

A Clinician's Guide to Interpreting an Impedance Cardiography (ICG) Hemodynamic Status Report

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This document outlines general steps in using an ICG report obtained from the BioZ to evaluate and manage patients with various cardiovascular conditions. It is not intended to be a comprehensive clinical guide and cannot replace clinical judgment or specific evaluation of an individual patient.

In assessing a patient using hemodynamic information from the BioZ, the clinician may be called upon to:

- formulate clinical question(s) based on the patient's history, symptoms and examination
- verify that demographic information entered into the BioZ such as age, gender, height, weight, are correct
- evaluate the ECG and ICG waveforms for consistency
- compare current findings with previous hemodynamic information, if available
- analyze the various parameters for an assessment of flow, resistance, fluid status and ventricular performance
- interpret the hemodynamic findings, in the context of the clinical setting
- decide on management implications based on all information available using appropriate algorithms
- document interpretation and plan in the medical record

Comments regarding these processes are listed below.

Steps	Comments/Discussion
Verify name, age, height and weight	If height, gender or weight is entered incorrectly (such as wrong units), it will significantly affect results, including BSA and all indexed parameters. BSA is calculated and is typically 1.4 to 2.3.
Review ECG wave form	Look for absence of artifact. The total recording for the ECG is 6 seconds. If the ECG is read properly, the HR at the top of the page should be ten times the number of R-to-R intervals in the ECG tracing. Look for regularity; if grossly irregular, suspect atrial fibrillation, which may affect both cardiac efficiency and the ability to record hemodynamics consistently (especially at higher heart rates).
Review ICG (delta Z) waveform	<p>1) Is there a periodic variation that occurs at the same frequency as the QRS complex in the ECG (these curves can be superimposed)?</p> <p>2) Does the steepest upstroke of the curve begin about where the QRS would fall (superimpose them to be sure)?</p> <p>3) Look at the steepness of the upslope of the curve just after where the QRS complex would fall; this is a gross estimate of the Velocity Index (dZ/dt). If very flat, VI will be low as will SV, CI and CO (remember, $SV=VI*LVET*VEPT$).</p> <p>If the curve is very flat or does not show the expected rhythm with each QRS complex, check lead connections, sensors and consider a test using the simulator. Occasionally, sitting the patient up may improve the amplitude of the wave form, although it does alter hemodynamics by lowering TFC, raising SVR and often lowering CO, SV, etc.</p>
Compare to prior Status Report(s) if available	Although a single ICG test is valid and helpful in patient assessment, conclusions can be drawn with greater confidence when values show significant changes from past tests. Typically, changes of 10-15% or more in a particular parameter from a prior test is significant and reflects greater than the expected variation for CI, STR, TFC, etc. This generalization presumes that both tracings have been performed in a similar fashion and are technically interpretable. Note: Hemodynamic parameters are highly influenced by body position. When comparing serial measurements be sure to verify that the patient was in the same position for both tests.

Individual Parameters

Heart Rate (HR)	Tachycardia may be a response to heart failure, anxiety or other reason for increased catecholamines. Consider a primary arrhythmia, such as atrial fibrillation. Check to make sure the HR is consistent with the ECG tracing.
Blood Pressure (BP) and Mean Arterial Pressure (MAP)	BP/MAP is of great importance in patients with hypertension and any other type of cardiovascular disease. The BP taken by the automatic cuff on the BioZ uses the oscillometric method to measure MAP and then calculate SBP and DBP. This is opposite to standard BP techniques using auscultation where SBP and DBP are measured and MAP calculated. Also note: for the true definition of hypertension: 1) BP should be measured with the patient sitting quietly for 5 minutes with arm supported at heart height and feet flat on the floor (the average of 2 or more measurements are used). 2) In contrast, for the accurate determination of SVR, BP should be measured with the patient <u>supine</u> . Nonetheless, despite variations in findings, the automatic cuff on the BioZ is highly accurate. Moreover, if there are differences between SBP and DBP, you are likely to find that MAP (as estimated by $DBP + \frac{1}{3} * (SBP-DBP)$) are quite close.
Cardiac Index (CI) or Cardiac Output (CO)	This is the best indicator of adequacy of blood flow. It contributes to blood pressure ($MAP = CO \times SVR$) and is decreased in patients with decompensated heart failure. Studies suggest $CI < 2.5$ in a patient with dyspnea suggests cardiac origin. If CI is very low, e.g., below 1.5 L/min/m^2 , suspect a technical problem with the recording and check sensor placement, lead connections and the delta Z waveform carefully.
Stroke Volume (SV) or Stroke Index (SI)	This is CO or CI divided by HR—the amount of blood pumped with each heart beat. Patients with heart failure and depressed left ventricular function may compensate by increasing HR to maintain CO or CI in the normal range. In such patients, SV or SI will be decreased.
Systemic Vascular Resistance (SVR) or Index (SVRI)	These are the best indicators of the degree of vasoconstriction. Elevated levels are the most common hemodynamic findings in uncontrolled hypertension or suboptimal afterload reduction in patients with heart failure.
Thoracic Fluid Content (TFC)	This is simply the reciprocal of total thoracic impedance. Because the thoracic impedance is determined by intravascular and extravascular fluid and by the electrical properties of muscle, bone, fat, and air, this number can be within a relatively wide range. Thus, there is poor correlation with filling pressures or wedge pressure. However, the number is quite reproducible and changes in TFC reflect changes in thoracic fluid volumes quite well. In a given patient, even a 7-10% increase in TFC suggests fluid retention, often due to venous congestion or alveolar fluid, although it can signal a pleural or pericardial effusion.
Systolic Time Ratio (STR)	Calculated as the ratio of pre-ejection period (PEP) to left ventricular ejection time (LVET). With worsening LV function, PEP increases and LVET decreases, resulting in significant increases in STR. An increase in STR to > 0.50 is a fairly reliable marker of cardiac dysfunction. Studies show that changes in STR correlate with changes in ejection fraction; thus an increase in STR by 15-20% may suggest an underlying change in LV function and prompt further evaluation (e.g., an echocardiogram). Note: Conduction abnormalities or pacemakers alone can increase STR.
Acceleration Index (ACI) or Velocity Index (VI)	These indices reflect the acceleration and velocity of blood ejected into the aorta, respectively. They are moderately sensitive and specific for changes in LV function. For example, ACI and VI are reduced when forcefulness of contraction is reduced, such as, with decreased contractility and impaired left ventricular performance in patients with systolic dysfunction due to cardiomyopathy.
Left Cardiac Work (LCW)	LCW is approximately the product of MAP and CO (or CI, in the case of LCW index). Cardiac work reflects more about the oxygen demands of the heart (due to the amount of work performed) than cardiac performance itself. Although useful in theory for predicting level of ischemic threshold, it does not add much beyond CI, SI, VI, ACI, and STR in assessing cardiac performance or contractility.

