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Impedance cardiography: A noninvasive way to monitor hemodynamics

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Impedance cardiography is a noninvasive way to measure hemodynamics and thoracic fluid status that can be quickly implemented by the nurse in any setting. This article describes the principles and use of impedance cardiography and how it can allow nurses to improve patient outcomes through early recognition of hemodynamic abnormalities.

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Early hemodynamic monitoring and interventions may improve patient outcomes,[1] but clinicians often hesitate or do not insert a pulmonary artery (PA) catheter in order to avoid invasive procedures. Impedance cardiography (ICG) offers a noninvasive way to quickly measure hemodynamics and thoracic fluid status and can be initiated by nurses in any health care setting, from telemetry units to patients' homes.

This article will review the principles of ICG and how nurses can use the information provided by ICG to recognize perfusion problems early and intervene appropriately.

UNDERSTANDING ICG

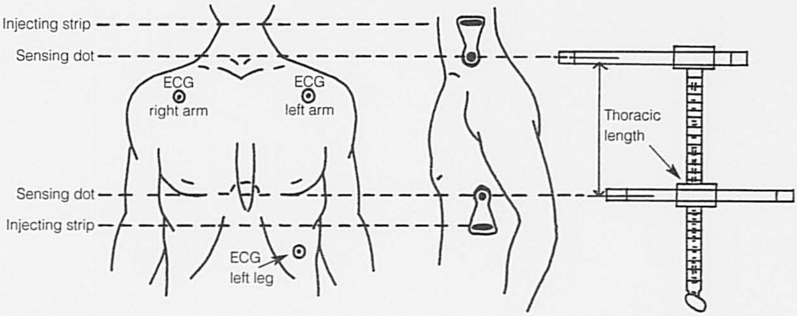
Unlike conventional hemodynamic monitoring, which requires a PA catheter and a physician to insert it, ICG monitoring can be started by nurses at the bedside, using four ICG electrodes and three electrocardiograph (ECG) electrodes that can be placed in a matter of minutes (Figure 1). Data provided by ICG reflects overall fluid in the chest and information about left ventricular (LV) function, preload, afterload, and contractility (Table 1).

Historically, NASA used ICG to evaluate the effects of zero gravity on the astronauts while in space.[2] Although the technology worked well on healthy individuals, it was unreliable in critically ill patients. Advances in computer technology and signal processing have made the technology more reliable and suitable for use in the critical care setting.

Like conventional hemodynamic

FIGURE 1

How to use impedance cardiography



Applying the impedance cardiography (ICG) and ECG electrodes and setting up the monitor takes about 10 minutes. The four ICG electrodes are placed on landmarks defining the thorax (the illustration shows only one side of the body). Two electrodes are placed at the base of the neck and two at the level of the xiphoid-sternal junction. Each electrode consists of an injecting strip and a sensing dot. The strip emits a high-frequency, low-amplitude alternating electrical current that passes through the patient's chest. (The patient cannot feel the current, which is about equal to that of a pediatric apnea monitor.) The dot senses the voltage change—the impedance met by the current as it flows through the patient's thorax. The three ECG leads are placed in a standard lead II configuration, and patient data (including the thoracic length, or the distance between the upper and lower dot electrodes) is entered into the computer. All electrodes are connected to the portable ICG monitor, which calculates the ICG parameters.

monitoring, ICG looks at the factors affecting tissue and organ perfusion: blood volume, vascular tone, and cardiac action. Studies show excellent correlation between ICG and thermodilution estimates of cardiac output (CO) in multiple patient populations.[3,4] Other studies have demonstrated a strong correlation between Zo, the ICG parameter that measures thoracic fluid status, and chest X-ray findings.[5,6]

About the base impedance

As its name implies, impedance cardiography measures the total impedance, or resistance to the flow of electricity, in the chest. Impedance is represented by the symbol Z and is measured in ohms.

