serious errors in blood pressure determination can result, even with persons well trained in the auscultatory technique.

We have previously shown that the wideband external pulse recorded by a pressure sensor positioned over the brachial artery and under the distal portion of the blood pressure cuff during deflation can be separated into three components (K1, K2, and K3), one of which (K2) is closely related to the Korotkoff (K) sound.5 K1 and K3 are of low frequency and unrelated to audible sound (See Results for further description of K1, K2, and K3).

The purpose of this study was to characterize the phenomenon of auscultatory gaps by using wideband external pulse recording. The identification of these gaps in relation to the wideband external pulse can be of significant value in better understanding aspects of the auscultatory blood pressure measurement technique and may provide an objective basis with which to better understand the mechanisms that cause them.

Methods

The results reported here are derived from previous experience5 and 60 additional unselected hypertensive patients at The New York Hospital-Cornell University Medical College Cardiovascular Center. All subjects gave informed consent to the study protocol.

The wideband external pulse was recorded with a specially designed (AT&T Bell Laboratories, Murray Hill, N.J.) Foil Electret Sensor (FES) that is similar in principle to conventional electret microphones used for airborne sound reception. When coupled to a high input impedance amplifier, the FES has a flat frequency response from below 0.1 Hz to above 2,000 Hz. Detailed characteristics of this sensor have been described earlier.6,7

From the Cardiovascular Center (S.G.B., F.B.M., M.S.P., J.H.L., T.G.P.), The New York Hospital-Cornell University Medical Center, New York, and the Acoustics Research Department, (J.E.W.) AT&T Bell Laboratories, Murray Hill, N.J.

Address for correspondence: Seymour G. Blank, PhD, Cardiovascular Center, The New York Hospital-Cornell University Medical Center, 1300 York Avenue, New York, NY 10021.

Received February 6, 1990; accepted in revised form September 11, 1990.
Recordings were obtained with the subject in either the supine or the seated position. Wideband external pulses were recorded from above systolic to below diastolic pressure with the FES positioned over the brachial artery and under the distal portion of the blood pressure cuff during cuff deflation. The signal from the FES was coupled into a high impedance (10^9 Ω) electrometer amplifier (model 600B, Keithley Instruments, Inc., Cleveland, Ohio) and then into a direct-current DCV-20 amplifier of a VR6 physiological recording system (Electronics for Medicine, Pleasantville, N.Y.). The electrocardiogram was also recorded.

In 30 of the hypertensive subjects, simultaneous auscultation was performed by a trained observer. The trained observer used a switch to mark the onset and disappearance of sound. The stethoscope was modified to enable Korotkoff sounds to be recorded during cuff deflation. This was accomplished by adding a "Y" connector and coupling the additional air-filled pathway to a pulse transducer (Cambridge Electronics, Ossining, N.Y.). The Cambridge pulse transducer was coupled to a VR6 V2207 phonocardiographic amplifier.

In place of a stethoscope in the other 30 hypertensive subjects, the external pulse distal to the blood pressure cuff was recorded with an HP21050A contact sensor (Hewlett Packard, Andover, Mass.) coupled into two separate electronic filters. The first was a high pass filter with a lower 3 dB cutoff frequency of 50 Hz to simulate a stethoscope signal, and the second was a filter whose band pass frequency range was from 0.1 Hz to 50 Hz to record (and detect the presence of) pulse wave transmission distal to the blood pressure cuff.