Clinical Applications

Heart Failure (HF)	<ul style="list-style-type: none">○ When a patient has HF, the most important variables are SVR (reflects adequacy of vasodilation), CI or SI (reflects adequacy of flow/pump function), and change in TFC from previous measure (reflects relative gain or loss of fluid). SI may be more sensitive than CI, as patients with depressed cardiac function may have a compensatory increase in HR.○ Reduced ACI and VI are consistent with reduced left ventricular performance.○ High STR suggests significant cardiac impairment such as might be seen with systolic dysfunction. In patients with known HF, STR < 0.50 suggests preserved ejection fraction, such as when the HF is primarily due to diastolic dysfunction.○ A rise in STR or drop in CI on serial study has been correlated with an interval worsening of cardiac function as might be seen with a decrease in ejection fraction.○ As the right ventricular forward output equals the left ventricular forward output (in the absence of intracardiac shunt or significant aortic or pulmonic insufficiency), CO/CI/SI will be accurate with left ventricular dysfunction alone, right ventricular dysfunction alone or with biventricular dysfunction.○ In the PREDICT study of stable HF patients, higher TFC (>35) with lower SI (<35) identified a high-risk subset with a 7-fold increase in risk of a HF event within the next 2 weeks compared to patients with lower TFC + higher SI.
Hypertension	<ul style="list-style-type: none">○ When a patient has uncontrolled BP or must have a change in medications due to intolerance, the key is to define the hemodynamic mechanism of the BP elevation. As $MAP = CO \times SVR$, an elevated MAP is due to elevated CO, SVR or both.○ Drugs that lower CO include the beta-blockers and calcium channel blockers (CCBs) such as verapamil and diltiazem, as well as diuretics. Drugs that lower SVR include ACE inhibitors, ARBs, dihydropyridine CCBs, direct vasodilators (e.g., hydralazine), and alpha-blockers.○ An increase in TFC with titration of non-diuretic antihypertensives suggests a compensatory fluid retention and may be a reason to intensify diuretic therapy. When someone has developed edema with medications, an increasing TFC might confirm central volume overload, whereas a stable TFC suggests lower extremity pooling due to venodilation, as with dihydropyridine CCBs.
Dyspnea	<ul style="list-style-type: none">○ When a patient has dyspnea, it may be due to cardiac or noncardiac (usually pulmonary) causes. Studies have shown [Springfield, CHF 2004] that $CI \leq 2.4$ or the combination of $STR \geq 0.55$ with $CI < 3.0$ suggests a cardiac cause.○ As with all hemodynamic measurements, much greater confidence is possible when the current readings reflect a significant change from prior measurements.

Other Considerations

Treatment Protocols	Several treatment algorithms are available based on hemodynamic data obtained using ICG. Specifically, recommendations for treatment of hypertension and congestive heart failure based on hemodynamic subsets are available, as are protocols for optimizing atrioventricular delay in patients with dual chamber pacemakers.
Documentation	It is important to document the key hemodynamic findings either on the ICG Printed Status Report itself or elsewhere in the medical record. Current BP medications should be listed. The plan should include medication changes, nonpharmacologic recommendations, future ICG, or other tests indicated on the basis of hemodynamic status and follow up.
