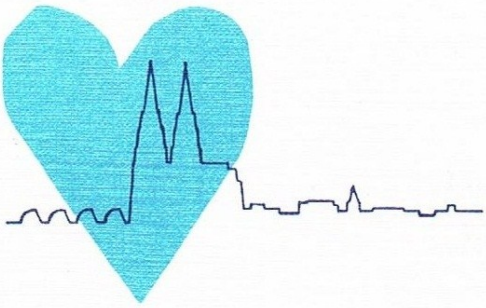
Thoracic Impedance Measurements in Clinical Cardiology

International Symposium, Cologne

Edited by U. J. Winter, R. K. Klocke, W. G. Kubicek and W. Niederlag



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Preface

Impedance measurement technology is now widely applied in medical research and clinical medicine. Despite the generally used term "impedance," the methods are quite different in the various application fields. Although all these methods are based on how the electrical impedance of tissues or vessels is influenced by electrolyte, fluid, or blood changes, special considerations have to be taken into account for every special organ or organ system. Many colleagues are talking and writing about impedance measurements in their fields of medicine, but they mean completely different things and often forget that the roots lie in the theoretical impedance research.

Thus, we found it necessary to bring together the "fathers" of impedance technology and some of the "daughters" and "sons" working in clinical medicine in an international meeting of experts („International Symposium on Thoracic Impedance Measurements in Clinical Cardiology"), which was held from March 13th to 14th, 1991 in Cologne. Many of the contributions are collected in this book, which tries to give a certain overview of what's going on in impedance measurements in cardiovascular medicine.

We hope that the book will be warmly accepted by the medical world.

U. J. Winter
for the editors
Cologne, Spring 1994

Bioimpedance Technology in Noninvasive Hemodynamic Management: The CDDP(II) System

R. K. Klocke and I. B. Reuver

Summary

The NCCOM 3-R7/CDDP system (BoMed Medical Manufacturing), consisting of the noninvasive continuous cardiac output monitor (NCCOM 3-R7) and the cardiodynamic data processing (CDDP) software, a computer program that simplifies data collection and data processing through bioimpedance analysis, provides a risk-free method of continuous hemodynamic monitoring. The easy-to-read graphic display of individual hemodynamic parameters in relation to reference standards allows for specific therapeutic intervention with continuous monitoring of the response of the cardiovascular system. Thus, acute pharmacologic effects can be recognized before the changes become clinically apparent, and individual responses to a particular therapy can be assessed. The validity and reproducibility of the method are well established in selected cases.

Introduction

The desire for noninvasive methods for the routine clinical assessment of hemodynamic parameters has prompted modifications of the original bioimpedance technique of Atzler and Lehmann [2], including an improved stroke volume equation introduced by Kubicek et al. [8–10] in the Minnesota impedance cardiograph.

Sramek [14] further refined the computation by taking into account individual patient parameters [3, 14]. He replaced band electrodes with conventional Ag/AgCl adhesive electrodes like those used for ECG ranging recordings. Sramek assumes that the basal impedance remains constant at hematocrits from 26% to 66%, so he does not adjust the data for individual hematocrit values.

To simplify data collection and processing, Sramek et al. developed a menu-driven, user-friendly computer program called the cardiodynamic data processing system (CDDP). This software provides as easy-to-read graphic display of individual parameters juxtaposed to reference standards indicating whether the quantity is below, within, or above normal limits. This allows for rapid orientation and the continuous, noninvasive monitoring of hemodynamic parameters.

The NCCOM 3-R7 Monitor

The noninvasive continuous cardiac output monitor (NCCOM 3-R7, BoMed Medical Manufacturing) measures the impedance change of a high-frequency alternating current (2.5 mA, f = 70 kHz) applied across the thoracic region (Fig. **1**) [15, 17, 18]. The pulsatile blood flow produces a maximum impedance change in systole $(dZ/dt)_{max}$, which is divided by the basal thoracic impedance (thoracic fluid index TFI) to obtain the index of contractility (IC):

$$IC = (dZ/dt)_{max}/TFI\ (s^{-1}).$$

This index is directly proportional to the peak flow (PF) during left ventricular ejection and, as such, is dependent on the preload and afterload. The peak flow itself is given in ml/s.

If we consider only the acceleration of the blood flow with the left ventricular pulse during the first 10–20 ms after opening of the aortic valve, this quantity is less dependent on preload and afterload and is represented by the acceleration index (ACI) [14]:

$$ACI = d^2Z/dt^2/TFI\ (s^{-2}).$$

Thus, IC, PF, and ACI are **contractility parameters** that characterize the inotropic status of the left ventricle.

