On the Production and Measurement of Cardiac Sounds in the Ear Canal

Kenneth Christofferson
University of Toronto
Toronto, Ontario, Canada

Sejal Bhalla
University of Toronto
Toronto, Ontario, Canada

Joseph Cafazzo
University Health Network, University of Toronto
Toronto, Ontario, Canada

Alex Mariakakis
University of Toronto
Toronto, Ontario, Canada

Abstract

Over the past several years, a growing body of literature has proposed systems that use earable-based acoustic sensing to assess cardiac function. These works have offered various explanations of how in-ear cardiac audio is produced. Most claim that the sounds are caused by compressive waves that travel directly from the chest, while others claim that the sounds are caused by the pulse wave producing arterial expansion near the ear canal. Although these explanations are not mutually exclusive, the lack of consensus raises questions about the working principles and possibilities in this growing research area. We present a series of experiments using a multimodal dataset of cardiac signals to test various hypotheses related to the production of heart sounds in the ear canal. Our results suggest that in-ear cardiac audio contains components produced by both compressive waves and pulse waves.

CCS Concepts

• **Human-centered computing** → **Mobile devices**; • **Applied computing** → **Consumer health**.

Keywords

Earbuds, earables, hearing aids, cardiac sensing, heart sounds, acoustic sensing

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1 Introduction

Earable devices like wireless earbuds and hearing aids have recently been proposed for various tasks including input interaction, authentication, and health monitoring [17]. A subset of this literature leverages a microphone placed in the occluded ear canal to detect cardiac sounds. These sounds have been used to estimate cardiac timing metrics [2, 7, 10, 13, 15], reconstruct other waveforms [4], and estimate pulse wave velocity (PWV) [12, 21].

The diverse uses of occluded ear canal audio for cardiac sensing rely on different signal characteristics. Heart rate tracking can be achieved using a representation of the pulse wave produced at the ear canal, whereas blood pressure estimation and heart sound reconstruction rely on information about valvular closure produced at the heart. Proposed systems also vary in the hardware used to acquire cardiac sounds from the ear canal. Microphone placement, audio filters, and earbud tip material can all influence audio quality. Furthermore, custom devices can be designed to optimally record signal characteristics in a way that does not generalize to commodity hardware. These differences in application and hardware have led to diverging viewpoints regarding the source of in-ear cardiac audio production. To the best of our knowledge, most papers claim that heart sounds travel directly from the chest to the ear as compressive sound waves, and a smaller subset claims that the pulse wave produces sound via arterial expansion near the ear canal.

This paper empirically investigates the likely sources of cardiac sounds detectable in the ear canal using data collected using a commodity earbud. Our dataset was collected from 50 healthy participants who had their cardiac state momentarily manipulated during stressor activities while data was recorded using synchronous electrocardiography (ECG), photoplethysmography (PPG), phonocardiography (PCG), and binaural earbud audio. This work contributes an analysis of the signals' arrival times according to distinct frequency bands at both a central site and the ears. We find evidence that in-ear cardiac audio is generated by both centrally-produced compressive waves and distally-produced sounds from the pulse wave. Our results suggest that low-frequency audio (<20 Hz) is primarily produced by the pulse wave, while centrally produced heart sounds are detectable in higher-frequency audio (35–50 Hz).

2 Background

In this section, we first describe proposed sources of in-ear cardiac audio. We then provide a brief overview of the literature that utilizes this audio with a focus on the sources required for each application.

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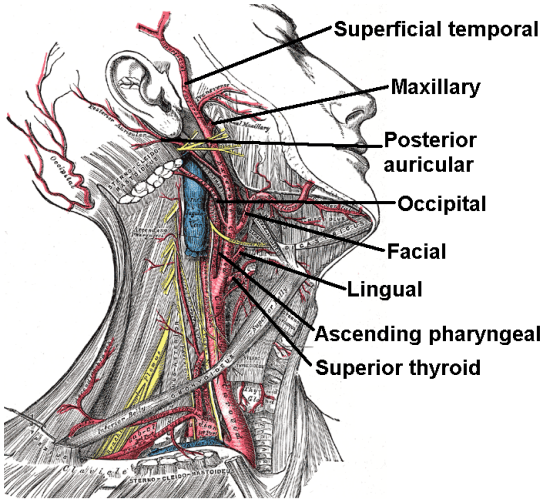


Figure 1: The superficial temporal and posterior auricular segments of the external carotid are hypothesized to deform the ear canal under pressure from the pulse wave [11].