Average thoracic impedance, or Zo, is also known as the base impedance and reflects the total fluid status of the chest. Electrical current naturally seeks the path of least resistance, and fluid is an excellent conductor of electricity. Electrical current travels more easily through a "wet" chest in which plasma and fluid have accumulated due to pulmonary edema, effusions, or infiltrates, so the reduced impedance is reflected in a low Zo value.

Conversely, bone, tissue, and air are poor conductors of electricity. In such a "dry" chest, electrical current meets a higher amount of resistance or impedance, resulting in a higher Zo. This inverse relationship between thoracic fluid volume and Zo can be remembered with the saying "high is dry."

The impedance waveform

The impedance waveform (Figure 2) is known as the dZ/dt waveform, with dZ representing the change in impedance and dt the change in time. This value, derived from Zo, provides data regarding blood flow: The change in impedance is produced by variations in blood flow and volume in the ascending aorta during one cardiac cycle.[7] Changes in the volume of blood and the alignment of the erythrocytes cause a relative impedance change during systole and diastole and contribute to the impedance signal.[7]

The dZ/dt also has an associated value that reflects cardiac contractility and serves as a measure of signal strength.

During systole, the increased volume and velocity of blood in the aorta cause the erythrocytes to align, lowering impedance (Figure 3). During diastole, less volume and velocity of blood in the aorta cause a more

TABLE 1
Impedance cardiography parameters

Hemodynamic variable	Parameter	Description	Normal values
Thoracic fluid status	Zo—base thoracic impedance	Measures the fluid status of the chest	Males: 20-30 ohms Females: 25-35 ohms
Left ventricular function	CO—cardiac output	Amount of blood ejected from the left ventricle per minute	4-8 liters/min
	CI—cardiac index	CO indexed to body surface area	2.5-4.5 liters/min/m^2
Preload	SV—stroke volume	Amount of blood ejected per heartbeat	60-100 ml/beat
Afterload	SVR—systemic vascular resistance	Amount of resistance against which the heart must pump	800-1,200 dynes/sec/m^{-5}
Contractility	dZ/dt—change in impedance over time	Reflects the force of left ventricular contraction	0.8-2.5 ohms/sec
	ACI—acceleration contractility index	Reflects the force of left ventricular contraction	2-5 ohms/sec^2
	LCWI—left cardiac work index	Measures myocardial oxygen demand	3-5 kg/min/m^2
	PEP—preejection period	Isovolumetric contraction	0.05-0.12 sec
	VET—ventricular ejection time	Period of time blood is ejected from the left ventricle	0.25-0.35 sec

random alignment of erythrocytes and contributes to higher impedance. The dZ/dt waveform therefore represents the mechanical activity of the left ventricle. Figure 2 shows the relationship of electrical stimulation on the ECG to the mechanical activity represented by the dZ/dt waveform.

LV function

As with conventional hemodynamic monitoring, ICG measures LV function through the CO and the cardiac index (CI). To estimate CO, the ICG monitor multiplies the patient's stroke volume (SV) by his heart rate. The CI is calculated in the usual method, by dividing CO by body surface area.

Measuring preload through SV

The SV is calculated on a beat-to-beat basis and is derived from the dZ/dt waveform. Manufacturers of ICG monitors each use various methods of signal processing for data acquisition.[8] To obtain SV, one manufacturer uses a time-power-frequency distribution and power spectrum analysis to identify specific landmarks on the dZ/dt waveform that correspond to the opening and closing of the aortic valve.[9] Figure 2 shows the B and X points, which denote aortic valve opening and closure, respectively. These landmarks have been validated by phonocardiography.[10] The time interval between the B and X points reflects ventricular ejection time. The C point (dZ/dt_{max}) corresponds with the peak flow of blood from the LV into the aorta, or the maximum impedance change, which is influenced by the SV.

In patients with a low CO, it is not always obvious if the low flow state is preload-dependent. Traditionally, LV filling pressures, via a PA catheter, are used to gauge preload. This practice assumes that the pulmonary artery occlusion pressure (PAOP) reflects pulmonary venous pressure, which, in turn, is in agreement with left atrial pressure and LV end-diastolic pressure.