The pressure in the cuff was controlled by an ambulatory blood pressure monitor (ABPM-630, Colin, South Plainfield, N.J.) or an E-10 cuff inflator (Hokanson, Issaquah, Wash.) and was read both by a mercury column and a pressure transducer (model T4812 AD-R, Gould-Statham, Oxnard, Calif.) connected to a PDV-20 pressure amplifier in the VR6. The cuff pressure deflation rate for the Colin instrument was 3–4 mm Hg/sec. When using the Hokanson E-10, cuff pressure was manually deflated by using a rotary pressure regulator handle, and with minimal training, the cuff pressure could be deflated smoothly at a rate of 2–4 mm Hg/sec. Occasionally, to more clearly elucidate auscultatory gap phenomena (Gl, Figures 3, 4, and 5; G2, Figure 6), the subject was asked to breathe deeply, thus increasing phasic variation of blood pressure while cuff pressure deflation rates were reduced below 2 mm Hg/sec. However, the results reported (i.e., general nature and relative frequency of each type of gap) were based on each subject's normal, unaltered breathing pattern and a 2–4 mm Hg/sec cuff pressure deflation rate.

A noninvasive beat-to-beat representation of the arterial pressure was provided by a Finapres (model 2300, Ohmeda, Englewood, Colo.) finger pulse recording on the opposite arm. The Finapres device has been shown to accurately reflect changes in intra-arterial pressure. Each channel of the VR6 recorder was sampled at 500 Hz by a 12-bit analog-to-digital converter for storage into an IBM PC/AT computer using the CODAS (Dataq, Akron, Ohio) data acquisition software.

Auscultatory gaps were characterized by comparing the auscultatory markers or stethoscope signals with the corresponding wideband external pulse.

Results

A normal auscultatory blood pressure determination is shown in Figure 1 as cuff pressure is reduced...
WIDEBAND EXTERNAL PULSE (FES)

CUFF PRESSURE

> SP

K1

a

SP

K1+K2

b

K2

c

K1+K2+K3

d

MP

K1+K2+K3

e

≤ DP

K3

f

ECG

0.5 sec

FIGURE 2. Representative recordings of wideband external pulse (FES) recorded at various levels of cuff pressure. The timing of each trace is linked to the electrocardiogram. Panel a: With cuff pressure above systolic pressure (SP), a low frequency signal, K1, is observed (pure K1). Panel b: With cuff pressure at systolic pressure, a relatively high frequency signal, K2, appears. K2 usually appears as a triphasic signal with an initial negative deflection (K2-a), a rising upstroke (K2-b), and a second negative deflection (K2-c). Panel c: As cuff pressure is reduced further below systolic pressure, a third component, K3, appears. Panel d: With cuff pressure approximately equal to mean pressure (MP), K3 increases in amplitude and dominates the observed signal. Panel e: With cuff pressure just above diastolic, the domination of signal by K3 obscures second negative deflection of K2 (K2-c). Presence of K2 reflected by negative notch (K2-a,b). Panel f: With cuff pressure at diastolic pressure (DP) and below, K2 disappears and K1 can no longer be independently observed, leaving a pure K3 signal.

from above systolic to below diastolic pressure. The Korotkoff sounds appear and disappear when the cuff pressure drops below systolic and diastolic pressures, respectively, as indicated by the auscultatory marker. Despite the absence of signal from the stethoscope (K sound) channel with cuff pressure above systolic and the diminished stethoscope signal with cuff pressure below diastolic, there was activity from the FES at all levels of cuff pressure. This activity has been described in detail previously and is shown in Figure 2.

Briefly, K1 is a low frequency signal seen with cuff pressures above systolic and not visually present below diastolic. K2 is a high frequency signal, related to the Korotkoff sound, that appears at systolic and disappears at diastolic pressure. K2 usually appears initially as a triphasic signal with an initial negative deflection (K2-a), a rising upstroke (K2-b), and a second negative deflection (K2-c). K3 is a large amplitude, low frequency signal that appears at cuff pressures between systolic and diastolic and is present with cuff pressures below diastolic.

By using wideband external pulse recording during normal blood pressure cuff deflation, three types of auscultatory gaps were identified and designated G1, G2, and G3.

G1 Gap

The first and most prevalent gap to be identified has been designated G1, which was exhibited by 13 of the 60 hypertensive patients. This type of auscultatory gap occurred when cuff pressure was just below systolic. It was characterized by the visual presence of K1 and K2 when sound was heard with intermittent disappearance of K2 when sound disappeared. A pulse distal to the blood pressure cuff could not be observed during G1.

