Articles

Invasive and Noninvasive Hemodynamic Monitoring of Patients With Cerebrovascular Accidents

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Seventeen patients with hemodynamic instability from acute cerebrovascular accidents were evaluated shortly after arrival at the emergency department of a university-run county hospital with both invasive Swan-Ganz pulmonary artery catheter placement and a new, noninvasive, thoracic electrical bioimpedance device. Values were recorded and temporal patterns of survivors and nonsurvivors were described. Cardiac indices obtained simultaneously by the 2 techniques were compared. Of the 17 patients, 11 (65%) died. Survivors had higher values than nonsurvivors for mean arterial pressure, cardiac index, and oxygen saturation, delivery, and consumption at comparable times. Cardiac index values, as measured by invasive and noninvasive methods, were correlated. We concluded that hemodynamic monitoring in an acute care setting may recognize temporal circulatory patterns associated with outcome. Noninvasive electrical bioimpedance technology offers a new method for early hemodynamic evaluation. Further research in this area is warranted.

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Hemodynamics after stroke usually are initially evaluated by noting the blood pressure, skin color, pulse rate, temperature, respiratory rate, and mental status. Subsequent circulatory monitoring is often limited to blood pressure and heart rate recording, plus occasional arterial blood gas measurements and determinations of hematocrits and urinary output rates.

The temporal progression of the clinical status of central nervous system disorders is routinely assessed by evaluating neurologic signs and symptoms and by radiography, computed tomography, and magnetic resonance imaging. Progression of an anatomic lesion of the brain should be evaluated separately from the associated circulatory deficiencies, however, because low-flow shock and increased intracerebral pressure may limit cerebral blood flow, oxygenation, and metabolism. Except in extreme conditions, the blood pressure and the heart rate are not well correlated with blood flow in acute clinical situations[1]; the routine criteria for the early recognition of shock are subjective and imprecisely reflect underlying physiologic problems.[2]

The measurement of hemodynamic variables by invasive pulmonary artery balloon-tipped thermodilution (Swan-Ganz) catheters has been the criterion standard for evaluating circulatory function in critically ill patients. Although this technology is expensive, time-consuming, and personnel-intensive, it provides maximal hemodynamic monitoring that can be used at the bedsides of comatose patients in an emergency department (ED) shortly after admission. Substantial cost, complication rates, and logistic problems, however, prevent its being routinely used in the acute care setting.

Cardiac output monitoring by an electrical bioimpedance technique is emerging as a new method of monitoring hemodynamics that can be used anywhere in a hospital.[3] Its ease of application, combined with continuous on-line displays of data, may greatly affect hemodynamic monitoring. Handelsman extensively reviewed the noninvasive electrical bioimpedance methods for measuring cardiac output and concluded that they relate changes in thoracic electrical conductivity to changes in aortic blood volume and blood flow,[4] but their application to specific disease categories has not been confirmed. Recently, a new thoracic electrical bioimpedance device was described by Wang and colleagues at Drexel University, Philadelphia, Pennsylvania, and

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ABBREVIATIONS USED IN TEXT

CI = cardiac index
ECG = electrocardiographic
ED = emergency department
$PtcO_2$ = partial pressure of transcutaneous oxygen
SaO_2 = oxygen saturation, arterial
VO_2 = oxygen consumption

commercially developed by Renaissance Technologies (Newtown, Pennsylvania).[5,6] The new system included technology from the defense industries, state-of-the-art hardware that provides more stable signals, and redesigned software that improves data analysis, processing, and management.[7]

In the present study, we evaluated the accuracy and reliability of this new technology compared with the standard thermodilution method for measuring cardiac output in critically ill stroke patients shortly after admission to an ED. We also evaluated the feasibility of multiple noninvasive monitoring systems to assess the three components of circulation: cardiac function, pulmonary function, and tissue perfusion.

