Impedance cardiography in haemodynamic monitoring of septic patients: a prospective study

Mariusz Piechota[1], Robert Irzmański[2], Maciej Banach[3], Jan Kowalski[2], Lucjan Pawlicki[2]

[1]Department of Anaesthesiology and Intensive Care Unit, University Hospital No. 5 in Lodz, Medical University of Lodz, Poland
[2]Department of Internal Diseases and Cardiologic Rehabilitation, University Hospital No. 5, Medical University of Lodz, Poland
[3]1st Chair of Cardiology and Cardiac Surgery, University Hospital No. 3 in Lodz, Medical University of Lodz, Poland

Submitted: 29 November 2005
Accepted: 23 August 2006

Arch Med Sci 2007; 3, 2: 145-151

Corresponding author:
Mariusz Piechota, MD, PhD
Department of Anaesthesiology and Intensive Care Unit
University Hospital No. 5 in Lodz,
Medical University of Lodz,
Hallera Square 1; 90-647 Lodz,
Poland
Phone: +48 42 639 30 90
Fax: +48 42 639 30 97
E-mail:
mariuszpiechota@poczta.onet.pl

Abstract

Introduction: The aim of the study was to evaluate the usefulness of impedance cardiography (ICG) in monitoring of septic patients.
Material and methods: It was a prospective study performed in 20 consecutive patients with sepsis. The criteria of sepsis according to the definition accepted at the ACCP/SCCM conference were the basis for being enrolled in the study. Six patients died in the course of the study and the investigated group was divided into subgroups treated effectively and ineffectively.
Results: The quality of ICG signal was determined in all 128 measurements. The quality of ICG was ≥70% in 53.91% of the measurements, ≥30% in 88.28% of the measurements. In 11.72% of the measurements the signal quality was <30%. In the effectively treated subgroup the values of NT-proBNP concentration in blood and the number of scores in SOFA were significantly lower ($p<0.05$), and a moderate positive significant correlation was found between SOFA score and stroke volume, cardiac output and index, acceleration index and NT-proBNP concentration. A significant negative correlation was demonstrated in relation to systemic vascular resistance index, left ventricular ejection time, mean arterial pressure, systolic and diastolic blood pressure. The following indices had a positive correlation with NT-proBNP concentration: heart rate, cardiac output and acceleration index. In the ineffectively treated subgroup only NT-proBNP concentration correlated with SOFA score.
Conclusions: ICG is a useful method for monitoring septic patients. It has an advantage over SOFA score. Indices investigated with ICG correlate with NT-proBNP concentration.

Key words: haemodynamic parameters, impedance cardiography, sepsis.

Introduction

In 1992 the decisions were published of the American College of Chest Physicians (ACCP) and the American Society of Critical Care Medicine (SCCM) defining among others: sepsis, severe sepsis and septic shock [1, 2]. The mentioned septic states, particularly severe sepsis and septic shock, are associated with serious prognostication and increased mortality [3]. The annually increasing number of septic patients is the consequence of aging of the population, the increase of resistance to antibiotics and popularization of invasive methods of treatment. Owing to severe clinical condition, septic states require multidirectional and precise monitoring.

Descriptive score scales also related to organ functioning have been introduced for diagnostic and prognostic needs [4-9]. Circulatory system is estimated on the basis of the values of mean arterial pressure and/or doses of the applied pressors. Testing more precise haemodynamic parameters requires introduction of invasive methods, quite costly and with the risk of serious complications [10, 11].

Impedance cardiography (ICG) is an interesting alternative to these methods. It is a totally non-invasive and easily applied technique for monitoring a wide spectrum of haemodynamic parameters. ICG was developed for NASA in the 1960s [12]. In the meantime a lot of validation studies have been published [13-27]. If the limitations of impedance cardiography are considered, impedance measurements – combined with high level signal evaluation, like the PASA algorithms – are comparable with established invasive methods.

The aim of the study was to assess the practical usefulness of impedance cardiography in monitoring of septic patients, its comparison with Sepsis-related Organ Failure Assessment (SOFA) score and verification of the objectivity of the investigated haemodynamic parameters by parallel testing of N-terminal brain natriuretic propeptide (NT-proBNP) concentration.