G1 can be explained by a phasic decrease of arterial (systolic) pressure. As seen in Figure 3, the silent period (no K sound signal output) coincides with the phasic decrease of arterial systolic pressure below the cuff pressure. In this example, the auscultatory marker was activated when sound initially appeared and when sound reappeared. Although there was no output from the stethoscope (K sound) channel, there was activity from the wideband external pulse (FES). This activity can be seen in Figures 4 and 5. When arterial systolic pressure dipped below the cuff pressure, the Korotkoff sound disappeared along with K2 (Figure 4). When arterial systolic pressure increased sufficiently above cuff pressure, both the Korotkoff sound and K2 reappeared (Figure 5).

G2 Gap

The second type of gap, G2, occurred when the cuff pressure was just above diastolic. Seven of the 60 hypertensive patients exhibited a G2 gap. G2 was characterized by the presence of K1, K2, and K3 when sound was heard, with intermittent disappearance of K2 when sound disappeared. A pulse distal to
the blood pressure cuff could be observed when G2 occurred.

G2 can be explained by a phasic increase of arterial (diastolic) pressure. As seen in Figure 6, the silent period coincides with the phasic increase of arterial diastolic pressure above the cuff pressure. In this example, the auscultatory markers show where sound initially and finally disappeared. Although there was no output from the stethoscope (K sound) channel, there was activity from the FES. When arterial diastolic pressure increased above the cuff pressure, the Korotkoff sound disappeared along with K2. When arterial diastolic pressure decreased sufficiently below cuff pressure, both the Korotkoff sound and K2 reappeared.

**G3 Gap**

Three of the 60 hypertensive patients exhibited the third type of gap, G3, which occurred with cuff pressure between systolic and diastolic. This can be seen in Figure 7 where the silent period (diminished Korotkoff sound output) was not associated with any
FIGURE 5. Representative recordings showing reappearance of the K sound (stethoscope channel) and K2 associated with a G1 gap. K2 and the K sound reappear when a pressure gradient reversal (arterial pressure greater than cuff pressure) occurs. ECG, electrocardiogram; FES, wideband external pulse signal; Finapres, finger pulse recording.

phasic change in arterial pressure. In this example, the auscultatory markers show where sound initially appeared, where sound disappeared and reappeared (silent period), and where sound finally disappeared. Although there was diminished output from the stethoscope (K sound) channel, there was continued activity from the FES. A pulse distal to the blood pressure cuff could be observed when G3 occurred.

G3, unlike G1 and G2, was not characterized by the disappearance and reappearance of K2, but rather was associated with a visually present but underdeveloped K2 signal (Figure 8). The diminished sound coincided with a diminished or blunted K2 rising edge (K2-b). The blunted K2 rising edge was the most prominent feature of an underdeveloped K2.

The G3 type gap (i.e., underdeveloped K2) is not necessarily associated with the reappearance of the Korotkoff sound. On at least two occasions sound disappeared in a G3-type fashion and its reappearance was not detected. On one occasion, sound could

FIGURE 6. Representative recordings showing G2 auscultatory gap. A G2 silent period occurs with cuff pressure slightly above diastolic and a phasic increase of arterial pressure occurs. Cuff pressure during the silent period is less than arterial diastolic pressure (no pressure gradient reversal). Although cuff deflation continues, arterial pressure decreases faster to where diastolic pressure is again below cuff pressure, resulting in pressure gradient reversals and the subsequent reappearance of Korotkoff sounds. Wideband external pulse signal (FES) shows activity even during the silent period. ECG, electrocardiogram; K sound, stethoscope channel; Finapres, finger pulse recording.
not be heard at all during the entire cuff deflation. On all occasions, although sound could not be heard, the visual appearance and disappearance of an underdeveloped K2 could be observed as with the normal K2 algorithm of blood pressure determination.