2.1 Candidate Explanations

Prior work proposes two potential sources of in-ear cardiac audio. The first suggests that heart sounds emanate from the heart throughout the body as compressive waves. These waves could be produced by cardiac cycle events such as muscle contractions, valvular closures, and ventricular ejection [3, 16, 19]. The second proposal is related to the pulse wave, which is the pressure wave produced by rapid blood ejection from the heart. The pulse wave causes arteries to expand as it traverses the body, compressing nearby tissues. The ear canal is surrounded by small vessels and the large external carotid artery as shown in Figure 1. When the pulse wave is proximate to the ear canal, tissue compression subtly deforms it in a way that reportedly produces detectable sounds related to cardiac activity.

2.2 Overview of Related Work

In-ear cardiac audio has been used to estimate a variety of metrics such as heart rate [2, 8, 10, 13, 15], blood pressure [21, 22], and PWV [12]. This audio has also been used to reconstruct other cardiac signals like PCG [4] with the ultimate goal of enabling diagnosis. Table 1 provides an abridged review of papers in this space and a categorization of the claimed working principle underlying them. These works generally require at least one of the following dimensions of cardiac activity:

- Pulse arrival timing at a distal site,
- Pulse origination timing,
- Relative timing of cardiac cycle events (e.g., delay between atrioventricular and semilunar valve closure),
- Heart sound characteristics (e.g., pitch of atrioventricular valve closure, murmurs).

Neither the pulse wave nor compressive wave transduction alone contains the information required for all the systems listed in Table 1. Calculating heart rate only requires knowing pulse arrival timing at a distal site, while calculating PWV requires knowing the delay between pulse production at the heart and arrival at a distal site. Heart sound reconstruction is more arduous as it requires preserving abnormal heart sounds and the relative timing between S1 and S2 heart sounds. Therefore, our experiments aim to support both hypothesized sources in order to justify prior work and motivate future explorations.

Table 1: An abridged summary of prior work on earable cardiac sensing according to their target outcomes and hypothesized working principle.

	Metric/Signal Predicted	Pulse Wave	Heart Sounds
Nirjon et al. [15]	HR	✗	✓
Fan et al. [8]	HR/HRV	✓	✗
Fan et al. [7]	HR	✓	✗
Martin et al. [13]	HR	✗	✓
Gilliam III et al. [10]	HR/HRV/IBI	✓	✗
Butkow et al. [2]	HR	✗	✓
Zhao et al. [22]	BP/PWV	✗	✓
Truong et al. [21]	BP	✗	✓
Kusche et al. [12]	PWV	✓	✓
Chen et al. [4]	PCG	✗	✓

HR: heart rate, HRV: heart rate variability, IBI: inter-beat interval, BP: blood pressure, PWV: pulse wave velocity, PCG: phonocardiography

2.3 Signal Speeds

Our experiments rely on the documented speeds of compressive waves and the pulse wave. Compressive waves travel at speeds between 1,430 and 1,670 m/s in human tissue [1]. To the best of our knowledge, PWV between the ascending aorta and ear canal has not been directly reported. The closest approximation we could find reports PWV in the common carotid artery to be between 3 and 5.5 m/s [18, 20]. This is slower than the commonly reported PWV measured between the carotid and femoral arteries, which lies between 3.1 and 13.4 m/s [6].

We use these ranges to inform our experiments, but it is important to acknowledge the uncertainty in them. Compressive wave velocity can vary depending on factors like body composition and structure [1]. Meanwhile, PWV varies along the arterial tree because of changes in arterial cross-sectional area and stiffness [18]. PWV is also typically measured while people are supine [6], meaning that different postures can add gravitational forces that speed up or slow down the pulse wave [14]. All of these factors can vary across and within individuals.

3 Dataset

We probe the presence of both pulse wave and compressive wave information by comparing characteristics of in-ear cardiac audio across multiple frequency bands against reference cardiac signals. It is important to note that because these reference signals rely on different phenomena, they are not expected to have identical morphology; we account for these discrepancies throughout our experimental design.