Patients and Methods

Patients

We studied a consecutive series of 17 patients with hemodynamic instability from acute cerebrovascular accidents using invasive and noninvasive physiologic monitoring systems shortly after the patients were admitted to the ED. There were 13 men and 4 women; 6 patients survived. The patients' mean (±SD) age was 61.5 + 18.5 years. All patients had coma with evidence of acute circulatory failure or shock. The final diagnosis in all patients was acute thrombotic cerebrovascular accident. The study was approved by the hospital Institutional Review Board. Informed consent was obtained for all invasive monitoring by pulmonary artery catheter.

Invasive Hemodynamic and Oxygen Transport Measurements

Hemodynamic monitoring was performed using a radial artery catheter and a percutaneously placed 7F flow-directed pulmonary artery catheter. All measurements were made during relatively stable periods when a patient did not exhibit anxiety, restlessness, or agitation. Cardiac output and cardiac index were calculated by the thermodilution technique (cardiac output computer, Model 9520A, Baxter Edwards Laboratories, Santa Ana, California). Three to five measurements were taken and the results averaged. Intravascular pressures were calibrated against a mercury sphygmomanometer and were measured using the midthoracic level as the zero reference point. The pressure values at the end of more than three expirations were averaged. At the time of cardiac output determination, arterial and mixed venous blood specimens were obtained anaerobically in heparinized syringes, stored in iced water, and promptly analyzed for PO_2, PCO_2, and pH and arterial (SaO_2) and mixed venous oxygen saturation values. Arterial and mixed venous oxygen contents were calculated from the saturation and hemoglobin values, with corrections made for dissolved oxygen in the plasma. Cardiac index (CI), oxygen delivery, oxygen consumption (VO_2), and other derived variables were calculated by standard formulas. Each complete data set was taken within a two-minute period and keyed to the time of cardiac output measurement. All flow variables and flow-derived variables were indexed to body surface area.

Thoracic Electrical Bioimpedance Measurement of Cardiac Index

We used a prototype model of a new thoracic electrical bioimpedance device developed for clinical use from earlier research.[5–7] An array of 11 noninvasive, disposable, prewired hydrogen electrodes, designed to reduce electrical polarization time, were positioned on the skin: two injecting electrodes were placed on the side of the neck at the junction with the shoulders, and two other injecting electrodes were placed at the lateral aspect of the lower thorax at the level of the xiphisternal junction. Four sensing electrodes were placed 5 cm inside the area defined by the injecting electrodes. Three electrocardiographic (ECG) leads were placed across the precordium and left shoulder.

A 100-kHz, 4-mA alternating current was propagated through a patient's thorax by the paired outer electrodes, and the voltage was sensed by the paired inner electrodes to record the ECG, the baseline impedance (Z_0), the impedance waveform for each heartbeat (dZ), and the first derivative of the impedance waveform (dZ/dt). The filters for signal processing used an all-integer-coefficient filtering technology that allows fast signal processing using less computational power. The digital signal-processing technique uses time-frequency distribution (a modified Wigner distribution) with a high signal-to-noise ratio.[5] The digital signal processing identifies and measures the following events on the impedance tracing: the beginning of systole, the opening of the aortic valve, the maximum ventricular contraction (dZ/dt_{max}), closing of the aortic valve, and mitral valve opening. The ECG signal is also evaluated by a state-of-the-art digital signal-processing technique for detecting the standard R-R spikes and Q, S and T waves, which then are used to reconstruct the QRST signal. The maximum value of the dZ/dt signal during the cardiac cycle, dZ/dt_{max} was calculated and displayed. A touch-screen interface displayed the real-time waveforms and calculated values; stroke volume, heart rate, cardiac output, and cardiac index were routinely calculated and displayed.[3–8]

Pulse Oximetry

A standard pulse oximeter was attached to the left forefinger, and after a short warm-up period, values were recorded at frequent intervals. At appropriate times dur-