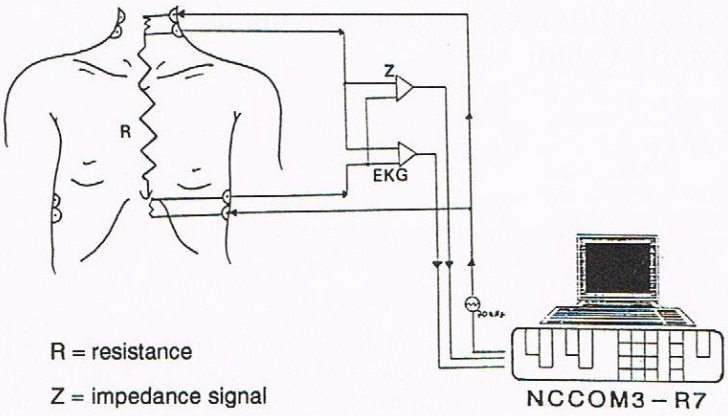


Fig. **1** Electrode placement and equipment setup.

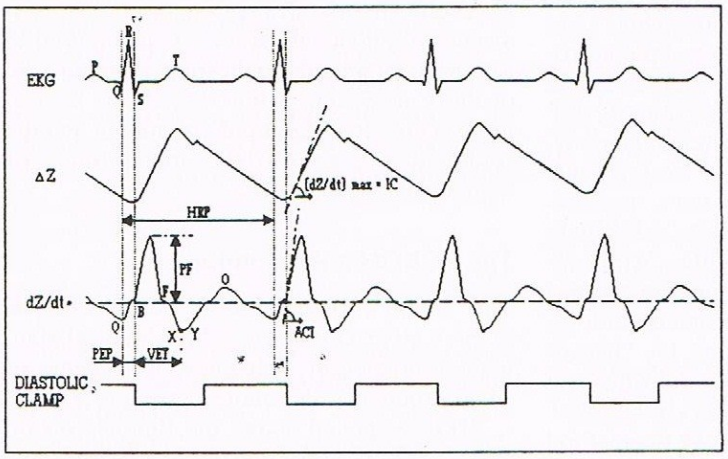


Fig. **2** Temporal correlation of EKG, the impedance signal dZ, and the first derivative dZ/dt. IC = Index of contractility, dZ = impedance change, $(dZ/dt)_{max}$ = maximum impedance change over time, B = opening of aortic valve, X = closure of aortic valve, PF = peak flow, ACI = acceleration index, PEP = pre-ejection period, VET = ventricular ejection time, HRP = cardiac cycle.

Another important parameter for describing left ventricular performance is the stroke index, i. e., the stroke volume (SV) relative to body surface area. Sramek calculates SV from the IC, the volume of electrically participating tissue (VEPT, dependent on sex, weight, and size), and the ventricular ejection time (VET = duration of mechanical systole), which is calculated from the dZ/dt signal (Fig. **2**):

$$SV = VEPT \cdot VET \cdot IC \text{ (ml)}.$$

The cardiac output (CO) can now be calculated from the stroke volume and heart rate (HR, obtained from the synchronous EKG trace) using the formula:

$$CO = (SV \cdot HR)/1000 \text{ (l/min)}.$$

The ejection fraction (EF) is calculated from the systolic time interval (STR), which represents the ratio of the pre-ejection period (PEP) to the ventricular ejection time (VET):

$$STR = PEP/VET \text{ (s)}.$$

PEP is defined as the interval between Q (from the QRS complex of the EKG) and the opening of the aortic valve (from the dZ/dt signal). VET is the interval between the opening and closing of the aortic valve.

The NCCOM 3-R7 determines the EF using an algorithm developed by Capan et al. The limits of an EF determination over systolic time intervals are affected by changes in PEP and VET. **In a patient with a left bundle branch block, for example, a lengthening of PEP with a constant VET leads to an underestimation of EF. Adrenergic influences in an acute myocardial infarction lead to a shortening of both PEP and VET.** Their relationship remains unchanged, but the **EF value is overestimated**.