3.1 Data Collection

We collected data from 50 healthy adult participants. This cohort included 22 males and 28 females with an average age 29.2 ± 17.5. Participants were invited to participate in a 30-minute session during which they were instrumented with five physiological sensors: ECG at the chest, PPG at the left earlobe, PCG from a digital stethoscope at the chest, and binaural occluded ear canal audio. ECG reflects electrical activity at the heart, making it a reliable indicator of the beginning of a cardiac cycle. PPG was used to reflect pulse wave information near the ear because it detects local changes in blood volume due to arterial dilation. Lastly, the digital stethoscope PCG was used to reflect compressive wave information at the chest according to acoustic energy.

All of the signals were captured synchronously at a sampling rate of 10 kHz using amplifiers, ADCs, and data acquisition hardware made by BIOPAC Systems[1]. The occluded ear canal audio was recorded using a pair of Sony WF-1000XM3 active noise-cancelling earbuds[2]. The earbuds' active electronics were removed and the inner microphone was physically wired out to access raw audio, but otherwise, the earbuds were unmodified. A foam earbud tip was used in all experiments.

Data was recorded throughout the 30-minute session. During that time, each participant completed multiple cardio-respiratory stressor activities — breath holding, the Valsalva maneuver, the cold pressor test, and light exercise — to induce diverse changes in hemodynamic function [9]. The order of these stressors was counterbalanced, and participants took a 3-minute break after each one to ensure their cardiac activity returned to a relative baseline.

3.2 Signal Preprocessing and Feature Extraction

We applied various digital filters to all of the raw physiological signals except for the digital stethoscope PCG. The filters were zero-phase, 5^{th}-order Butterworth filters with different cutoff frequencies. Earlobe PPG data was processed using a low-pass filter with a cutoff of 20 Hz to reduce noise, and ECG data was processed using a high-pass filter with a cutoff of 15 Hz to facilitate R-peak detection. Compressive waves produced by the heart contain higher frequencies and attenuate more dramatically relative to the pulse wave. Therefore, we implemented two filters on the occluded ear canal audio to separate pulse wave information from compressive wave information. Pulse wave information was isolated using the same filter as the one used for earlobe PPG data: a low-pass filter with a 20 Hz cutoff. Compressive wave information was isolated using a bandpass filter with cutoffs of 35 Hz and 50 Hz. The lower cutoff was designed to get sufficient spectral separation from the pulse wave, while the higher cutoff was designed to suppress ambient noise.

To identify relevant cardiac events in these signals, we first identified ECG R-peaks using standard peak-finding methods. We used these timestamps as references for the following events across other modalities, assuming that each event occurred between consecutive R-peaks:

- **S1 heart sound (digital stethoscope):** We assumed the first large peak in the PCG following an ECG R-peak to be the S1 heart sound.
- **Pulse arrival (PPG):** We used the foot of the PPG signal to indicate pulse arrival because it represents the beginning of arterial dilation. To identify the foot, we first located the first large in the PPG after an ECG R-peak and then looked back in the PPG to find the previous trough.
- **Pulse arrival (earbud audio <20 Hz):** The typical structure of in-ear cardiac audio for a signal pulse contains two peaks with a large trough in between. We used the first peak in this structure to indicate pulse arrival.

Because this work investigates the source of in-ear cardiac audio and not the consistency of those sounds, we limited our experiments to cycles that had sufficiently clear signal quality according to sensor placement and cardiac event extraction. To filter out instances of poor sensor placement in any of the signals, we removed segments when the signal's amplitude envelope deviated beyond a sensor-specific threshold within the period between two consecutive R-peaks. We conducted three tests to check the validity of cardiac event extraction:

- In the PPG signal, we identified the largest value during the first 50% of each cycle duration and tested whether it was greater than the value at 80% of the cycle duration. This test was designed to confirm that the systolic peak was larger than the diastolic component of the cycle.
- In the occluded ear canal audio, we removed cycles where the low-frequency sound peak was detected before S1 sound production since that is impossible.
- In the occluded ear canal audio, we removed cycles where the detected left and right in-ear peaks were more than 50 ms apart, as this would imply a difference in arterial path far exceeding what is feasible according to literature [5] (see Section 4.2 for this calculation).

4 Comparative Pulse Wave Arrival Timing

In this section, we examine the timing of low-frequency in-ear cardiac audio to confirm that the pulse wave produces a component of in-ear cardiac audio.