It should be noted that in situations where G3 gaps were exhibited, when the cuff was initially inflated to a mid-gap pressure (i.e., where sound would be diminished or not present), the visual recognition of an underdeveloped K2 alerted the observer that an increase of the maximum cuff inflation pressure was necessary.

Discussion

Poor understanding of auscultatory gaps stems from a lack of understanding of the nature of the Korotkoff sound. Since Ettinger's classical description in 1907, the Korotkoff sounds have traditionally been divided into five phases. Although it is not disputed that the quality of sound changes during cuff deflation, this classification has never, to our knowledge, been validated objectively. Over a half century ago, Mudd and White reported three different types of gaps, but possibly because of an inadequate structure on which to base their observations, they and subsequent investigators could not always differentiate between the types. It has become the general consensus that the auscultatory gap occurs primarily during the second phase of the Korotkoff sound. The last three statements of the American Heart Association (AHA) Committee on Recommendations for Human Blood Pressure Determination have contained the following: "This early temporary disappearance of sound is called the auscultatory gap and occurs during the latter part of phase 1 and phase 2." This consensus obscures recognition of the finding that there are different types of gaps. The classification of the wideband external pulse during cuff deflation into three components (K1, K2, and K3) and the recognition that K2 is related to the audible Korotkoff sound does provide an objective visual basis on which to classify auscultatory gaps. This recognition and classification provides a structure to further explore each of the gaps without confounding the analysis.

As previously described, K2 and the Korotkoff sound are present whenever intra-arterial pressure increases sufficiently above cuff pressure such that a critical transmural pressure gradient (i.e., intra-arterial minus cuff pressure) is achieved and the compressed segment of artery opens. K2 may develop as a result of the dynamic events created by the reestablishment of fluid contact between the proximal and distal portions of the occluded artery. There is an intrinsic relation between the wideband external signal recorded at the distal portion of the blood pressure cuff and the arterial pressure pulse. Thus, the presence of a pure K1 signal indicates the arterial systolic pressure is below the cuff pressure, and the presence of a pure K3 signal indicates the arterial diastolic pressure is above the cuff pressure.

Two of the gaps, G1 (pure K1) and G2 (pure K3), are similar in that they can be explained by a phasic decrease or increase of arterial pressure, resulting in the corresponding disappearance and reappearance of K2. G1 and G2 only occur if the natural phasic change of the arterial pressure is greater than the change in cuff pressure. In G1 the arterial systolic pressure decreases faster than cuff deflation. The auscultatory gap documented by Roman et al and attributed to respiratory variation of blood pressure is an example of a G1 gap. In G2, the arterial diastolic pressure increases and then decreases back below cuff pressure. Thus, the occurrence of G1 and G2 is highly dependent on the range of phasic blood
pressure fluctuation and the rate of cuff deflation. The larger the blood pressure fluctuations and the slower the cuff deflation, the more likely is the occurrence of G1 or G2.

It thus follows that the presence of G1 and G2 gaps during standard cuff deflation rates suggests considerable beat-to-beat variability of arterial pressure, and its relatively frequent occurrence in hypertensive patients as opposed to normotensive subjects reflects a greater blood pressure variability. This increased blood pressure variability has been reported previously.17-20 G1 and G2 may also occur in certain clinical situations when expiratory airway resistance is pronounced, resulting in an increased phasic variation of blood pressure (e.g., dyspnea of congestive failure, chronic emphysema, asthma). Phasic variation in chronic emphysema and asthma may be from 20 to 30 mm Hg.21

Ironically, the recognition of G1 or G2 gaps crystallizes an ambiguity in the auscultatory cuff deflation determination of a phasically changing systolic and diastolic pressure. What is the systolic pressure when G1 is present? Should it be the cuff pressure when sound (or K2) initially appeared (possibly for only one cardiac cycle) or the reduced cuff pressure when sound (or K2) reappeared (corresponding to a phasically reduced arterial systolic pressure)? Similarly, what is the diastolic pressure when G2 is present? Should it be the cuff pressure when sound (or K2) initially disappeared or the reduced cuff pressure when sound disappeared for good (corresponding to a phasically reduced arterial diastolic pressure)? This

FIGURE 8. Panel a: Normal K2 development. Korotkoff sound coincides with fully developed K2 signal. Panel b: Underdeveloped K2. A very slight perturbation of the stethoscope channel (K sound) occurs when an underdeveloped K2 occurs. The most significant characteristic of an underdeveloped K2 is a blunted rising edge (K2-b).
may be particularly relevant for borderline hypertensive patients whose diastolic pressure varies above and below 90 (or 95) mm Hg.