Clinicians often interpret pressures to equal blood flow and volumes. But this is frequently not the case, because of pathophysiologic derangements in the critically ill patient.[11,12]

Using ICG, critical care nurses can perform a simple maneuver to assess intravascular fluid status. This is administering a noninvasive fluid challenge using the patient's own circulating volume as the fluid bolus. When the patient changes from a sitting to supine position, gravity moves fluid into the patient's torso from the lower extremities, increasing venous return. The degree of change in SV caused by increased preload reflects the cardiac reserve.

In a normal patient, the SV will increase moderately. Patients who are hypovolemic may show a significant improvement in SV, signifying adequate cardiac reserve and improved LV contractile force (Frank-Starling law). These patients may benefit from volume administration.

Patients with LV dysfunction or fluid overload may have minimal or no change in SV in response to a physiologic fluid bolus. These individuals do not have adequate cardiac reserve and cannot tolerate additional fluid. These patients may require treatment with positive inotropes or agents that reduce preload and afterload.

Assessing afterload

Systemic vascular resistance (SVR) is a derived parameter affected by many factors, including CO, mean arterial pressure, and central venous pressure (CVP). Because CVP is not measured in ICG, a default is used in the SVR calculation. The actual measured CVP should always be entered into the computer when available. An elevated SVR is frequently a compensatory mechanism in response to hypovolemia or neurohormonal activation. Therefore it is important to treat the primary cause of an elevated SVR, not the value itself.

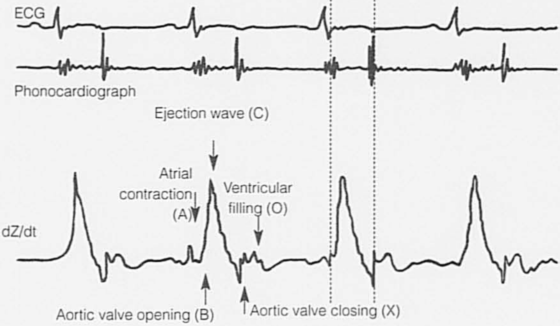
Cardiac contractility

Impedance cardiography contractility indexes include the dZ/dt and the acceleration contractility index (ACI), which are derived from the dZ/dt waveform. The dZ/dt describes the maximum impedance change. The dZ/dt value must be greater that 0.3 ohm/second, reflecting adequate signal strength, to ensure reliable data acquisition. The ACI describes acceleration or the rate of change of blood flow as it leaves the LV. The upslope of the dZ/dt waveform (from points B to C) is directly related to the velocity of blood in the ascending aorta as it is ejected from the LV, which is associated with the contractile properties of the heart. Greater contractility is associated with a steeper upslope. These contractility parameters are useful in determining the effectiveness of interventions, such as the administration of positive inotropes, by following trends in ACI and dZ/dt over time.

FIGURE 2

The dZ/dt waveform

This illustration shows how the ECG, which measures the heart's electrical activity, corresponds to the dZ/dt waveform, which measures the heart's mechanical activity.



The left cardiac work index (LCWI), which reflects myocardial work and oxygen demand, is a derived parameter. Mean arterial pressure, PAOP, and CI influence the LCWI. Default CVP and PAOP values are used in the LCWI calculation, but the measured values should always be entered when available.

Systolic time intervals reflect the length of electromechanical systole and LV function by providing a temporal relationship between the preejection period (PEP) and the ventricular ejection time (VET). Electromechanical systole spans the time from the onset of the QRS complex to the closing of the aortic valve. The PEP is the period of time from the onset of ventricular depolarization to the beginning of ventricular ejection. It is the amount of time required for the myocardium to generate enough pressure to open the aortic valve (isovolumetric contraction). The VET is the interval of ventricular ejection during systole, or the amount of time that the aortic valve is open. In hearts with impaired ventricular function, the ratio of PEP to VET is increased, where the PEP lengthens and the VET shortens.[13] Interventions that improve LV function and contractility should shorten the PEP. Systolic time intervals may be used to augment other measures of ventricular performance.