To eliminate the effect of heart rate on VET, the latter is divided by period of one cardiac cycle (HRP) to yield the "ejection ratio" (ER), whose value is chiefly dependent on a change in the preload.

$$ER = VET/HRT \cdot 100\ (\%)$$

The end-diastolic volume (EDV), as a measure of preload, is determined from the ratio of the stroke volume to the ejection fraction:

$$EDV = SV/EF \cdot 100\ (ml).$$

As a general rule, index values are referred to body surface area so that interindividual comparisons can be made. To suppress artifacts, the diastolic component is damped during processing of the dZ/dt signal by the microprocessor, and only the systolic component is evaluated **("diastolic clamp")**. The undamped curve can be simultaneously recorded at any time. Six cardiodynamic parameters (PF, EF, EDV, CO, HR, SV) one of a selection of six additional values (IC, ACI, TFI, VET, ER, STR), and four derivative values (PFI, EDI, CI, SI) can be simultaneously generated by the NCCOM 3-R7. These values can be displayed after every beat (beat-to-beat mode) or shown as an average over 16 beats. Connected to a printer, the computer can produce a direct printout of six parameters in the beat-to-beat mode or 11 parameters in the mean-value mode. An additional long-term mode can be selected to generate a printout at 5-min intervals.

Validity of the Bioimpedance Method

A number of publications dealing with the question of validity indicate high reliability of the data obtained by impedance cardiography [1, 5, 11, 12]. Our own studies [6] confirm this **reliability for cardiac output data** (CO/CI). A comparison of the measured cardiac index values with values obtained by the Fick method showed a good correlation within the limits imposed by biologic methods of measurement: $n=109$, $r=0.82$, $p \leq 0.001$. **Comparison with the thermodilution method** in intensive care patients also demonstrated a **high**, clinically useful **correlation**: $n=28$, $r=0.90$, $p \leq 0.001$. In interpreting our results, however, it must be considered that we performed our studies in patients with stable cardiovascular function. **Patients with significant cardiac valve defects, valvular incompetence, shunts, or clinically relevant intrathoracic fluid collections, as in pulmonary edema, were not evaluated.**

The CDDP System

Sramek et al. [16, 18] have developed a user-friendly computer program for the clinical evaluation of the extensive data on global flow, cardiac performance, and volume status provided by noninvasive thoracic impedance measurements. This program (CDDP I), available on 5.25-inch or 3.5-inch diskettes, runs on an IBM PC/XT or corresponding IBM-compatible desktop computer. The manu-driven software provides a graphic display of 10 hemodynamic parameters, which can be updated beat-to-beat or every 16 beats by EKG triggering. Immediate on-screen comparison with reference standards permits a rapid assessment of cardiodynamic status, and deviations from the normal range are clearly recognized (Fig. **3**).

The monitor displays the cardiac index (CI) as a measure of global perfusion, the stroke volume index (SI) as an expression of pump performance, and the end-diastolic index (EDI) as a parameter of preload.

The **patient's inotropic status** is characterized by the volume-based index of contractility (IC) and the relatively volume-independent acceleration index (ACI).

The system also displays the ejection fraction (EF) as a measure of pump efficiency and the TFC as a **measure of intrathoracic fluids**. The latter parameter is the reciprocal of TFI and increases with the amount of fluid in the chest.

If desired, the systolic/diastolic or mean arterial blood pressure (MAP) can be entered and used to calculate the afterload, displayed as the systemic vascular resistance index (SVRI), and the left ventricular work index (LCWI). The blood pressure can be measured, for example, by a noninvasive oscillometric sphygmanometer such as the Dinamap (Criticon). The MAP itself is also graphically displayed. The heart rate (HR) and patient data are additionally displayed on the monitor screen.

The user can store all data and retrieve it at any time in numeric or graphic form. If neces-

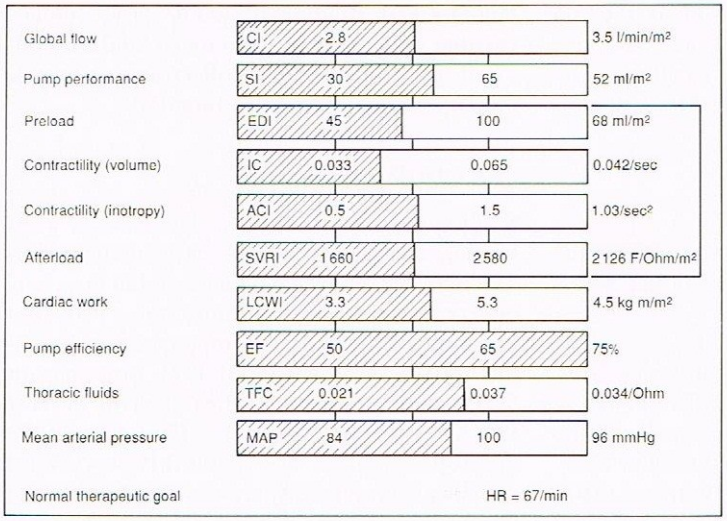


Fig. **3** Bar graph display indicating absolute values in appropriate units as compared with reference standards.

