Impedance cardiography: What is the source of the signal?

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Abstract. Impedance cardiography continues to be investigated for various applications. Instruments for its use are available commercially. Almost all of the recent presentations and articles along with commercial advertisements have assumed that aortic volume pulsation is the source of the signal. A review of the literature will reveal that there is no clear evidence for this assumption. Starting with the first paper on impedance cardiography in 1964, which assumed the lung was the source of the signal, the presentation will review many studies in the 60's, 70's and 80's, which suggest the aorta and other vessels as well as atria and again the lung as possible sources. Current studies based on high resolution thoracic models will be presented that show the aorta as contributing only approximately 1% of the total impedance measurement, making it an unlikely candidate for the major contributor to the signal. Combining the results of past studies along with recent work based on models, suggest other vessels and regions as possible sources.

1. Introduction

Impedance cardiography continues to be used in research and for clinical applications for stroke volume and cardiac output measurements. Almost all of the recent presentations and articles along with commercial advertisements have assumed that aortic volume pulsation is the source of the signal. This paper will review past studies that attempted to determine the physiological origin of the signal and present new modelling data that suggest alternate sources for the signal. The assumption that the aorta is the most significant source of the signal is on very weak grounds.

2. Background and past studies

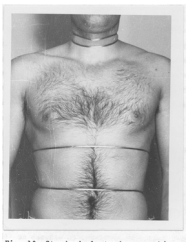

Figure 1. Original electrode band arrangement

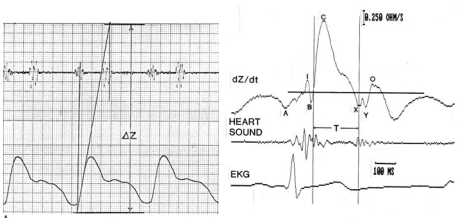

Figure 2. Original ΔZ slope extraction process and the current dZ/dt. Decreasing impedance upward.

In order to develop an electrode configuration that was consistent and repeatable over a wide range of individuals and somewhat independent of the amount of air in the lungs, Patterson et al [1,2] devised an arrangement of band electrodes at the ends of the thorax. More recently this configuration has used spot electrodes in the previous band electrode locations. The waveform from such an arrangement showed decreasing impedance during systole. Based on a volume event this would suggest that the

region causing the impedance change was increasing in volume during systole. This would eliminate the ventricles as a source of the signal. At the time of the original development it was assumed that the lungs would be the most likely candidate for the source. The right ventricle delivers the stroke volume to the lungs during systole, but at the same time the pulmonary veins drain blood from the lungs. An empirical forward graphical slope procedure was developed to account for the pulmonary drainage during systole. This resulted in an equation that gives reasonable stroke volume values [1,2]. The graphical slope procedure was later replaced by an equivalent method using the first derivative of the waveform [2].

2.1. Animal studies

In 1965, NASA funded for 5 years, a research group at the University of Minnesota to develop the impedance method to measure cardiac output on the astronauts for the Apollo moon mission. As part of this contract a large series of animal experiments were undertaken to determine the source of the waveform in order to develop a sound formula for cardiac stroke volume. Dogs were studied with pressure catheters placed in all the major vessels and heart chambers. Electromagnetic flow transducers were placed on the pulmonary artery and aorta. Many different manoeuvres were used to change stroke volume. At the start of the studies it was assumed that the lungs were the source of the signals. By accident one day one of the animals developed complete left mechanical alternates. Tracings from the flow probe and impedance only showed a change when the left heart ejected blood. This is shown in Figure 3. In many dogs with complete left mechanical alternans there was no change in delta Z with only the right ventricle ejecting, but in some dogs there was a change of about 50% compared to both ventricles ejecting as shown on the right frame of Fig. 3. This suggested that the signal came from the arterial system. One region considered for the source was the aorta, which was reported [3,4].