DZ/DT WAVEFORM MORPHOLOGY

The morphology of the systolic portion of the dZ/dt waveform, including the opening (B point) and closing (X point) of the aortic valve, the peak flow of blood (C point), and the maximum impedance change was previously discussed. An A wave (Figure 2) precedes the opening of the aortic valve and corresponds to atrial filling (atrial diastole). The ventricular diastolic portion of the waveform includes an O wave, which has a temporal relationship with mitral valve closure. An O wave exaggerated in amplitude, also known as an early diastolic wave, is associated with the rapid ventricular filling phase (early ventricular diastole).[14-16]

The presence of a large early diastolic O wave identified patients at higher risk for various forms of cardiomyopathies and may be used as a predictor of outcomes, both in terms of functional disability and death.[14] Patients with mitral regurgitation were found to have a modified dZ/dt waveform, including a pronounced O wave. These changes correlated with the severity of mitral regurgitation as measured by cardiac catheterization, and the O wave returned to normal in all patients who underwent a mitral valve replacement.[15]

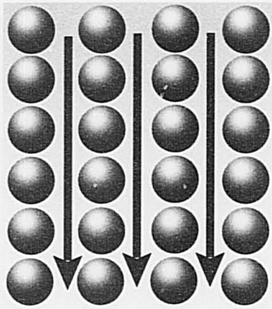
Investigators note that the dZ/dt waveform may prove to be a sensitive noninvasive measure of the severity of aortic regurgitation.[17] Heart failure patients were found to have abnormal dZ/dt waveforms. The systolic portion of the waveform was low and widened, with a notched or W pattern observed in some patients, and an exaggerated O wave was present in the diastolic portion of the waveform.[16]

Like many physiologic waveforms, the dZ/dt waveform provides a significant amount of diagnostic information. Although several investigators have examined the morphology of the dZ/dt waveform, much work is yet to be done, providing multiple opportunities for nursing research in the area of waveform analysis.

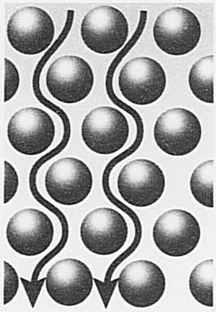
FIGURE 3

Impedance and the cardiac cycle

Impedance is reduced during systole (left) because erythrocytes are aligned in the aorta, allowing an increased flow of electrical current. During diastole, however, the random arrangement of erythrocytes (right) increases impedance and reduces the flow of electrical current.



Systole—low Zo



Diastole—high Zo

POTENTIAL PROBLEMS WITH ICG

Extremely large amounts of thoracic fluid may interfere with the impedance signal, making hemodynamic data unattainable or unreliable.[18] Conditions such as severe pulmonary edema may decrease the signal-to-noise ratio, damp the dZ/dt waveform, and inhibit hemodynamic data acquisition. However, Zo, the primary ICG measurement, can always be obtained and displayed. As the extreme volumes of thoracic fluid decrease, the signal-to-noise ratio improves and hemodynamic data becomes available.

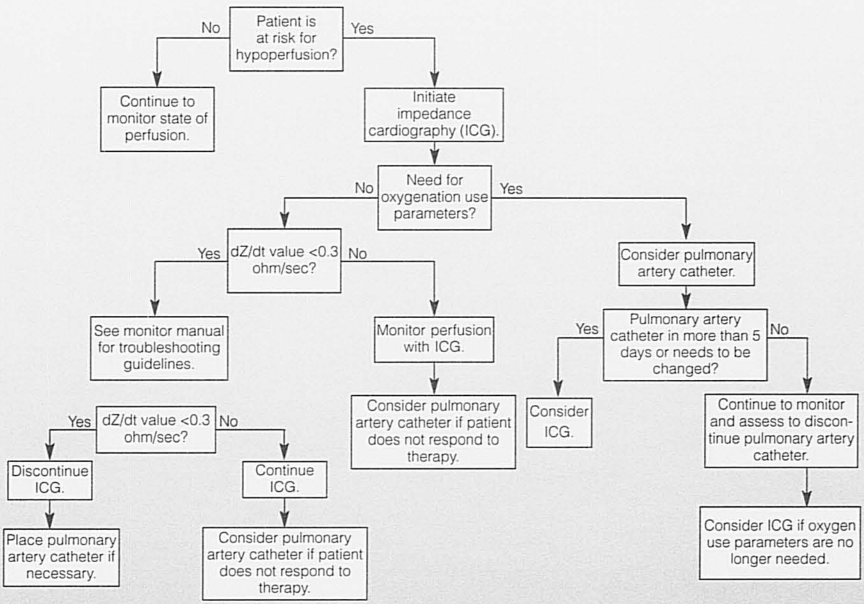
WHO BENEFITS?