4.1 Delay Between S1 Heart Sound Production and Pulse Arrival

First, we examine the delay between S1 heart sound production and pulse arrival measured by the earlobe PPG to support the validity of our experimental approach and PWV estimates. Figure 2 shows the timing of these two events. The average delay between them is 117.9 ± 28.6 ms across our dataset. Assuming that the average PWV is 3.5 m/s, the midpoint reported by Sorensen et al. [18], this delay maps to a distance of 41.3 ± 10.0 cm, which reasonably represents the arterial path length between the heart and the earlobe and supports our experimental approach.

Figure 3 shows the timing between S1 heart sound production at the chest and pulse arrival according to occluded ear canal audio; note that the left earbud is used since the earlobe PPG was placed on that side of the head. The average delay between the two events is 94.7 ± 28.4 ms. Using a similar calculation as before, this

[1]https://www.biopac.com/
[2]https://www.sony.com/electronics/truly-wireless/wf-1000xm3

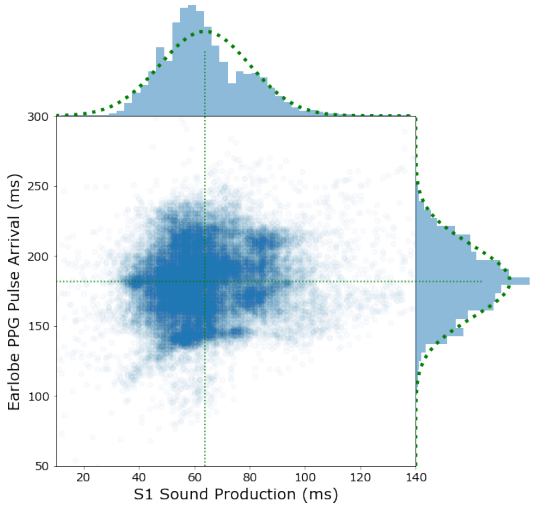


Figure 2: A scatter plot showing the timing relationship between S1 heart sound production and pulse arrival measured by the earlobe PPG.

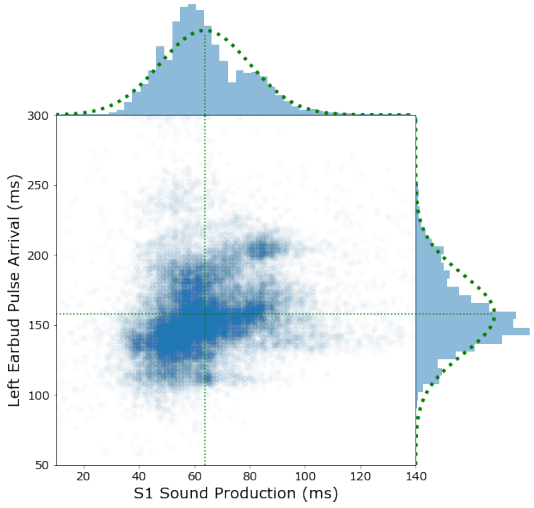


Figure 3: A scatter plot showing the timing relationship between S1 heart sound production and pulse arrival measured using low-frequency in-ear cardiac audio recorded at the left ear.

delay implies a distance of 33.1 ± 9.9 cm. The external carotid begins to deform the ear canal before it pushes blood to capillaries in the earlobe, so the fact that this delay is shorter aligns with expectations. The difference between the two average delay distributions is 23.2 ms, which translates to 8.1 cm. This distance is a bit longer than the perceivable distance between the carotid and the earlobe, but given the range of potential speeds and the uncertainty in that range, this distance is within a reasonable order of magnitude to support the argument that some content from in-ear cardiac audio relates to the pulse wave.

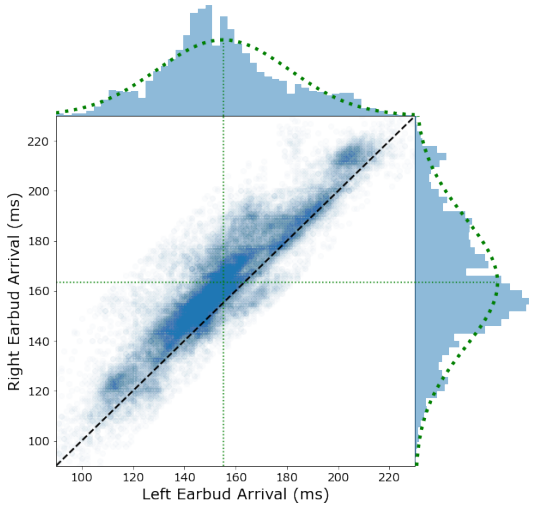


Figure 4: The distribution of pulse arrival times at the left and right ears measured using low-frequency in-ear cardiac audio.

