Stroke volume equation for impedance cardiography

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Abstract—*The study's goal was to determine if cardiac output (CO), obtained by impedance cardiography (ICG), would be improved by a new equation N, implementing a square root transformation for $dZ/dt_{max}/Z_0$, and a variable magnitude, mass-based volume conductor V_C. Pulmonary artery catheterisation was performed on 106 cardiac surgery patients pre-operatively. Post-operatively, thermodilution cardiac output (TDCO) was simultaneously compared with ICG CO. $dZ/dt_{max}/Z_0$ and Z_0 were obtained from a proprietary bioimpedance device. The impedance variables, in addition to left ventricular ejection time T_{LVE} and patient height and weight, were input using four stroke volume (SV) equations: Kubicek (K), Sramek (S), Sramek–Bernstein (SB), and a new equation N. CO was calculated as SV $\times$ heart rate. Data are presented as mean $\pm$ SD. One way repeated measures of ANOVA followed by the Tukey test were used for inter-group comparisons. Bland–Altman methods were used to assess bias, precision and limits of agreement. $P < 0.05$ was considered statistically significant. CO implementing N (6.06 ± 1.48 l min^{-1}) was not different from TDCO (5.97 ± 1.41 l min^{-1}). By contrast, CO calculated using K (3.70 ± 1.53 l min^{-1}), S (4.16 ± 1.83 l min^{-1}) and SB (4.37 ± 1.82 l min^{-1}) was significantly less than TDCO. Bland–Altman analysis showed poor agreement between TDCO and K, S and SB, but not between TDCO and N. Compared with TDCO, equation N, using a square-root transformation for $dZ/dt_{max}/Z_0$, and a mass-based V_C, was superior to existing transthoracic impedance techniques for SV and CO determination.*

Keywords—*Stroke volume, Cardiac output, Impedance cardiography, Acceleration, dZ/dt_{max}*

Med. Biol. Eng. Comput., 2005, **43**, 443–450

1 Introduction

TRANSTHORACIC ELECTRICAL bioimpedance cardiography, or, simply, impedance cardiography (ICG), is a non-invasive, continuous, beat-to-beat method for estimating left ventricular stroke volume (SV) and cardiac output (CO) (NEWMAN and CALLISTER, 1999; OVSYSHCHER and FURMAN, 1993; WOLTJER *et al.*, 1997).

Because of its non-invasive nature, ICG has been proposed as an alternative to thermodilution CO (TDCO) (SHOEMAKER *et al.*, 2001). Unfortunately, however, poor agreement with TDCO in the critically ill, and especially those with cardiopulmonary pathology, has generally precluded its application in clinical decision making (RAAIJMAKERS *et al.*, 1999; 1998; CRITCHLEY *et al.*, 2000; YOUNG and MCQUILLAN, 1993; GENONI *et al.*, 1998).

A review of the literature indicates that the theoretical basis for existing ICG SV equations and their operational implementation depend upon the most basic laws of electricity, namely, Ohm's law and its corollaries (PENNEY, 1986; OVSYSHCHER and FURMAN, 1993). These equations, in turn, are simply modifications of an equation originally intended for extremity blood volume changes (NYBOER, 1950). Rather predictably, they inadequately articulate the much more complex and dynamic, vascular and haemorheologic inter-relationships of pulsatile thoracic blood flow (SAKAMOTO and KANAI, 1979; KOSICKI *et al.*, 1986; VISSER *et al.*, 1990). Thus a coherent, biophysically meaningful and theoretically robust mathematical expression, linking impedance-derived SV and CO to other standard methods has been suggested as being necessary (RAAIJMAKERS *et al.*, 1999). Accordingly, this study takes issue with some of the theoretical assumptions of existing methods; namely that

(*a*) the magnitude of SV is directly related to power functions of thoracic length, or height-based thoracic length equivalents (BERNSTEIN, 1986)
(*b*) all pulsatile impedance changes $\Delta Z(t)$ (Ω) and rates of change of impedance dZ/dt ($\Omega\,s^{-1}$) are due to vessel volume changes $\Delta V(t)$ (ml) and rates of change of volume dV/dt (ml s^{-1}), respectively (KUBICEK *et al.*, 1966; 1974)
(*c*) the specific resistance (i.e. 'resistivity') of blood ρ_b (Ωcm) is constant during ventricular ejection (KUBICEK *et al.*, 1974).