Material and methods

Having obtained the approval of the Bioethical Committee of the Medical University in Lodz, Poland (No. RNN/26/03/KB), 20 consecutive patients were qualified for the study: 15 men, aged 21 to 69 years (mean 48 years) and 5 women, aged 27 to 90 years (mean 61 years). The basic data about the investigated group are shown in Table I.

The criteria of sepsis according to the definition accepted at the ACCP/SCCM conference were the basis for being enrolled in the study.

Patients with aortic regurgitation, atrial septal defect, ventricular septal defect, severe hypertension [mean arterial pressure (MAP) >130 mmHg], tachycardia over 250/min, and extreme cases of overweight or underweight and height were excluded from the study.

The investigations were carried out in each patient until they stopped meeting the criteria of sepsis according to the definition accepted at the ACCP/SCCM conference or when the patient died.

Subjects were recruited consecutively from patients attending the Intensive Care Unit (ICU) from 1 July 2003 to 31 July 2004. All the patients were treated by the same team of physicians. The standard treatment included administration of adequate antibiotics, control of the source of infection and supportive therapy (intravenous fluids, medication aiding circulatory system, vasopressors, aiding the failing organs). The applied protocol ordered, among others, maintaining SpO_2 >90%, mean arterial pressure (MAP) at the level at least 70 mmHg, central venous pressure (CVP) within the limit 8-12 mmHg. If application of mechanical ventilation was necessary, $paCO_2$ had to be maintained within the limit of 35-45 mmHg.

Despite intensive therapy 6 patients died, which caused the division into two subgroups of effectively treated (n=14) and ineffectively treated (n=6) patients.

In each patient within 12 h of inclusion in the study, then 12, 24, 48, 96 h afterwards (next 48 h after the previous one), the following haemodynamic parameters were investigated: heart rate (HR) in beats/min, stroke volume (SV) in ml, cardiac output (CO) in l/min, cardiac index (CI) in l/min/m^2, pre-ejection period (PEP) in ms, acceleration index (ACI) in ms, systemic vascular resistance index (SVRI) in dyne.s/cm^{-5}/m^2, mean arterial pressure in mmHg, systolic blood pressure (BPs) in mmHg and diastolic blood pressure (BPd) in mmHg. Furthermore, NT-proBNP concentration was determined in arterial blood and the clinical state was evaluated according to SOFA score (Table II).

Haemodynamic indices were determined with impedance cardiograph NICCOMO (Medis. Medizinische Messtechnik GmbH, Germany) working on the basis of the Sramek-Berstein rule. The quality of the ICG signal was determined objectively with NICCOMO software in % from 0 to 100% (PASA algorithm).

Impedance cardiography is based on the measurement of the alternating current resistance (impedance) to a particular body part. As blood is a better conductor of current than other tissues, the change of electric resistance of a given body part enables the blood volume in this area to be evaluated. Thoracic aorta is the main source of ICG signal during systole. During diastole and in some particular pathologies (aortal valve defect, high pressure in pulmonary artery), pulsatile changes in large veins and pulmonary artery may significantly modify the source of the signal. The change of thoracic electric resistance is registered by a system of eight electrodes placed in strictly defined points of the thorax and neck. The principles of electrode location are: two at the base of the neck on both sides of the body; the second pair should be placed 5 cm over these; the third pair is located (one electrode on each side of the body) at the level of the xiphoid process; the fourth pair should be attached 5 cm below the third one. Electric current of low intensity and high frequency, which does not cause any discomfort to the patient, is introduced into the thorax through four electrodes placed on the skin. The reception of the returning current after passing through the thorax is completed by four receiving electrodes. The obtained data enable the impedance of this current to be calculated