G3 may represent the auscultatory gap most commonly described in the literature. It occurs predominantly with cuff pressures toward the upper portion of the pulse pressure (i.e., during the so-called phase 2 of the Korotkoff sound), and a distal pulse can be observed during its occurrence. The auscultatory gap documented by Ragan and Bordley is an example of a G3 gap. The diminished intensity of sound that characterizes G3 is associated with an underdeveloped K2, predominantly manifesting itself as a blunted K2 rising edge (K2-b).

Previously, Tavel et al were able to show that the intensity of the Korotkoff sound during cuff deflation was related to the rate of rise of the "steep wave front" after the "initial negative dip" of the intraarterial pressure just distal to the blood pressure cuff. We believe K2 is a noninvasive equivalent of the phenomenon Tavel et al described. Thus, we observed that if the rising edge of K2 is sufficiently blunted, sound intensity can diminish, often to the point of inaudibility.

The G3 phenomenon is not always associated with the reappearance of sound. We observed on two occasions that sound diminished or disappeared without reappearing while K2 was detectable, resulting in an incorrectly elevated auscultatory determination of diastolic pressure.

G3, unlike G1 and G2, cannot be explained by phasic changes of arterial pressure. Possible mechanisms might include a reduced peripheral blood flow, a decrease in the longitudinal pressure gradient between the pressure above and below the constricted artery, an anacrotic upstroke of the arterial pressure pulse, or a failure of the arterial wall to resonate on opening as a result of change of vascular tone or the physical properties of the arterial wall. A more complete understanding of K2 may contribute to information about the nature of the Korotkoff sound and the mechanisms of G3 auscultatory gaps.

Appropriate initial cuff pressure inflation is essential for the correct determination of systolic pressure when auscultatory gaps are present. In a G1 gap, the artery goes from intermittently opening and closing during each cardiac cycle to completely closing during G1 (pure K1). Thus, during a G1 gap a distal pulse would not be palpable. The possible presence of large beat-to-beat variation of systolic pressure highlights the significance of AHA recommendations for determining the maximum cuff pressure inflation (that of adding 30 mm Hg after palpating for disappearance of the radial pulse as cuff pressure is raised) to insure correct blood pressure determinations. When using wideband external pulse recording with a G3 gap present, the observer is visually alerted to improper maximum cuff pressure inflation by the recognition of an underdeveloped K2 signal and thus to the need to raise the maximum cuff pressure.

In summary, three types of auscultatory gaps have been identified. Identifying auscultatory gaps by using wideband external pulse recording provides, in addition to an accurate determination of blood pressure, insight into the mechanism that causes G1 and G2 and may provide a structure with which to better understand G3. G1 and G2 can be attributed to phasic variation of the arterial pressure, and G3 has been associated with a blunted K2 signal.

Acknowledgments

We thank the following members of Cornell University Medical College: Will Perry for his ongoing support and helpful suggestions and Dr. Gary James for his excellent comments in reviewing the manuscript. We also thank Robert Kubli of AT&T Bell Laboratories for transducer construction and electronics support.

References


**KEY WORDS** • auscultation • sphygmomanometers • blood pressure determination
Characterization of auscultatory gaps with wideband external pulse recording.
S G Blank, J E West, F B Müller, M S Pecker, J H Laragh and T G Pickering

Hypertension. 1991;17:225-233
doi: 10.1161/01.HYP.17.2.225

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1991 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://hyper.ahajournals.org/content/17/2/225