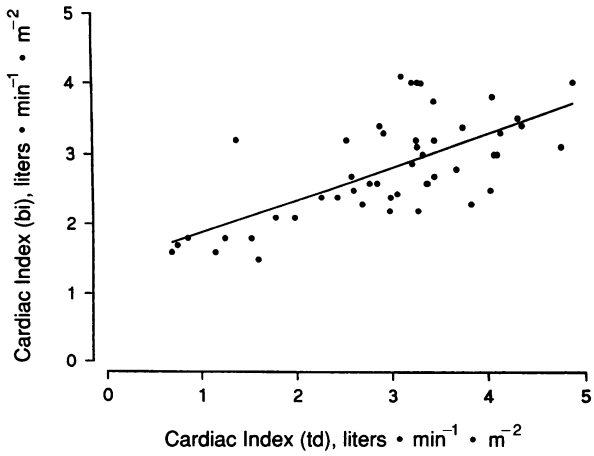

Figure 1.—Cardiac index values of bioimpedance (bi) are plotted against their simultaneously measured thermodilution (td) values.

ing the observation period, an arterial blood gas measurement was taken to compare the standard SaO_2 values with those obtained by pulse oximetry.

Transcutaneous Oxygen Tension

After calibration, a transcutaneous oxygen tension ($PtcO_2$) sensor, heated to 44°C to overcome local vasoconstriction, was placed on the left or right shoulder anteriorly. This sensor uses the same Clark polarographic electrode as is used in the standard blood-gas analyzer; it has been shown to reflect tissue oxygenation of a segment of heated skin.[9,10] A period of 15 to 20 minutes was allowed for equilibration. The sensor was moved every four hours to avoid skin irritation and burn.

Protocol

Shortly after admission to the ED, the patients were evaluated for circulatory instability, that is, hypotension, tachycardia, altered mental status, cold wet skin, and pallor. As soon as circulatory problems were recognized or suspected, the bioimpedance electrodes, pulse oximeter, and $PtcO_2$ sensors were attached, and measurements of each were observed and recorded. Pulmonary artery catheters were placed when indicated for clinical reasons, and corresponding estimations were observed, recorded, and subsequently analyzed.

Primary fluids such as albumin or hetastarch were administered for the resuscitation of circulatory deficiencies; colloids were given to expand the intravascular space without inappropriately expanding interstitial fluid.[11] On occasion, inotropes were used, when it was appropriate to avoid fluid overload. Indications for therapy were a mean arterial pressure of less than 80 mm of mercury, a cardiac index of less than 3 liters per minute per meter squared, a $PtcO_2$ of less than 60 mm of mercury with the patient breathing room air, oxygen delivery of less than 450 ml per minute per meter squared, and oxygen consumption of less than 120 ml per minute per meter squared. The therapeutic goal was to optimize the cardiac function, pulmonary function, and tissue perfusion.

Measurements were made at frequent intervals during periods of resuscitation and changes of clinical findings. After the initial resuscitation, measurements were made when the patients were resting, free of signs of anxiety, and in relatively stable clinical condition. Measurements were made before the initiation of therapeutic interventions and at various periods throughout a patient's course.

Statistical Analysis

Data from variables collected sequentially were compared using a one-way analysis of variance and the Newman-Keuls test. Linear regression analysis was used to compare the two methods of measuring cardiac output. Statistical comparisons between data obtained under different conditions were evaluated using the two-tailed Student's t test for paired distributions. Differences were considered significant at probability values of less than .05.

Results

Comparison of Cardiac Index Estimated by Thermodilution and Bioimpedance

Figure 1 compares 50 paired values of simultaneously measured cardiac index by invasive thermodilution catheters with that obtained by the noninvasive bioimpedance system. Cardiac index values varied from 2 to 5 liters per minute per meter squared. The regression equation was $Y = 0.47x + 14$ ($r^2 = 0.68$; $P < .001$). This was not as close a correlation as for a larger series of patients in an intensive care unit[8] but was considered adequate for initial physiologic analysis in the ED.