Table I. Basic data on the studied group

Patient number	Basis of inclusion in the study	Infection site	No. of scores in APACHE II on the study initiation	Number of scores in SOFA on the study initiation	Number of measurements	The highest number of organ failure	Death in the course of the study	Cause of death
1	Sepsis	Abdominal cavity	7	1	5	0	No	–
2	Sepsis	Abdominal cavity	14	5	13	3	No	–
3	Severe sepsis	Abdominal cavity	17	5	6	1	No	–
4	Sepsis	Abdominal cavity	9	2	9	5	Yes	MODS, DIC
5	Severe sepsis	Abdominal cavity	10	2	6	2	No	–
6	Severe sepsis	Lungs/ Abdominal cavity	19	8	14	4	No	–
7	Sepsis	Abdominal cavity	3	1	5	1	No	–
8	Sepsis	Lungs	12	6	6	1	No	–
9	Severe sepsis	Lungs	14	9	4	1	No	–
10	Severe sepsis	Abdominal cavity	17	5	11	4	Yes	MODS
11	Severe sepsis	CNS	11	6	4	2	No	–
12	Severe sepsis	Abdominal cavity	8	4	4	1	No	–
13	Severe sepsis	Abdominal cavity	12	7	2	3	Yes	Respiratory – circulatory failure
14	Severe sepsis	Abdominal cavity	10	3	4	1	No	–
15	Severe sepsis	Abdominal cavity	13	8	6	3	Yes	Respiratory – circulatory failure
16	Severe sepsis	Lungs	24	13	3	4	Yes	Respiratory – circulatory failure
17	Severe sepsis	Abdominal cavity	7	5	5	2	No	–
18	Severe sepsis	Abdominal cavity	4	3	9	2	No	–
19	Severe sepsis	Abdominal cavity	11	6	8	2	No	–
20	Severe sepsis	Lungs	17	12	4	4	Yes	Respiratory – circulatory failure

according to the appropriate algorithm. The same electrodes register ECG.

NT-proBNP concentration in arterial blood was estimated with an immunoenzymatic test (the test is based on competitive EIA method). The reading was performed on an ETI Max 3000 analyzer (DiaSorin) using Biomedica reagents.

To compare the tested parameters appropriate statistical tests were used dependent on the quantity, sample matching and the type of the

Table II. SOFA score

System	Scores				
	0	1	2	3	4
Respiratory PaO_2/FiO_2	>400	≤400	≤300	≤200	≤100
Urinary creatinine (mcmol/l)	≥110	110-170	171-299	300-400 diuresis ≤500 ml/d	>440 diuresis <200 ml/d
Liver bilirubin (mcmol/l)	≤20	20-32	33-101	102-204	>204
Circulatory Hypotension	Lack of hypotension	MAP <70 mmHg	Dopamine ≤5* or dobutamine at optional dose	Dopamine >15* or adrenaline ≤0.1* or noradrenaline ≤0.1*	Dopamine >15* or adrenaline >0.1* or naradrenaline >0.1*
Hematopoietic No. of blood platelets	>150 000	≤150 000	≤100 000	≤50 000	≤20 000
Nervous Glasgow scale	15	13-14	10-12	6-9	<6

**catecholamines administered at least for an hour (doses in ucg/kg/min)*

Table III. Comparison of the investigated parameters in the effectively treated subgroup and ineffectively treated. Mean values, standard deviation, statistical significance p<0.05

Parameter	Effectively treated cases, mean SD n=14, 93 measurements	Death cases mean, SD n=6, 35 measurements	Significance of differences
Heart rate (1/min)	104.69±18.67	110.66±20.12	NS
Stroke volume (ml)	78.85±23.87	62.38±25.19	p<0.05
Cardiac output (l/min)	8.07±2.49	6.47±2.27	p<0.05
Cardiac index (l/min/m^2)	4.22±1.19	3.73±1.14	p<0.05
Pre-ejection period (ms)	50.35±24.91	46.11±29.42	NS
Acceleration index (1/s^2)	3.27±1.60	3.05±1.84	NS
Left ventricular ejection time (ms)	220.52±44.00	194.43±42.09	p<0.05
Systemic vascular resistance index (dyne.s/cm^{-5}/m^2)	1730.87±721.24	1796.97±723.33	NS
Mean arterial pressure (mmHg)	86.39±17.95	78.60±15.18	p<0.05
Blood pressure systolic (mmHg)	124.00±21.26	115.34±25.10	NS
Blood pressure diastolic (mmHg)	68.17±13.15	61.89±11.47	p<0.05
NT-proBNP (pg/ml)	124.45±77.75	184.27±87.93	p<0.05
SOFA score (pts)	5.30±3.08	9.00±4.07	p<0.05

investigated sample. To choose an appropriate test, the samples were checked whether they had normal distribution (Shapiro-Wilk test). When both samples had normal distribution, Student's t-test was used for unrelated samples. When at least one sample had a distribution different from normal, Wilcoxon's test was applied. The results of the testing are given in the form of p<max (e.g. p<0.05). This means that a significant difference was observed at the distinguished level of significance.