A wide range of patients in a variety of health care settings may benefit from ICG monitoring. Impedance cardiography aids in the assessment and management of thoracic fluid status, oxygen debt, hypoperfusion, preload, cardiac performance, and need for invasive monitoring. The ability to objectively and continuously quantify, trend, and manipulate the determinants of CO (preload, afterload, contractility, and heart rate) and the fluid status of the chest affords an innovative and essential pathway to patient management.

The ability to continuously monitor the fluid status of the chest may allow early detection and decrease or negate some complications observed in the critical care area. Pulmonary congestion and edema are frequent problems in the critical care unit. Aggressive fluid resuscitation, unavoidable in some situations, frequently contributes to increasing pulmonary fluid and additional complications. A slow decrease in Zo may indicate in-

FIGURE 4

Noninvasive hemodynamic monitoring (impedance cardiography) algorithm



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creasing pulmonary edema and alert clinicians to alter management. A decrease in Zo may also be associated with development of pleural effusions or infiltrates. A rapid downward trend in Zo may be observed in patients who develop mechanical bleeding after cardiothoracic surgery.

In the critically ill patient, evaluation of the components of oxygen delivery (pulmonary gas exchange, hemoglobin, the binding of oxygen to hemoglobin, and CO) is important to prevent oxygen debt accumulation. Prolonged oxygen debt is associated with the systemic inflammatory response syndrome, multiple organ dysfunction syndrome, and increased mortality due to cell death.[19] Reversal of oxygen debt requires timely and appropriate management. Using ICG data provides the ability to detect threats to circulatory impairment and oxygen delivery, which allows for early intervention and optimization of CO and its determinants.

Preload assessment is essential in any patient who may be at risk for hypoperfusion. Assessment and management of preload can be a challenge for clinicians. Clinical signs and symptoms often lag behind the physiologic derangements. The PAOP indirectly measures LV end-diastolic pressure and is related to LV end-diastolic volume, or preload. However, many factors affect the relationship of pressure and volume, such as reduced LV compliance or mechanical ventilation. Critical care nurses must evaluate the circumstances that alter this pressure-volume relationship and weaken the validity of the PAOP as an indicator of LV preload.[20-23]

Use of ICG to assess preload via a noninvasive fluid challenge is an alternative to inserting a PA catheter, requires little nursing time, and affords valuable information regarding the patient's ability to tolerate additional fluids. This 2-minute procedure provides quantitative and objective data that is sometimes left to an educated guess when clinicians are unable to use PA catheter data. The familiar routine in a patient with an unknown fluid status—administering volume if the patient does not respond to a diuretic—may be avoided.

Management of heart failure patients may be enhanced by the use of ICG. There are four primary determinants of cardiac performance—contractility, preload, afterload, and heart rate—all of which may be assessed with ICG. Contemporary management of heart failure encompasses correcting hemodynamic abnormalities as well as suppressing the neurohormonal responses that perpetuate the condition.[24] Impedance cardiography monitoring may contribute to optimizing titration of diuretics, inotropes, and afterload-reducing agents. Assessment of the Zo, CO, ACI, SVR, and SV, and the hemodynamic response to a noninvasive fluid challenge, can provide the clinician with data to make informed decisions about the patient's care and may improve outcomes. Responses to interventions may be quantified and support further titration of medications or confirm the need to augment the plan of therapy.