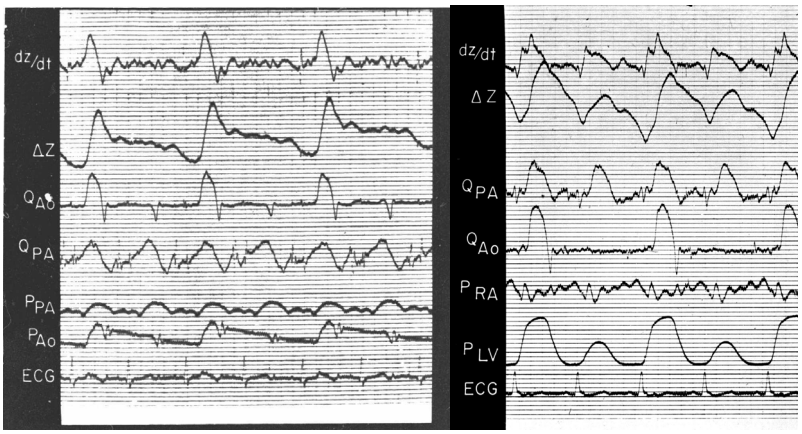

Figure 3. The left graph shows no change in ΔZ without left ventricular ejection whereas in the right graph there is a small change. Q_{Ao} is aortic flow and Q_{PA} is pulmonary artery flow. P_{PA} pulmonary artery pressure and P_{Ao} is aortic pressure.

2.2 Human Studies

Another approach using field mapping was undertaken to understand better the source of the cardiogenic impedance signal. The amplitude of dZ/dt was measured as a function of distance up the thorax. In Figure 4 on the left side is shown the position of strip electrodes on the chest and on the right is shown the increase in amplitude of dZ/dt with distance increasing from the xiphisternal joint up to the neck. The results show the lower part of the thorax contributes very little to the total signal. In the region from the supra-sternal notch down the sternum 10 cm gives a signal amplitude that accounts for about 75% of the total for the full bands.

3. Current model based studies

The new studies involve use our finite difference (FD) electrical model of the human thorax [6]. This model has 3.8 million control volumes or elements. The resolution is 1.5 mm in the x-y direction with 5 mm thick slices. The software used with the model can determine current and voltage distributions from external excitation along with sensitivity analysis, which can determine the contributions of various organs and regions to the total impedance.

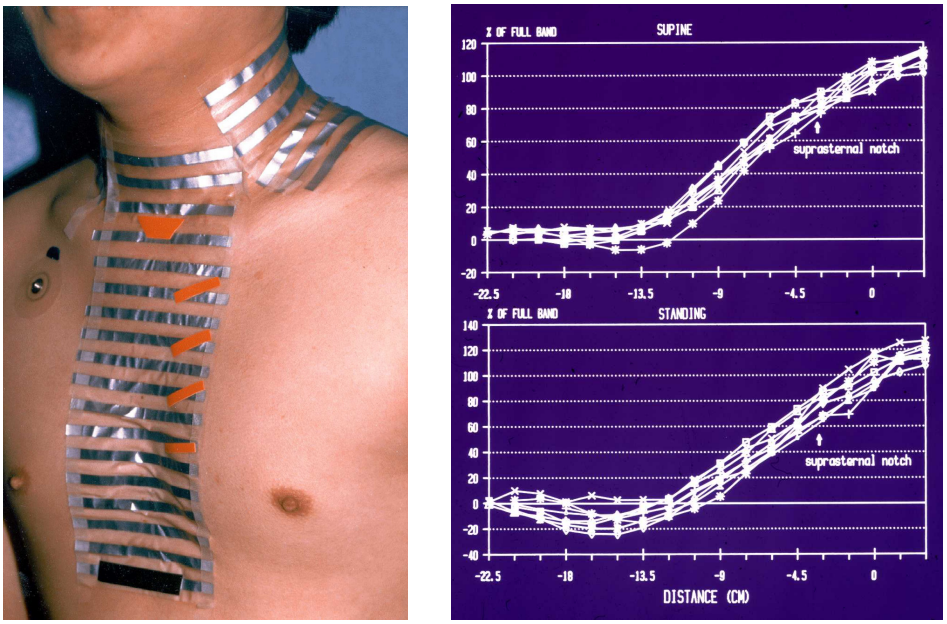

Figure 4. The electrode array for the voltage pick up electrodes is shown on the left. The reference electrode is at the bottom in black. The graph shows the change in the dZ/dt amplitude expressed as a percentage of the full band signal as a function of distance up the thorax from the xiphisternal.