To determine the correlations, correlation coefficient was calculated: Pearson's correlation coefficient (for normal distribution) and Spearman's correlation coefficient (when at least one sample had other than normal distribution). The result is given in the form of p<max (e.g. p<0.05). This means that the correlation is statistically significant at the distinguished level of significance.

Results

The quality of ICG signal was determined in all 128 measurements. The quality of ICG was ≥70% in 53.91% of the measurements, ≥30% in 88.28% of the measurements. In 11.72% of the measurements the signal quality was <30%.

In the subgroup of effectively treated patients the following values were found to be significantly higher:

Table IV. Correlations between haemodynamic parameters and SOFA score and NT-proBNP in the effectively treated subgroup of patients

Parameter	SOFA score	NT-proBNP
Heart rate	r=0.137 NS	r=0.4009 p<0.001
Stroke volume	r=0.25 p=0.016	r=0.13 NS
Cardiac output	r=0.3291 p=0.001	r=0.3282 p=0.001
Cardiac index	r=0.2405 p=0.020	r=0.3362 p=0.001
Pre-ejection period	r=0.1601 NS	r=–0.0461 NS
Acceleration index	r=0.3848 p<0.001	r=0.2461 p=0.017
Left ventricular ejection time	r=–0.2436 p=0.019	r=–0.2762 p=0.007
Systemic vascular resistance index	r=–0.3378 p=0.001	r=–0.3911 p<0.001
Mean arterial pressure	r=–0.2968 p=0.004	r=–0.2231 p=0.032
Blood pressure systolic	r=–0.2417 p=0.020	r=–0.2892 p=0.005
Blood pressure diastolic	r=–0.2636 p=0.011	r=–0.216 p=0.038
NT-proBNP	r=0.4965 p<0.001	– –

r – correlation coefficient
Correlation coefficients given in the table are statistically significant p<0.05

Table V. Correlations between haemodynamic parameters and SOFA score and NT-proBNP in the ineffectively treated subgroup of patients

Parameter	SOFA score	NT-proBNP
Heart rate	r=0.1077 NS	r=0.2077 NS
Stroke volume	r=–0.2874 NS	r=–0.0637 NS
Cardiac output	r=–0.0687 NS	r=–0.1102 NS
Cardiac index	r=–0.2433 NS	r=–0.0332 NS
Pre-ejection period	r=0.232 NS	r=0.2479 NS
Acceleration index	r=–0.0798 NS	r=0.1747 NS
Left ventricular ejection time	r=–0.1963 NS	r=–0.2926 NS
Systemic vascular resistance index	r=0.0317 NS	r=–0.2074 NS
Mean arterial pressure	r=–0.1736 NS	r=–0.2027 NS
Blood pressure systolic	r=–0.277 NS	r=–0.3012 NS
Blood pressure diastolic	r=–0.2091 NS	r=–0.2598 NS
NT-proBNP	r=0.3487 p=0.040	– –

r – correlation coefficient
Correlation coefficients given in the table are statistically significant p<0.05

stroke volume, cardiac output, cardiac index, left ventricular ejection time, mean arterial pressure and diastolic blood pressure. The values of acceleration index, pre-ejection period, systemic vascular resistance index and systolic blood pressure did not differ significantly between subgroups. In the subgroup of effectively treated NT-proBNP concentration in blood and the number of scores in SOFA score were significantly lower. The results are presented in Table III.