Thus the goal of this study was to determine if a new SV equation, using a square-root transformation of $dZ/dt_{max}/Z_0$, and a variable-magnitude, mass-based volume conductor

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Paper received 14 October 2004 and in final form 2 March 2005
MBEC online number: 20054015

(V_C), provides better CO results than existing SV methods. The new equation N is compared with the Kubicek (K), Sramek (S) and Sramek-Bernstein (SB) equations (KUBICEK *et al.*, 1966; 1974; BERNSTEIN, 1986), using TDCO as a reference standard.

2 Background and methods

2.1 *ICG SV equations implemented by the transthoracic approach and tetra-polar electrode configuration*

2.1.1 *Kubicek equation (K)*

$$SV_K = \frac{\rho L^2}{Z_0^2}\frac{dZ(t)}{dt_{\max}}T_{LVE} \quad (1)$$

2.1.2 *Sramek equation (S)*

$$SV_S = \frac{L^3}{4.25}\frac{dZ(t)/dt_{\max}}{Z_0}T_{LVE} \quad (2)$$

2.1.3 *Sramek–Bernstein equation (SB)*

$$SV_{SB} = \delta\frac{L^3}{4.25}\frac{dZ(t)/dt_{\max}}{Z_0}T_{LVE} \quad (3)$$

where $SV_{(K,\ S,\ SB)}$ = ml; $V_{C(K)} = \rho L^2/Z_0$; $V_{C(S)} = L^3/4.25$; $V_{C(SB)} = \delta L^3/4.25$ = ml (BERNSTEIN, 1986); ρ_b = static specific resistance of blood (Ωcm) = 135 Ωcm for $SV_{(K)}$ (QUAIL *et al.*, 1981); $\delta = \sqrt{(BMI_P/24\ \mathrm{kg\ m^{-2}})}$, where BMI_P = patient body mass index (kg m^{-2}), 24 kg m^{-2} = ideal BMI for $SV_{(SB)}$; L = the measured thoracic length between voltage sensing electrodes (cm), approximated as 17% of measured patient height ($0.17\times H$)(cm) for equations K, S and SB (BERNSTEIN, 1986; VAN DER MEER *et al.*, 1996; VAN DE WATER *et al.*, 2003); Z_0 = the measured transthoracic quasi-static base impedance (Ω); $dZ/dt_{\max}$ = the peak first time-derivative of the transthoracic cardiogenic impedance pulse variation ($\Omega\,\mathrm{s}^{-1}$) for equations K, S and SB; T_{LVE} = left ventricular ejection time (s).

2.1.4 *New ICG SV equation N (Bernstein)*

$$SV_N = \frac{V_{ITBV}}{\zeta^2}\sqrt{\frac{dZ(t)/dt_{\max}}{Z_0}}T_{LVE} \quad (4)$$

where SV_N = ml; $V_C = V_{ITBV}/\zeta^2$ = ml; $V_{ITBV} = 16W^{1.02}$ = intra-thoracic blood volume = ml; W = body weight (kg).

$$\zeta = \frac{Z_C^2 - Z_C Z_0 + K}{2Z_C^2 + Z_0^2 - 3Z_C Z_0 + K}(\text{dimensionless}) \quad (5)$$

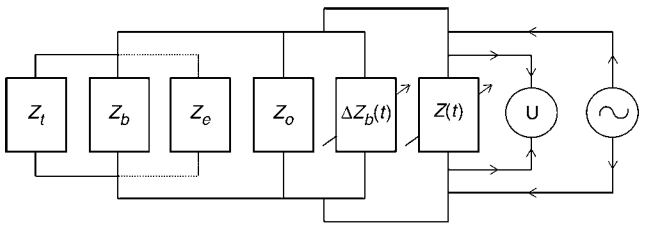

Fig. 1 *Schematic diagram of multi-compartment parallel conduction model of thorax. Transthoracic electrical impedance Z(t) (Ω) to applied AC field represents parallel connection of quasi-static base impedance Z_0 (Ω) and dynamic, time-dependent component of blood resistance $\Delta Z_b(t)$ (Ω). Z_0 represents parallel connection of all static tissue impedances Z_t (Ω) and static component of blood impedance Z_b (Ω). In disease states characterised by excess EVLW, quasi-static impedance Z_e (Ω) is added in parallel with Z_t and Z_b. Voltmeter (U) and AC generator (~) are shown*

where ζ = index of transthoracic aberrant conduction; Z_0 = measured transthoracic quasi-static base impedance (Ω); $Z_C = 20\Omega$ = the critical level of base impedance; and $K \to 0$. For all $Z_0 < 20\Omega$, $0 < \zeta < 1$. For all $Z_0 \geq 20\Omega$, $\zeta = 1$; $dZ/dt_{\max}$ = peak first time-derivative, peak slope, or maximum time-rate of change of the transthoracic cardiogenic impedance pulse variation ($\Omega\,\mathrm{s}^{-2}$); $\sqrt{[(dZ/dt_{\max})/Z_0]}$ = ICG acceleration step-down transformation (s^{-1}); T_{LVE} = left ventricular ejection time (s).