4.2 Difference in Pulse Arrival Across Ears

The arterial path from the heart is slightly longer to the right ear than it is to the left. Therefore, all cardiac information related to a given beat should arrive at the left earbud first, and the delay between these two sides should still be consistent with PWV.

Figure 4 shows the distribution of paired cardiac sound arrival times across the left and right ears. The black diagonal line delineates when the two arrival times were equal, so points above the line indicate cycles when sound was detected in the left ear first, while points below the line indicate the opposite. Roughly 97% of the points lie above the line, supporting the notion that the arterial path to the right side is longer; the points below the line are likely the result of confounding sounds or peak detection error. The average delay between the R-peak and the arrival time at the left ear is 157.9 ± 28.7 ms, while the corresponding average delay for the right side is 163.5 ± 26.5 ms.

For the average adult, the distance in arterial path length between the left and right sides is roughly 1.4 cm [5], which would result to a time delay of 4.0 ms at a PWV of 3.5 m/s. We observe that the average delay between the two sides was 5.6 ms in our dataset. Although it is slightly slower than expected, these delays are similar enough to support the argument that the delay can be at least partly attributed to the pulse wave.

5 In-ear Cardiac Sound Characteristics

Performing the above calculations with compressive waves is challenging given the attenuation they experience during transduction. We observed that even slight movement artifacts and environmental sounds significantly overwhelm high-frequency occluded ear canal audio. Therefore, this section relies on visualizations to confirm that compressive waves are partly responsible for in-ear cardiac audio.

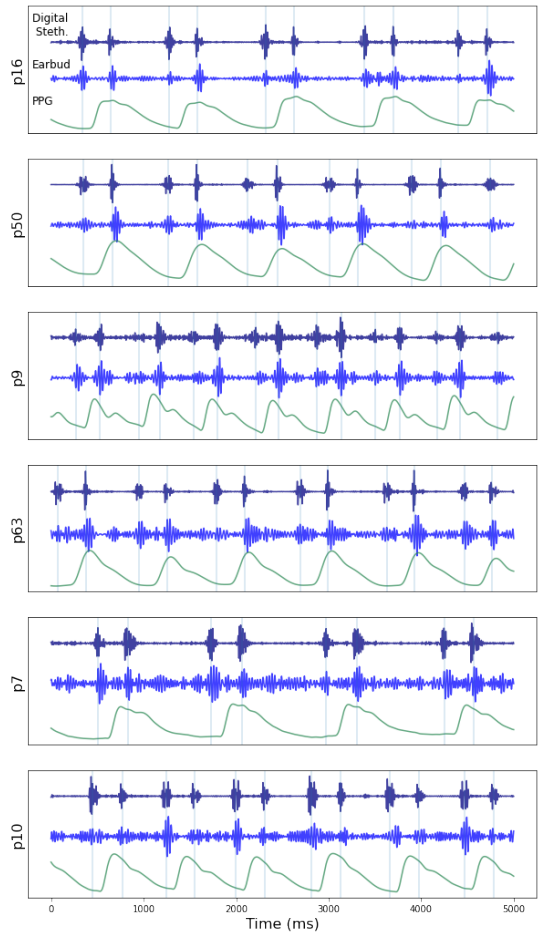
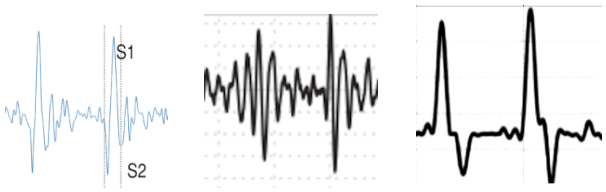


Figure 5: Example waveforms showing digital stethoscope PCG, high-frequency in-ear cardiac audio, and earlobe PPG from multiple participants.

5.1 Signal Similarity

Digital stethoscope PCG typically exhibits a pattern with clear S1 and S2 heart sounds. While PPG signals also include features with similar timing to these events, their morphology is significantly different with an added lag on the order of 100 ms according to PWV. Conversely, compressive waves travel at a quick enough speed to arrive at the ear canal within 1 ms. Therefore, we visually compare the morphology of digital stethoscope PCG, earlobe PPG, and high-frequency in-ear cardiac audio to assess whether any heart sounds can be detected before pulse wave arrival.