In the subgroup of effectively treated patients a moderate positive significant correlation was demonstrated with the number of scores in SOFA and stroke volume, cardiac output, cardiac index, acceleration index and NT-proBNP concentration. A negative significant correlation was observed in relation to systemic vascular resistance index, left ventricular ejection time, mean arterial pressure, systolic and diastolic blood pressure. In this subgroup pre-ejection period and heart rate did not correlate with SOFA score, while a positive correlation was observed between NT-proBNP concentration and HR, CO, CI and ACI. A negative correlation of NT-proBNP concentration was demonstrated in relation to SVRI, LVET (left ventricular ejection time), MAP, BPs and BPd. SV and PEP did not correlate with NT-proBNP concentration. The results are presented in Table IV.

In the subgroup of ineffectively treated patients only NT-proBNP concentration correlated with SOFA score. The results are presented in Table V.

Discussion

This study should be treated as a preliminary examination owing to the small number of samples.

Reliability of the obtained results of these haemodynamic parameters depends first of all on the quality of the ICG signal. According to the authors, the quality of the ICG signal was in this study very good (≥70%) in 53.91% of the measurements and satisfactory (≥30%) in 88.28% of the measurements. Only in 11.72% of the measurements should this quality be considered to be low (<30%).

In the course of the investigations there was unintentional division of patients into two subgroups: I – effectively treated (n=14), and II – ineffectively treated (n=6). The disproportion in the number of patients in each subgroup does not allow one to draw categorical conclusions, but the analysis of the results enables distinct trends to be observed.

In the subgroup of effectively treated patients significantly higher values of the following parameters were detected: stroke volume, cardiac output and its derivative, cardiac index, left ventricular ejection time, mean arterial pressure and diastolic blood pressure. The other parameters did not differ significantly between subgroups. It results from the above that the value of cardiac output was calculated from the product of HR and SV. In effectively treated patients it depends first of all on the increase of stroke value. The accompanying longer left ventricular ejection time clearly points to more efficient cardiac function in patients from this subgroup. Furthermore, significantly lower values of NT-proBNP concentration would confirm such a conclusion and lower SOFA score proves better clinical state of the effectively treated patients.

The correlation of some haemodynamic parameters with NT-proBNP and SOFA score, being more synthetic information than dynamic presentation of particular results, gives even more convincing evidence for this suggestion. A significant positive correlation was demonstrated with reference to heart rate, cardiac output, cardiac index and acceleration index, which is the measure of heart inotropic state independent of afterload. A negative correlation was observed between NT-proBNP concentration, SOFA score and systemic vascular resistance index, systolic, diastolic and mean blood pressure and left ventricular ejection time. None of the above-mentioned correlations were found in the patients who died.

The correlation of parameters associated with vascular tone, SVRI, BPs, BPd and MAP, seems to be of particular importance. In severe sepsis, as NT-proBNP concentration indicating the worsening of heart failure increases the peripheral vessels” resistance resulting in the decrease of arterial pressure diminishes. Such direction of changes, proving growing vasoplegia, is prognostically unbeneficial. It should be emphasized that lack of statistical differences in SVRI value between subgroups was caused by administration of higher doses of pressors due to lower values of arterial pressure in the subgroup of patients who died.

The MAP value of about 85 mmHg observed in the subgroup of effectively treated patients seems to be a limiting value. In the cases of diminishing arterial blood pressure, but with vascular tone assuring MAP maintenance at a level not lower than 85 mmHg, the increase in successive measurement values of volume haemodynamic parameters (SV, CO, CI) point to the existence of heart functional reserve capable of compensating diminishing pressure. This should be considered to be a beneficial phenomenon. With MAP below 85 mmHg the correlations between NT-proBNP and SOFA score and the investigated haemodynamic parameters disappear. Thus, the trend when the decrease of SVRI and MAP is accompanied by a decrease in volume indices (SV, CO, CI, ACI, LVET) should be considered to be particularly disturbing.

The results of our own studies demonstrated that in septic patients alterations of parameters describing vascular tone (SVRI, MAP) are more significant prognostically than other ones such as cardiac output. This was rather surprising for the authors as the role of this parameter in the assessment of cardiac performance is well established. However, similar results were obtained by other authors. Parker et al., in a group of 20 patients with septic shock, found normal or decreased values of cardiac index being a derivative of cardiac output [28]. Ellrodt et al. observed decreased LVET in 94% of patients with similar diagnosis, which may reflect the shortening of the time of mechanical left ventricular performance [29]. In the case of sepsis resulting in death, significantly lower values of SVRI and MAP were detected [30]. Some authors suggest using SVRI as an index of significant importance in the prognostication of the response to treatment [31].