2.2 *Implicit assumptions of the newly proposed ICG equation N*

(*a*) Excluding the effect of pulmonary ventilation, the transthoracic electrical impedance $Z(t)$ (Ω) to an applied alternating current (AC) field is considered the parallel connection of a quasi-static DC component Z_0 (Ω) and a dynamic, cardiogenically induced, pulsatile AC component $\Delta Z(t)$ (VISSER *et al.*, 1987; 1990) (Fig. 1).

(*b*) Z_0 is considered the parallel connection of a highly conductive cylindrical blood resistance R_b (Ω) (i.e. Z_b), embedded within a thoracic-encompassing, poorly conductive cylindrical tissue impedance Z_t (Ω) (VISSER *et al.*, 1990) (Fig. 1).

(*c*) $\Delta Z(t)$ is a composite signal comprising a velocity-induced, blood 'resistivity'-based component $\Delta Z_v(t)$ ($\Omega\ \mathrm{s}^{-1}$) (i.e. $\Delta\rho_b(t)$, $\Omega\mathrm{cm\ s}^{-1}$) and a component of equal magnitude, related to pressure-induced, compliance-modulated, vessel volume changes $\Delta Z_{vol}(t)$ ($\Delta\Omega(t)$) (SAKAMOTO and KANAI, 1979; VISSER, 1989; VISSER *et al.*, 1990; KOSICKI *et al.*, 1986).

(*d*) The peak first time-derivative of $\Delta Z(t)$, $dZ/dt_{\max}$ ($\Omega\ \mathrm{s}^{-2}$), is bio-electrically equivalent to the peak rate of change of the blood resistivity component of $\Delta Z(t)$; that is, $d\rho_b(t)/dt_{\max}$ (i.e. $d[\Delta\rho_b(t)]/dt_{\max}$) ($\Omega\mathrm{cm\ s}^{-2}$).

(*e*) In the context of (*d*), $dZ/dt_{\max}$ is the ohmic image of peak aortic blood acceleration $dv/dt_{\max}$ ($\mathrm{cm\ s}^{-2}$).

(*f*) Haemodynamically, $dZ/dt_{\max}/Z_0$ (s^{-2}) is the bio-electric analogue of peak aortic reduced average blood acceleration (PARABA) ($[dv/dt_{mean}]/R)_{\max}$ (s^{-2}), this being the quotient of mean acceleration ($\mathrm{cm\ s}^{-2}$) and the aortic valve radius (cm).

(*g*) $dZ/dt_{\max}/Z_0$ must undergo square-root transformation to yield a dimensionless ohmic equivalent of mean aortic blood velocity $\Delta Z_v(t)_{\max}/Z_0$ (s^{-1}).

(*h*) Haemodynamically, $\Delta Z_v(t)_{\max}/Z_0$, a derived value, is the dimensionless bio-electric analogue of peak aortic reduced average blood velocity (PARABV) ($v_{mean}/R)_{\max}$ (s^{-1}), this being the quotient of mean aortic blood velocity and the aortic valve radius (VISSER, 1989).

(*i*) The volume conductor V_C (ml), is bio-electrically equivalent to the blood resistance, R_b, in the absence of excess extra-vascular lung water (EVLW) (ml).

(*j*) The physical embodiment of R_b is, by magnitude, equivalent to the intra-thoracic blood volume (ITBV, V_{ITBV}) (ml).

(*k*) SV is proportional to V_{ITBV} through allometric equivalents of body mass (kg).

(*l*) The magnitude of V_C is increased in the presence of excess EVLW (ml).

(*m*) Uncorrected, the magnitude of $dZ/dt_{\max}$ and thus ICG-derived SV are inversely related to the magnitude of excess EVLW (CRITCHLEY *et al.*, 2000; RAAIJMAKERS *et al.*, 1998; YOUNG and MCQUILLAN, 1993).

2.3 *Origin of dZ/dt_{max} through differential analysis*

If a segment of aorta is considered a cylindrical, thin-walled, blood-filled structure at end-diastole, its impedance Z to an