Temporal Hemodynamic and Oxygen Transport Patterns After Stroke

The temporal patterns of hemodynamic and oxygen transport events are summarized in Table 1 beginning at the time of admission to the ED. Figure 2 compares the temporal patterns of survivors and nonsurvivors for mean arterial pressure, heart rate, cardiac index, $PtcO_2$, pulse oximetry (SaO_2), oxygen delivery, oxygen consumption (VO_2), and pulmonary venous admixture. In general, the survivors had higher values for mean arterial pressure, cardiac index, $PtcO_2$, SaO_2, oxygen delivery, and VO_2 than the nonsurvivors at comparable times after admission. The heart rate and pulmonary venous mixture were higher in those who died.

Hemodynamic Patterns Before and After Nadirs

Because the lowest value (nadir) of the various hemodynamic variables occurred at different times, the temporal patterns may be out of phase and indistinct. Figure 3 shows the survivors' and nonsurvivors' temporal patterns for pulmonary arterial wedge pressure, mean arterial pressure, cardiac index, $PtcO_2$, VO_2, and oxygen delivery when the data of each variable are aligned in

TABLE 1.—*Temporal Hemodynamic and Oxygen Transport Patterns in 17 Patients With Stroke**

Variable	Hours After Admission 0–4	4–8	8–12	12–16	16–24	24–48
CItd, liters·min^{-1}·m^{-2}	2.69±0.80	2.64±0.72	3.13±0.93	4.19±2.34	3.45±1.19	2.90±1.19
MAP, mm of mercury	77±31	90±7	98±19	85±16	85±26	90±30
PAOP, mm of mercury	10±3	9±1	11±4	7±2	11±6	14±5
CVP, mm of mercury	4±2	4±3	5±0	4±1	9±6	13±6
Heart rate, bpm	95±30	111±37	93±8	103±22	116±19	104±24
PO_2, %	95±5	98±4	99±1	98±1	91±10	95±6
$PtcO_2$, mm of mercury	111±89	98±29	88±36	97±19	97±26	74±58
$PtcCO_2$, mm of mercury	58±13	43±7	42±9	45±9	52±11	48±12
PaO_2, torr	147±105	207±47	210±34	154±50	96±49	113±59
O_2 delivery, ml·min^{-1}·m^{-2}	355±107	367±56	470±19	528±248	466±161	389±188
$\dot{V}O_2$, ml·min^{-1}·m^{-2}	145±26	123±14	87±28	77±2	146±54	122±57

CItd = cardiac index (thermodilution), MAP = mean arterial pressure, PAOP = pulmonary artery occlusion pressure, CVP = central venous pressure, bpm = beats per minute, PO_2 = partial arterial pressure of oxygen, $PtcO_2$ = partial pressure of transcutaneous oxygen, $PtcCO_2$ = partial pressure of transcutaneous carbon dioxide, O_2 = oxygen, $\dot{V}O_2$ = oxygen consumption

*Values are given as the mean±SD.