Conclusions

Impedance cardiography is a non-invasive, safe and objective method of testing haemodynamic parameters, useful in monitoring septic patients. This method enables assessment of numerous haemodynamic parameters in real time, with immediate reading of the results and repetition of the measurements in optional time intervals. Thus it has a significant advantage over descriptive SOFA score, with which however it demonstrates a good correlation. On the other hand, the indices tested with ICG correlate with NT-proBNP, which confirms their objectivity in cardiac efficiency description.

In septic patients the predictive value of particular haemodynamic parameters is variable. In the authors” opinion the decrease in the value of indices associated with vascular tone (SVRI and MAP) is of significant prognostic importance. However, binding conclusions may be drawn on the basis of the evaluation of a few indices taking into consideration the direction of their changes in time and the effect of the applied pressor drugs.

References

1. Bone RC, Balk RA, Cerra FB, Dellinger RP, Fein AM, Knaus WA, et al. Definitions for sepsis and organ failure and guidelines for the use of innovative therapies for sepsis. The ACCP/SCCM Concensus Conference Committee. American College of Chest Physicians/Society of Critical Care Medicine. Chest 1992; 101: 1644-55.
2. Members of the American College of Chest Physicians/Society of Crit Care Med Consensus Conference Committee: American College of Chest Physicians/Society