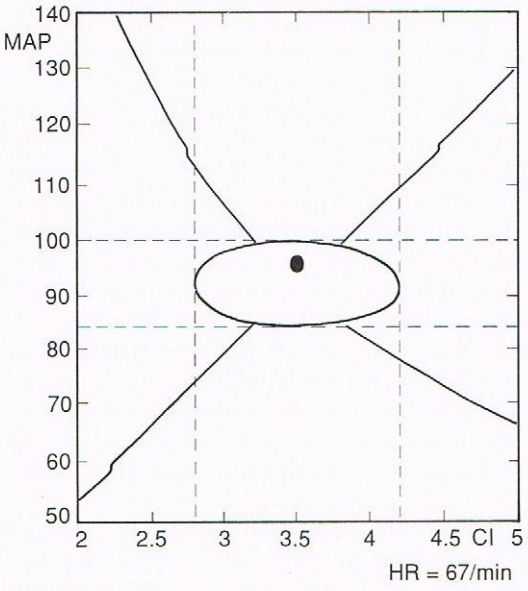


Fig. **4** Therapeutic diagram. Bipolar coordinate graph with a central ellipse indicating the therapeutic goal, a straight diagonal representing a normal vascular status, and a hyperbola representing normovolemia and/or normoinotropy. Corresponding absolute values are also indicated.

sary, a continuous, operator-independent data record can be obtained by having 11 parameters printed out in the mean-value or long-term mode concurrently with the CDDP program.

The operator can switch from the foregoing *diagnostic program* to the *therapeutic program* by pressing a function key. The screen then displays a bipolar coordinate graph with the mean arterial pressure as the ordinate and the cardiac index as the abscissa. An elliptical area at the center defines the normal range of CI = 2.8–4.2 (l/min/m²) and MAP = 80–100 (mm Hg) (Fig. **4**). A diagonal line from lower left to upper right represents the line of normal vascular status with SVRI = 2030 F.Ohm/m². A hyperbolic line from upper left to lower right represents the line of normovolemia and/or normoinotropy with LCWI = 4.35 kgm/m².

Deviations from these normal lines are indicated as a relative percentage of vasoconstriction/vasodilatation, hyper-/hypovolemia, and/or hyper-/hypoinotropy. Points inside the ellipse characterize **the normoinotropic, normovolemic, and normodynamic state** and thus represent the therapeutic goal. This normal range is defined for adults.

For postoperative monitoring, Shoemaker et al. [13] defined a modified therapeutic goal corresponding to the increased postsurgical perfusion demand, and this mode can be selected with a function key. The current values of CI, SVRI, MAP, and HR are indicated on the left side of the screen, and the corresponding point is plotted and displayed on the graph.

An example will serve to illustrate the application of the therapeutic program:

A patient with a **low LCWI** may be **hypovolemic and/or hypoinotropic**. This differentiation is made in reference to the IC and ACI. A low IC with a normal ACI is suggestive of hypovolemia, while a low ACI indicates hypoinotropy. Similar considerations apply to increased LCWI values. If the vascular resistance (SVRI) remains constant while the LCWI is changed (e. g., by a positive inotropic agent), the point in the coordinate system will move along the SVRI line (Fig. **5**). Conversely, a change in the vasoactive status (e. g., caused by a vasodilator) while inotropy and volume remain unchanged (constant LCWI) causes the point to shift along the hyperbola (Fig. **6**).

Figure **7** illustrates the findings of a patient in frank cardiac failure with a CI of 2.3 l/min/m² and a MAP of 83 mm Hg. The **low ACI**, signifying **deficient contractility**, combined with the **low LCWI** indicates a hypoinotropic status. The high SVRI indicates a significantly increased afterload. The most favorable therapeutic effect would be achieved by increasing the contractility, and thus the cardiac output, while reducing the afterload. Milrinone, a phosphodiesterase inhibitor with positive inotropic activity, produces both effects. The resultant vector produced by both pharmacologic actions moves the point in the coordinate system into the ellipse and thus into the range of our therapeutic goal (Figs. **8, 9**).