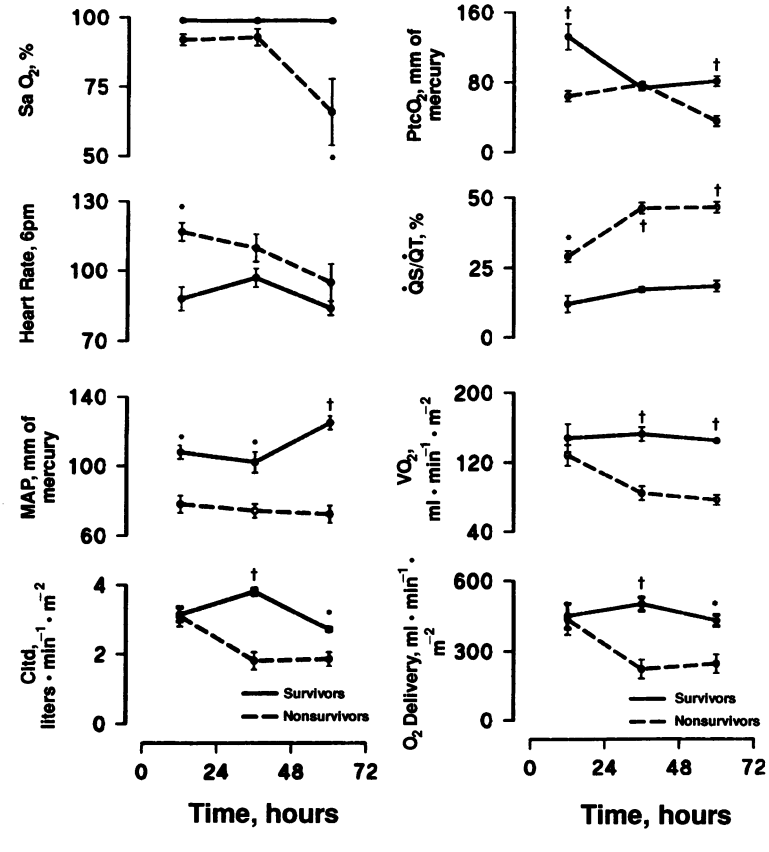

Figure 2.—Arterial hemoglobin oxygen saturation (SaO2), heart rate, mean arterial pressure (MAP), cardiac index by thermodilution (CItd), transcutaneous oxygen tension (PtcO2), pulmonary venous admixture or shunt (QS/QT), oxygen consumption (VO2), and oxygen (O2)delivery are shown in survivors and nonsurvivors during the first, second, and third days after admission to the emergency department. Note the nonsurvivors' significant decrease in SaO2, blood pressure, CItd, PtcO2, O2 delivery, and VO2 values ($P < .05$, indicated by the asterisk) and the significant rise in the QS/QT value ($P < .01$, indicated by the dagger). bpm = beats per minute

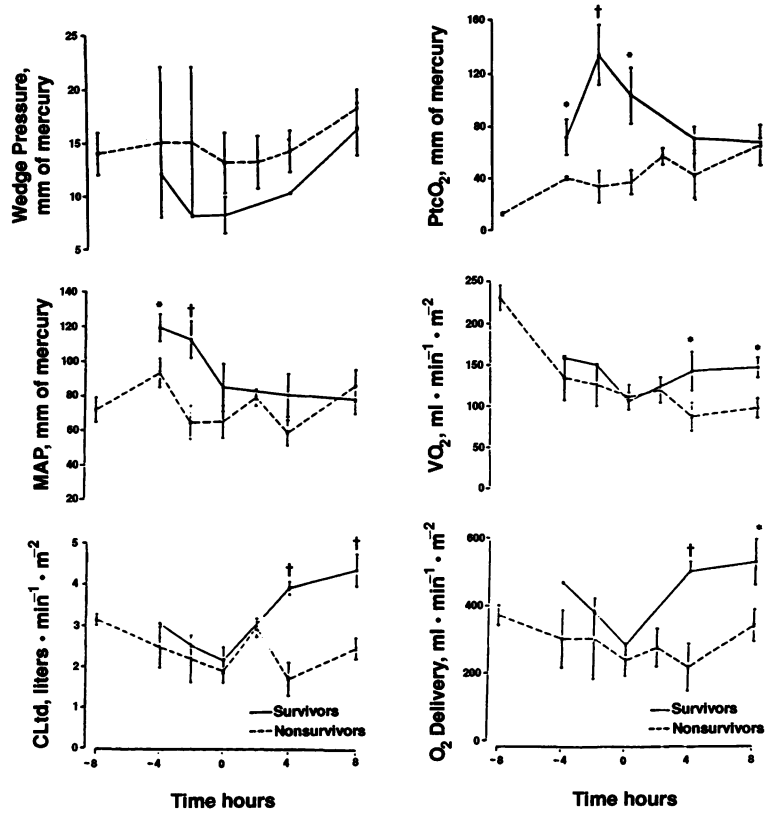

Figure 3.—Survivor and nonsurvivor patterns are shown immediately before and after the cardiac index nadir, as measured by thermodilution (CItd). Note the significant higher transcutaneous oxygen tension (PtcO2) before the nadir and the higher CItd, oxygen consumption (VO2), and oxygen (O2) delivery after the nadir (both P < .01, indicated by the dagger). MAP = mean arterial pressure, asterisk = P < .05

time before and after the cardiac index nadir. The survivors' values were generally higher in all monitored variables except the pulmonary wedge pressure.