Impedance cardiography monitoring may enable clinicians to justify the insertion of a PA catheter. Data may suggest that close observation and management with ICG may be sufficient or it may indicate that invasive monitoring is warranted. One institution has developed an algorithm to determine if and when invasive or noninvasive monitoring should be initiated or terminated (Figure 4). Implementation of this type of protocol may decrease the number of invasive procedures while increasing the number of patients who receive hemodynamic monitoring.

Using an ICG algorithm may increase the number of patients who receive hemodynamic monitoring.

CASE STUDY

The following case study demonstrates the use of early ICG monitoring in a postoperative patient as a tool for differential diagnosis. It reveals the advantage of obtaining objective data early in a patient's course and shows the usefulness of establishing baseline data and following trends over time.

Ms. R. was an 84-year-old woman with a history of two myocardial infarctions, hypertension, and Type 2 diabetes. She had coronary artery bypass graft surgery; her postoperative course was uncomplicated. Ms. R. was extubated late in the day of the surgery, and a baseline ICG profile was obtained. She was weaned off all drug infusions early on the second postoperative day.

Later that evening, Ms. R. became acutely short of breath, diaphoretic, cool and clammy, tachycardic, and hypotensive. Based on vital signs and symptoms alone, an initial reaction may have been to administer fluids to support the blood pressure. The nurse noted a decrease in CI from 2.3 to 1.2 liters/minute/m^2, and the Zo had fallen from 18 ohms, considered good for a second-day postoperative cardiac surgery patient, to 16 ohms, indicating that the chest was becoming wetter.

After evaluating the baseline ICG values, the nurse performed a noninvasive fluid challenge to assess the patient's preload. There was no change in SV with the physiologic fluid bolus, indicating that the LV may not tolerate the additional fluid. The decrease in Zo indicated an accumulation of fluid in the chest. The nurse concluded that the low CO state was not secondary to blood loss or a decrease in preload and that additional volume administration was inappropriate. The nurse also identified a decrease in the ACI, indicating a decline in cardiac contractility.

The nurse consulted with the physician and, based on the objective data, they chose to administer a positive inotrope to improve LV function. The patient showed a favorable response to the addition of the inotrope. Within 2 hours, the CI increased to 2.4 liters/minute/m^2, the Zo was back to 18 ohms, the ACI had increased, and all symptoms were resolving. Correct interpretation of ICG data and institution of appropriate interventions may have prevented a reintubation.

SUMMARY

Noninvasive thoracic and hemodynamic monitoring via ICG provides an alternative means of patient monitoring in the critical care setting. Impedance cardiography monitoring may improve patient management through use of nearly real time and continuous data and is not associated with the risks of invasive monitoring. Early detection of hemodynamic derangements in the critically ill may lead to early intervention and improved patient outcomes. Impedance cardiography is easy to apply, requires less nursing time, and can be used in any health care setting.

Impedance cardiography offers autonomy to critical care nurses in the area of thoracic fluid status and hemodynamic surveillance. Objective data from ICG can validate the hunches that clinicians often have but are not always able to substantiate with evidence. Most important, patients can benefit from early diagnosis of cardiopulmonary dysfunction and appropriate interventions, including those patients who may otherwise not be candidates for invasive hemodynamic surveillance.

In today's health care climate, clinicians are challenged to improve patient care and, at the same time, focus on cost containment by decreasing the length of stay and minimizing resource use. This challenge has forced critical care nurses to implement multiple changes to meet the needs of patient care and the requirements of managed-care companies. The integration of ICG monitoring provides the potential to both improve patient outcomes and decrease cost by allowing for early detection and intervention of hemodynamic derangements. ■