This case illustrates the practical capabilities of the therapeutic program for planning individual therapy and monitoring patient response continuously and noninvasively.

One problem that we noticed was a slight delay in data transfer from the NCCOM 3-R7 to the CDDP system. As a result, the data were not displayed on the monitor precisely in real time, and the data in both system did not agree in their absolute values. This deficiency was improved in Version II of the CDDP software, which eliminates the delay between the NCCOM and CDDP systems. Additionally, the new version allows for input of the current central venous pressure (CVP) and the pulmonary arterial occlusion pressure (PAOP), which in Version I had present values of CVP = 3 mm Hg and PAOP = 6 mm Hg. These still serve as default values in the new version of

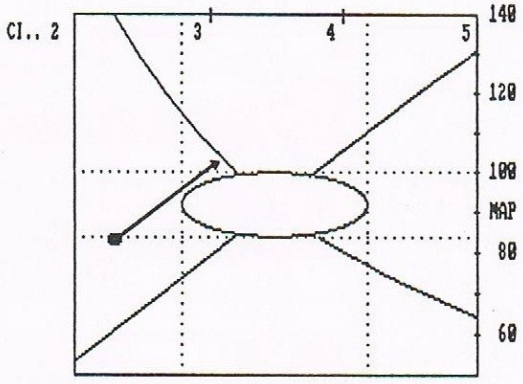


Fig. **5** Changes in the LCWI, caused for example by a positive inotropic agent, associated with a stable vasotonic status (SVRI).

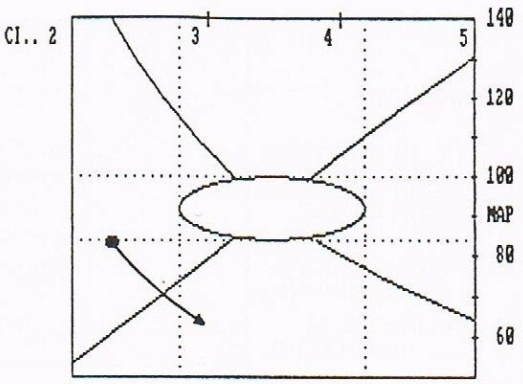


Fig. **6** Changes in the vasoactive status with a stable left ventricular work index (LCWI), caused for example by vasodilatation lowering the SVRI.

pressure data are not entered. The option of entering comments during data storage simplifies data identification and facilitates later review.

Clinical Application

At our center the NCCOM 3-R7/CDDP system is used principally at the intensive care unit, the outpatient cardiology department, and the cardiology ward.

In a study on the hemodynamic monitoring of pharmacologic effects by noninvasive bioimpedance, we were able to document individual response profiles for various vasodilating agents [7]. We found that bioimpedance could demonstrate the early beginning of acute

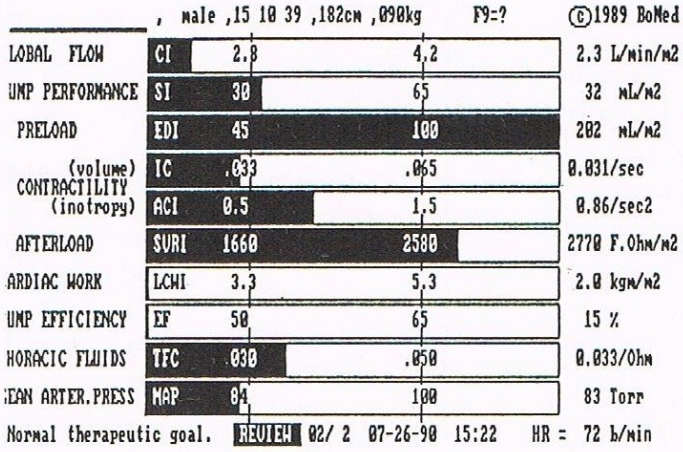
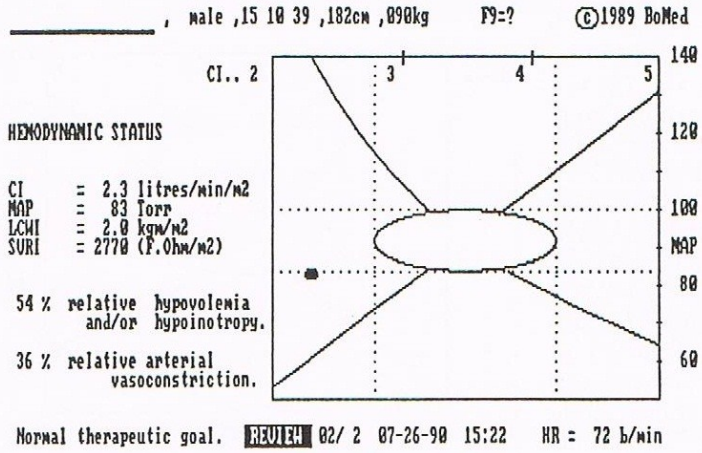


Fig. 7 Patient in frank cardiac failure with a cardiac index of 2.3 l/min/m² and a mean arterial pressure of 83 mmHg.