Invasive Monitoring of Cardiac Index, Oxygen Delivery, and VO_2

Table 2 summarizes the cardiac index, oxygen delivery, and VO_2 index for survivors and nonsurvivors of stroke obtained by invasive pulmonary artery catheter placement shortly after admission to the ED. In general, the survivors had higher values in these variables.

Discussion

The primary injury that occurs in the brain tissue of patients with acute stroke is usually irreversible, but the associated circulatory dysfunction may contribute to further deterioration of cerebral function.[12] The traditional methods of monitoring, such as recording the arterial blood pressure, heart rate, and respiratory rate, are not well correlated with blood flow, SaO_2, or tissue perfusion as reflected by the cardiac index, pulse oximetry, or transcutaneous oximetry, respectively.[13] This emphasizes the inadequacy of the vital signs to reflect the more important circulatory functions that include blood flow, arterial oxygenation, and tissue perfusion. The pulmonary artery catheter is considered to be the state-of-the-art tool for precise hemodynamic monitoring. Its placement and use are personnel-intensive, costly, and time-consuming, however, and are associated with complications and logistic problems. The data are retrieved in an operator-dependent and discontinuous manner.

TABLE 2.—*Cardiac Index by Thermodilution (Citd), Oxygen Delivery, Oxygen Consumption (VO_2), and Transcutaneous Oximetry ($PtcO_2$) Values Shortly After Hospital Admission in Survivors Compared With Nonsurvivors*[*]

	Hemodynamics			
Patients	*CItd, liters·min⁻¹·m⁻²*	*$PtcO_2$, torr*	*O_2 Delivery, ml·min⁻¹·m⁻²*	*VO_2, mlmin⁻¹·m⁻²*
Survivors	3.44±0.82	97±58	488±127	146±38
Nonsurvivors	2.64±1.33	61±55	336±181	105±47

[*]Values are given as the mean±SD.

The availability of a new thoracic electrical bioimpedance system allows the monitoring of cardiac output in a noninvasive, continuous on-line, real-time mode.[5–8] The electrodes can be applied immediately on admission to the ED almost as easily as ECG electrodes. Monitoring can be started as soon as a patient is admitted to the ED. Several limitations with this new bioimpedance device include low signal:noise ratios from pleural effusions, chest tubes, pulmonary edema, severe congestive heart failure, and severe pneumonia.[3]

In this series, the correlation of the bioimpedance method with the thermodilution method was somewhat less accurate than in a larger series of critically ill patients, previously reported.[8] In the face, however, of exigencies surrounding the emergency admission of patients with stroke and neurologic failure, the comparison was considered satisfactory. Far more important than isolated values are trends. By a method that offers continuous hemodynamic monitoring, the temporary patterns of circulatory insufficiency can be better understood, and therapy can be started promptly.

This study represents a new approach to the hemodynamic evaluation of patients with stroke on admission to the ED. Both invasive and noninvasive data show reduced cardiac output and poor tissue perfusion at the early stages after acute stroke. Survivors demonstrated better hemodynamic variables than nonsurvivors early after admission. Whether this is the determinant or just a marker of the final outcome remains to be studied. Although optimal supranormal values have been shown to increase survival in certain groups,[14,15] the applicability of these values in stroke patients was not examined in the present series. It would be reasonable to assume, however, that identifying and correcting circulatory deficits early may result in an improved outcome.

Conclusion

Noninvasive monitoring in the immediate postadmission period allows the temporal circulatory patterns in patients with stroke and neurologic failure to be described. Data obtained by invasive and noninvasive methods show an early reduction in hemodynamic and oxygen transport variables, particularly in the nonsurvivors. In general, the survivors' mean arterial pressure, cardiac index, $PtcO_2$, oxygen delivery, and VO_2 patterns are higher than those of the patients who die after stroke. Further investigation of noninvasive methods for monitoring hemodynamic and oxygen transport data is warranted.

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