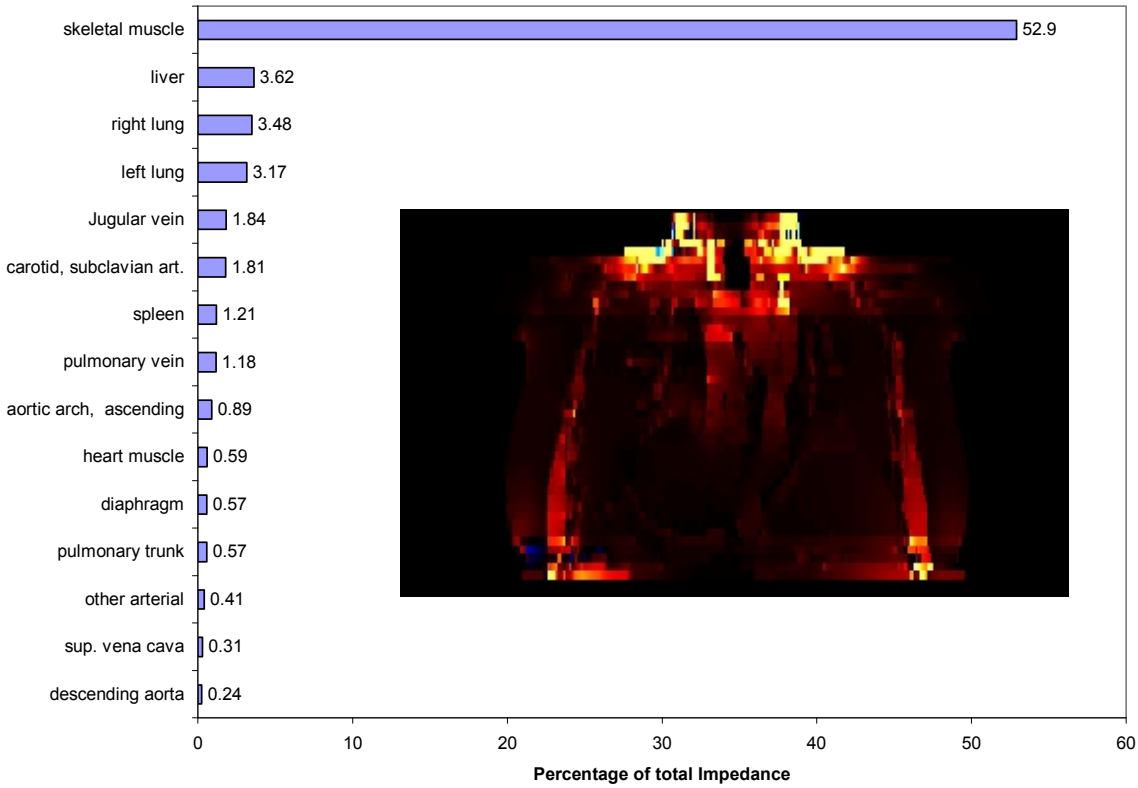

Figure 5. This gives the percentage contribution of various organs and regions to the total impedance as determined with the model along with an insert of showing regions with high sensitivity to local resistivity changes.

4. Discussion and Conclusion

The results of the modelling study agree very closely with Kaupinen et al. [7]. Both show the large contribution of the skeletal muscle to total impedance and a small contribution by the aorta, along with high sensitivity of impedance change in the upper thorax. With the aorta's contribution to total impedance of less than one percent, during systole the aortic volume would have to change more than 60%, which is unreasonable. Also the other arterial and venous vessels in the upper thorax contribute twice as much as the aorta to the total impedance. The fact that the jugular vein contribution is similar to the upper thoracic arteries may explain the distortion in the waveform in patients with conditions like heart failure who frequently have a large pulse in jugular vein.

It is highly unlikely that the aorta is a major contributor to the impedance cardiography waveform and also the venous pulsations from the neck region may cause significant distortions in the waveform leading to errors in the stroke volume determinations.

References

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