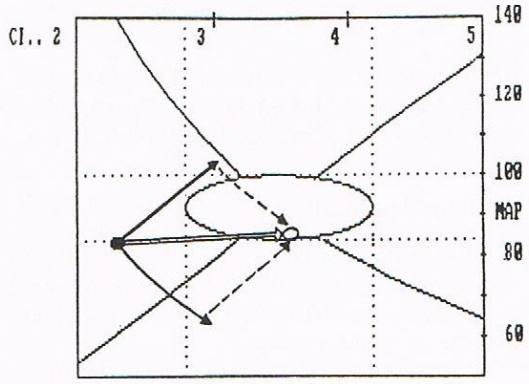


Fig. 8 Response to the positive inotropic and vasodilator actions of the phosphodiesterase inhibitor milrinone.

effects before those changes were clinically apparent and could provide early detection of individual deviations from the anticipated pharmacologic response profiles. This offers a means of testing therapeutic alternatives with respect to their efficiacy in the individual patient.

Conclusions

We believe **that measurement of the cardiac index by transthoracic impedance using the Sramek method** and "beat-to-beat" digital technology provides a determination of this circulatory parameter that is **acceptable for clinical requirements**. It permits a noninvasive assessment of hemodynamic status while taking into account such parameters as contractility, preload, afterload, etc.

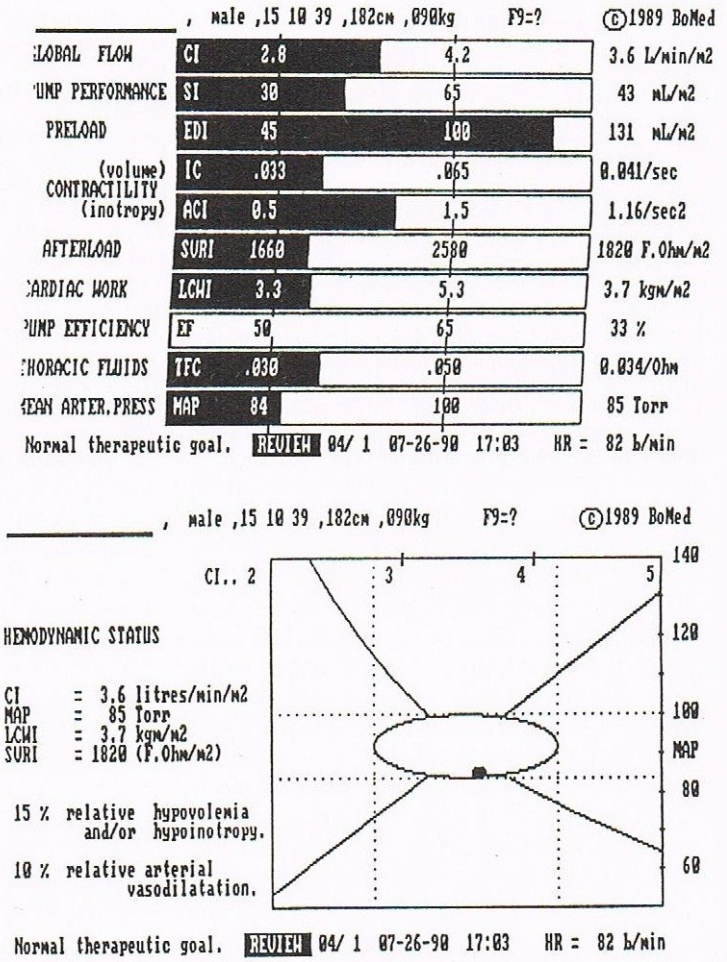


Fig. **9** Status of the patient in Fig. 7 following treatment with milrinone.

With allowance for the limitations noted above–particularly cardiac defects, valvular incompetence, shunts, significant intrathoracic fluid collections, and septic states–bioimpedance offers an important adjunct to more invasive diagnostic procedures.

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