of Crit Care Med Consensus Conference. Definitions for sepsis and organ failure and guidelines for the use of innovative therapies in sepsis. Crit Care Med 1992; 20: 864-74.
3. Angus DC, Linde-Zwirble WT, Lidicker J, Clermont G, Carcillo J, Pinsky MR. Epidemiology of severe sepsis in the United States: analysis of incidence, outcome and associated costs of care. Crit Care Med 2001; 29: 1303-10.
4. Antonelli M, Moreno R, Vincent JL, Sprung CL, Mendoca A, Passariello M, et al. Application of SOFA score to trauma patients. Sequential Organ Assessment. Intensive Care Med 1999; 25: 389-94.
5. Janssens U, Graf C, Graf J, Radke PW, Konigs B, Koch KC, et al. Evaluation of the SOFA score: a single-center experience of medical intensive care unit in 303 consecutive patients with predominantly cardiovascular disorders. Sequential Organ Failure Assessment. Intensive Care Med 2000; 26: 1037-45.
6. Moreno R, Vincent JL, Matos R, Mendonca A, Cantraine F, Thijs L, et al. The use of maximum SOFA score to qualify organ dysfunction/failure in intensive care. Results of prospective, multicenter study. Working Group on Sepsis Related Problems of the ESICM. Intensive Care Med 1999; 25: 686-96.
7. Oda S, Hirasawa H, Sugai T, Shiga H, Nakanishi K, Kitamura N, et al. Comparison of Sepsis-related Organ Failure Assessment (SOFA) score and CIS (cellular injury score) for scoring of severity for patients with multiple organ dysfunction syndrome (MODS). Intensive Care Med 2000; 26: 1786-93.
8. Vincent JL, de Mendonca A, Cantraine F, Moreno R, Takala J, Suter PM, et al. Use of SOFA score to assess the incidence of organ dysfunction/failure in intensive care units: result of a multicenter, prospective study. Working group on „sepsis related problems" of the European Society of Intensive Care Medicine. Crit Care Med 1998; 26: 1793-800.
9. Hantke M, Holzer K, Thone S, Schmandra T, Hanisch E. The SOFA score in evaluating septic illness. Correlations with the MOD and APACHE II score. Chirurg 2000; 71: 1270-6.
10. Elliott CG, Zimmerman GA, Clemmer TP. Complications of pulmonary artery catheterization in the care of critically-ill patients. A prospective study. Chest 1979; 76: 647-52.
11. Matthay MA, Chatterjee K. Bedside catheterization of the pulmonary artery: Risk compared with benefits. Ann Intern Med 1998; 109: 826-34.
12. Kubicek WG, Karnegis JN, Patterson RP, Witsoe DA, Mattson RH. Development and evaluation of an impedance cardiac output system. Aerosp Med 1966; 37: 1208-12.
13. Gotshall RW, Wood VC, Miles DS. Comparison of two impedance cardiographic techniques for measuring cardiac output in critically ill patients. Crit Care Med 1989; 17: 806-11.
14. Wong DH, Tremper KK, Stemmer EA, O'Connor D, Wilbur S, Zaccari J, et al. Noninvasive cardiac output: simultaneous comparison of two different methods with thermodilution. Anesthesiology 1990; 72: 784-92.
15. Raaijmakers E, Faes TJ, Kunst PW, Bakker J, Rommes JH, Goovaerts HG, et al. The influence of extravascular lung water on cardiac output measurements using thoracic impedance cardiography. Physiol Meas 1998; 19: 491-9.
16. Zacek P, Kunes P, Kobzova E, Dominik J. Thoracic electrical bioimpedance versus thermodilution in patients post open-heart surgery. Acta Medica (Hradec Kralove) 1999; 42: 19-23.
17. Critchley LA, Calcroft RM, Tan PY, Kew J, Critchley JA. The effect of lung injury and excessive lung fluid, on impedance cardiac output measurements, in the critically ill. Intensive Care Med 2000; 26: 679-85.
18. Van De Water JM, Miller TW, Vogel RL, Mount BE, Dalton ML. Impedance cardiography: the next vital sign technology? Chest 2003; 123: 2028-33.
19. Koobi T, Kaukinen S, Turjanmaa VM. Cardiac output can be reliably measured noninvasively after coronary artery bypass grafting operation. Crit Care Med 1999; 27: 2206-11.
20. Ng HW, Coleman N, Walley TJ, Mostafa SM, Breckenridge AM. Reproducibility and comparison of cardiac output measurement by transthoracic bioimpedance and thermodilution methods in critically ill patients. Clin Intens Care 1993; 4: 217-21.
21. Fuller HD. The validity of cardiac output measurement by thoracic impedance: a meta-analysis. Clin Invest Med 1992; 15: 103-12.
22. Jensen L, Yakimets J, Teo KK. A review of impedance cardiography. Heart Lung 1995; 24: 183-93.
23. Wong DH, Tremper KK, Stemmer EA, O'Connor D, Wilbur S, Zaccari J, et al. Noninvasive cardiac output: simultaneous comparison of two different methods with thermodilution. Anesthesiology 1990; 72: 784-92.
24. Young JD, McQuillan P. Comparison of thoracic electrical bioimpedance and thermodilution for the measurement of cardiac index in patients with severe sepsis. Br J Anaesth 1993; 70: 58-62.
25. Shoemaker WC, Wo CC, Yu S, Farjam F, Thangathurai D. Invasive and noninvasive haemodynamic monitoring of acutely ill sepsis and septic shock patients in the emergency department. Eur J Emerg Med 2000; 7: 169-75.
26. Rosenberg P, Yancy CW. Noninvasive assessment of hemodynamics: an emphasis on bioimpedance cardiography. Curr Opin Cardiol 2000; 15: 151-5.
27. Moshkovitz Y, Kaluski E, Milo O, Vered Z, Cotter G. Recent developements in cardiac output determination by bioimpedance: comparison with invasive cardiac output and potential cardiovascular applications. Curr Opin Cardiol 2004; 19: 229-37.
28. Parker MM, Shelhamer JH, Bacharach SL, Green MV, Natanson C, Frederick T, et al. Profound but reversible myocardial depression in patients with septic shock. Ann Intern Med 1984; 100: 483-90.
29. Ellrodt AG, Riedinger MS, Kimchi A, Berman DS, Maddahi J, Swan HJ, et al. Left ventricular performance in septic shock: reversible segmental and global abnormalities. Am Heart J 1985; 110: 402-9.
30. Groeneveld AB, Bronsveld W, Thijs LG. Hemodynamic determinants of mortality in human septic shock. Surgery 1986; 99: 140-53.
31. Pilz G, Werdan K. Cardiovascular parameters and scoring systems in the evaluation of response to therapy in sepsis and septic shock. Infection 1990; 18: 253-62.