Figure 5 compares 5-second segments of these signals selected from six participants. They show that high-frequency in-ear cardiac audio contains evident S1 and S2 heart sounds that are more aligned with the digital stethoscope PCG compared to the earlobe PPG. There are occasional delays between heart sound arrival according to PCG and in-ear cardiac audio, particularly when the signal-to-noise ratio is lower. While we can not speak confidently about the source of this delay, we speculate that these outliers are due to either measurement error, acoustic attenuation that weakens heart sounds below the noise floor, or spurious sounds.



(a) Butkow et al. [2] (b) Kusche et al. [12] (c) Gilliam III et al. [10]

Figure 6: Images provided by other authors of acoustic cardiac sensing work with earables to illustrate the occluded ear canal audio recorded by their hardware.

6 Discussion and Future Work

After summarizing our key findings, we briefly comment on the importance of hardware and software design choices when it comes to in-ear cardiac audio morphology. We then discuss the limitations of our work and opportunities for future exploration.

6.1 Key Findings

Our experiments provide evidence that both the pulse wave and compressive waves factor into in-ear cardiac audio. We first showed that the delay between S1 heart sound production and low-frequency audio aligns with expectations of PWV. We also observed a delay in pulse wave arrival between the left and right ears, further supporting the hypothesis that the pulse wave contributes to low-frequency audio in the ear canal. Conversely, we showed that there are morphological similarities between high-frequency audio in the ear canal and a synchronously recorded digital stethoscope that happen well before pulse wave arrival. These results suggest that compressive waves are a major component of high-frequency in-ear cardiac audio.

6.2 In-ear Cardiac Audio Heterogeneity

There are considerable differences in the morphology of in-ear cardiac audio reported by the community. Figure 6 illustrates the signals documented by some authors. Figure 6a shows the pattern we observed most in our own work and in the literature, with each cycle being characterized by a large peak surrounded by a series of smaller peaks. Figure 6b shows a different cycle pattern with two bursts representing the S1 and S2 heart sounds respectively. The signal in Figure 6c was the least commonly observed in our literature review, as it appears to have a higher signal-to-noise ratio.

Since most systems in this space are evaluated on healthy adult participants, this heterogeneity is more likely due to differences in hardware, ear canal sealing, and signal processing. These choices are important because they impact the dimensions of cardiac activity that can be extracted from occluded ear canal audio. For example, the signal in Figure 6c is particularly well-suited for heart rate estimation, but it lacks the higher frequency content needed to fully represent S1 and S2 heart sounds. The signal in Figure 6b is the only one that shows clear evidence of valvular closure, but it does not mean that closure cannot be identified in the others;

rather, the clarity of valvular closure is more likely to be related to the degree to which pulse wave sounds dominate the audio. In summary, researchers should be aware of these discrepancies as they design their systems and make claims about generalizability across different platforms.

6.3 Limitations

This study is limited in several ways. We collected data using only one set of commodity earbuds. While this allowed us to standardize data collection across participants, it limits our ability to comment on how hardware design factors like microphone placement and frequency response impact in-ear cardiac audio quality. Some previous studies have reported clearer S1 and S2 heart sounds from the occluded ear canal [12], but we suspect this may be due to differences in hardware or protocol. Future work could replicate our experiments with better hardware designs and software algorithms for heart sound detection. Our experiments also did not involve people diagnosed with cardiac illnesses because the cardio-respiratory stressor activities in our protocol would have put them at greater risk. One of our future goals is to recruit such individuals with additional precautions in place, but until then, we cannot confirm that abnormal heart sounds like murmurs can be transduced at the ear canal.

7 Conclusion

In this paper, we compared characteristics of occluded ear canal audio to reference cardiac signals in order to assess the validity of different explanations for in-ear cardiac audio. We first showed that low-frequency audio (<20 Hz) can be largely attributed to the pulse wave via arterial expansion near the ear canal, and then we showed that high-frequency audio (35–50 Hz) appears to be caused by compressive sound waves that emanate from the chest. Although computational models informed by biomechanics would further support these possible explanations of in-ear cardiac audio production, we believe that our work provides sufficient objective evidence to support the working principles underlying most works on earable-based systems for cardiac sensing.

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