REFERENCES

1. Shoemaker WC, et al. Early physiologic patterns in acute illness and accidents: Toward a concept of circulatory dysfunction and shock based on invasive and noninvasive hemodynamic monitoring. *New Horizons* 1996; 4(4):395-412.
2. Kubicek WG, et al. Development and evaluation of an impedance cardiac output system. *Aerospace Medicine* 1966;37:1208-12.
3. Thangathurai D, et al. Continuous intraoperative noninvasive cardiac output monitoring using a new thoracic bioimpedance device. *Journal of Cardiothoracic and Vascular Anesthesiology* 1997;11:440-4.
4. Shoemaker WC, et al. Multicenter study of noninvasive monitoring systems as alternatives to invasive monitoring of acutely ill emergency patients. *Chest* 1998;114:1643-52.
5. Peacock WF, et al. Bioimpedance monitoring: Better than chest X-ray for predicting abnormal pulmonary fluid? [abstract] *Academic Emergency Medicine* 1999;6(5):441.
6. Milzman D, et al. The utility of thoracic impedance to evaluate chest radiograph changes from acute heart failure patients in the emergency department. *Journal of Cardiac Failure* 1998;4(Suppl):37.
7. Lamberts R, Visser KR, Zijlstra WG. *Impedance Cardiography.* Assen, The Netherlands: Van Gorcum & Co., 1984:76-103.
8. Von Rueden KT, Turner M. Advances in continuous, noninvasive hemodynamic surveillance. *Critical Care Nursing Clinics of North America* 1999;11(1):63-75.
9. Wang X, et al. Time frequency distribution technique in biological signal processing. *Biomedical Instrumentation and Technology* 1995;29:203-12.
10. Lamberts R, et al. *Impedance Cardiography.* Assen, The Netherlands: Van Gorcum & Co., 1984:29-30.
11. Enger EL. Pulmonary artery wedge pressure: When it's valid, when it's not. *Critical Care Nursing Clinics of North America* 1989; 1(3):603-18.
12. Brandstetter RD, et al. Swan-Ganz catheter: Misconceptions, pitfalls, and incomplete user knowledge—an identified trilogy in need of correction. *Heart & Lung* 1998;27:218-22.
13. Boudoulas H. Systolic time intervals. *European Heart Journal* 1990;11(Suppl I):93-104.
14. Ramos MU. An abnormal early diastolic impedance waveform: A predictor of poor prognosis in the cardiac patient? *American Heart Journal* 1977;94(3):274-81.
15. Schieken RM, et al. Effect of mitral valvular regurgitation on transthoracic impedance cardiogram. *British Heart Journal* 1981;45:166-72.
16. Hubbard WN, Fish DR, McBrien DJ. The use of impedance cardiography in heart failure. *International Journal of Cardiology* 1986; 12:71-9.
17. Schieken RM, et al. Effect of aortic valvular regurgitation upon the impedance cardiogram. *British Heart Journal* 1978;40:958-63.
18. Wang X, et al. Hemodynamic monitoring by impedance cardiography with an improved signal processing technique. *IEEE* 1993; 15:699-700.
19. Von Rueden K, Dunham C. Evaluation and management of oxygen delivery and consumption in MODS. In Secor V, ed. *Multiple Organ Dysfunction and Failure.* St. Louis, Mo.: Mosby-Year Book, Inc., 1996;384-401.
20. Lipman J. Pitfalls in the interpretation of pulmonary capillary wedge pressure. *South African Medical Journal* 1985;67(5):174-7.
21. Calvin JE, Driedger AA, Sibbald WJ. Does the pulmonary capillary wedge pressure predict left ventricular preload in critically ill patients? *Critical Care Medicine* 1981;9(6):437-43.
22. Hansen RM, et al. Poor correlation between pulmonary arterial wedge pressure and left ventricular end-diastolic volume after coronary artery bypass graft surgery. *Anesthesiology* 1986;64(6):764-70.
23. Tuman KJ, Gilbert CC, Ivankovich AD. Pitfalls in interpretation of pulmonary artery catheter data. *Journal of Cardiothoracic Anesthesia* 1989;3(5):625-41.
24. Albert N. Heart failure: The physiologic basis for current therapeutic concept. *Critical Care Nurse* 1999;19(Suppl June):1-15.

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