Proceedings of the

UNIVERSITY OF OTAGO MEDICAL SCHOOL

VOLUME 62 NUMBER 2 JULY 1984

The One Hundred and First Meeting of the Otago Medical School Research Society, 26 July 1984.

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Address for all authors:
University of Otago Medical School,
P.O. Box 913, Dunedin, New Zealand.

INTRA-THORACIC IMPEDANCE AND AORTIC BLOOD FLOW

A. B. Baker, A. J. Roxburgh,
Department of Anaesthesia
and C. McLeod,
Department of Paediatrics, University of Oxford

Mitchell and Newbower[1] have produced a theoretical model which shows that any change in the intra-thoracic impedance is unlikely to be correlated with stroke volume, due to the inability to distinguish aortic movement from blood flow. Their model, whilst having some anatomical validity, did not take account of the increase in the electrical conductivity of blood which occurs when blood flows.[2-6] Because the fractional change in conductivity can be as high as 25% for peak flows, there is a large error in their results. In particular, the changes in aortic blood velocity would make as great a contribution to the changes in intra-thoracic impedance as movement of the aorta. The aim of this study has been to refine this model, and generate an equation that defines the relationship between blood velocity and

When $A = 0.5$ cm, $B = 12$ cm, $C = 11$ cm, $R = 1.25$ cm, $\sigma_1 = 1/600$ $(\text{ohm-cm})^{-1}$, σ_2 $(v = 0) = 1/160$ $(\text{ohm-cm})^{-1}$, $a = -0.34$, $b = 0.15$:

If A reduced by 0.1cm: $\triangle Z = -5.1\Omega$
If R increased by 0.1 cm: $\triangle Z = -0.3\Omega$
If σ_2 increased by 25% $\triangle Z = -5.0\Omega$

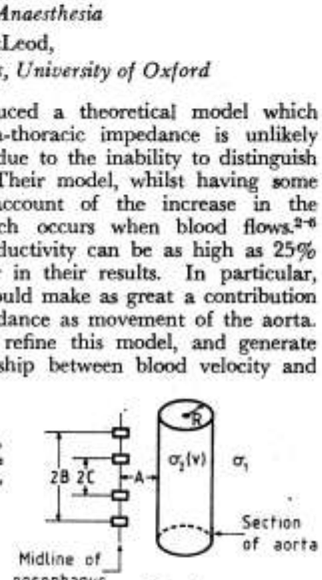

Fig. A

Fig. A. The simplified anatomical model used, adapted from Mitchell and Newbower.[1] Refer to the text for further explanation.

$$Z = \frac{1}{2\pi\sigma_1}\left[\frac{2C}{B^2-C^2} + \left(\frac{\sigma_2-\sigma_1}{\sigma_2+\sigma_1}\right)\left(\frac{1}{\sqrt{(2A+A^2/R)^2+(B+C)^2}} - \frac{1}{\sqrt{(2A+A^2/R)^2+(B-C)^2}}\right)\right] \quad (1)$$

$$\frac{\Delta\sigma_2}{\sigma_2} = -a(1-e^{-b\sqrt{v}}) \quad (2)$$

$$\Delta Z = \frac{-\sigma_2 a(1-e^{-b\sqrt{v}})}{\pi(\sigma_1+\sigma_2)^2}\left(\frac{1}{\sqrt{(2A+A^2/R)^2+(B+C)^2}} - \frac{1}{\sqrt{(2A+A^2/R)^2+(B-C)^2}}\right) \quad (3)$$

Fig. B

Fig. B. Equations relating Z and $\triangle Z$ to σ_1, σ_2, v, A, B, C and R. (1) is from Mitchell and Newbower.[1] (2) is adapted from Visser.[3] For a haematocrit of 45%, $a \doteq -0.34$, $b \doteq 0.15$ $(\text{cm/s})^{-1/2}$. (3) is obtained from (2) and the derivative of (1) with respect to σ_2. Note that in (2) and (3), σ_2 refers to the electrical conductivity of blood during the diastolic phase (no flow) only.

the pulsatile component of the intra-thoracic impedance. From blood velocity, stroke volume may be derived.

Methods. In the simplified anatomical model (Fig. A), the aorta is represented as an infinite cylinder of conductivity σ_2 (v) and radius R passing through the thorax represented as an infinite homogeneous medium with electrical conductivity of σ_1. The flow velocity of blood in the aorta is v. A four-electrode oesophageal probe is aligned parallel to the lumen of the aorta at a distance A from it. B and C are respectively the half-spacings of the current source and voltage sensing electrodes of the probe. The new factor that we have added to this model is the velocity dependence of the electrical conductivity of blood. The equations linking change in oesophageal impedance ($\triangle Z$) with blood velocity are shown together with a brief resume of their derivation (Fig. B). Values of $\triangle Z$ have been generated for different conditions of the model.

Results and discussion. Results using different assumptions (Table) demonstrate that there is a likelihood of obtaining a $\triangle Z$ of the order of 5 ohm due to the aortic blood flow during systole. This is similar to previous measured changes,[6, 7] and also similar to the impedance change which might be generated by aortic movement ($\triangle A$). The rapid negative impedance change at the start of systole is probably due only to the flow of blood in the aorta.[5] Thus stroke volume should be obtainable if aortic diameter is known.

Although the model used here is not directly applicable to the trans-thoracic impedance method, the strong inverse dependence of $\triangle Z$ on A (Fig. B, 3) suggests that the trans-thoracic aortic $\triangle Z$ signal will be severely attenuated, which is indeed the case.[7]

This research was supported by the Medical Research Council of New Zealand.

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