Is Measurement of Cardiac Output Using Impedance Cardiography Accurate?

To the Editor:

I believe the article by Pianosi (February 1997) concerning the measurement of cardiac output using impedance cardiography (ICG) illustrates a potentially serious defect in the evaluation of measurement techniques, related to results calculated using a formula with parameters not directly determined by the proposed measurement. Specifically, the formula for calculation of cardiac output, \( Q \) (stroke volume \( \times \) heart rate) contains variables for the resistivity of blood, \( r \); the distance between electrodes, \( L \) (which is an indirect measurement of height); ventricular ejection time, \( VET \); heart rate; and the measured parameter \( [dZ/dt]/Z_0^2 \). The authors compare \( Q \) determined from this formula to \( Q \) determined from \( CO_2 \) rebreathing and from oxygen consumption. I suspect that in this experiment, \( VET \) and heart rate contribute much more significantly to the described correlation between differently determined \( Q \)s than does \( [dZ/dt]/Z_0^2 \), the presumed basis for the measurement. Without presentation of the behavior of the impedance data related to the alternatively measured cardiac output, it is not possible to establish the contribution of the impedance measurement itself to the final result, and thus it is not possible to conclude that the ICG is any better for measuring changes in \( Q \) than, for example, a value of \( Q \) calculated on the basis of \( VET \) and heart rate alone.

Bernard E. Pennock, PhD
Allegheny General Hospital
Pittsburgh

REFERENCE

1 Pianosi PT. Impedance cardiography accurately measures cardiac output during exercise in children with cystic fibrosis. Chest 1997; 111:333-37

To the Editor:

According to the protocol previously described, \( \) heart rate was measured by the impedance device and used to compute cardiac output by both impedance and rebreathing. Hence, one could think of our results as simply comparing stroke volume measurements by each method, multiplied by the same constant. As such, the correlations observed would not be due to heart rate.

I will address the question of the relationship of cardiac output to ventricular ejection time (VET) by saying I demonstrated that stroke volume increased with increasing exercise intensity, while VET decreased as heart rate increased. There is no direct correlation between the two, and thus one cannot postulate that VET contributes to the increasing cardiac output seen with increasing oxygen uptake. If one looks at Figure 1 in my paper (February 1997), \( \) there was illustrated a 56% reduction in \( dZ/dt \) from the first and second to the third heartbeat, accompanied by a 60% reduction in stroke volume; there was only a 28% difference in VET. This figure was placed to show the quality-control features of the device. Although the third beat was tainted by movement artifact, Figure 1 nonetheless illustrates the relationship between stroke volume and \( dZ/dt \). I did not directly correlate \( dZ/dt \) or changes thereof with stroke volume, but there was a definite observable trend toward greater \( dZ/dt \) values with increasing stroke volume within individuals.

Paul T. Pianosi, MD
IWK-Grace Health Care Centre
Halifax, Nova Scotia

REFERENCE

1 Pianosi PT. Impedance cardiography accurately measures cardiac output during exercise in children with cystic fibrosis. Chest 1997; 111:333-37

Air, By Any Other Name . . .

To the Editor:

I found the “Roentgenogram of the Month” (February 1997) showing a chordoma of the thoracic spine very interesting. Just as interesting was the statement that arterial blood gas tensions were measured while the patient breathed “room air.” The term “room air” has always confused me, since it implies that air outside the “room” is somehow different in composition! I believe that the adjective “room” is usually superfluous and that we simply should describe the usual gas mixture which we all breathe as “air.”

Carl M. Kirsch, MD, FCCP
Santa Clara Valley Medical Center
San Jose, Calif

REFERENCE

1 Cury JD, Peterson RJ, Lacy GD, et al. Tracheal deviation from an atypical mediastinal mass. Chest 1997; 111:503-05

An Alternative to the Football Helmet

To the Editor:

Esophageal variceal hemorrhage is an all too common and difficult condition to manage in critical care practice. One treatment for this disorder, balloon tamponade with a Sengstaken-Blakemore (SB) tube, may have utility as a temporizing measure to stabilize patients pending a more definitive procedure to lower portal pressures (e.g., transjugular intrahepatic portosystemic shunt, or TIPS). Unfortunately, since balloon tamponade itself is of minimal long-term benefit and is associated with considerable risk of aspiration and esophageal rupture, many centers have no recent experience in its use. Therefore, needed adjunctive equipment (e.g., a football helmet with a face guard) to anchor the SB tube in place is often unavailable when urgently needed.

One improvisational anchoring tool we have used in this circumstance is the Ultra-Fit anesthesia face mask (Model #43410—600 Ultra-Fit; Baxter Healthcare Corp; Round Lake Beach, Ill), which is readily available and commonly used with Ambu-bags (Ambu Inc; Lintlicum, Md) or breathing circuits. Our technique is simple. After SB tube placement, the face mask is positioned with the SB tube through the central orifice. The gastric balloon is then inflated, and the tube is pulled tight and then anchored with surgical tape to the mask. The mask itself is then taped or strapped in place on the patient. This device has a number of attributes which make it useful for this purpose: 1) it is readily available in emergency departments and ICUs; 2) it is made of transparent material to allow clear visualization of the patient; 3) it does not preclude use of an endotracheal tube for airway protection; 4) it has soft cushioning at contact points to limit tissue damage; and 5) it is easily removed when necessary.
Hepatic Bleeding and Hemorrhagic Shock Following Thrombolytic Therapy in Patients With Acute Myocardial Infarction

To the Editor:

Severe bleeding is the major adverse effect of thrombolytic therapy (TT), though the occurrence of hepatic hematoma (HH) and hemorrhagic shock as results of the aforementioned treatment is very unusual.1,2 Liver hemangioma and hepatic trauma are conditions that can facilitate this complication. To our knowledge, there is only one report (that of Fox et al3) of nontraumatic hepatic bleeding related to TT in a patient with acute myocardial infarction (AMI). We report here two patients with AMI who developed this complication following TT.

Case 1

A 71-year-old woman was admitted to the ICU with an inferior AMI. Her physical examination, and laboratory and coagulation studies were normal. The patient received aspirin, heparin, and rt-PA, 100 mg in 90 min. During the following 12 h, the patient had recurrent episodes of vomiting and abdominal distension, and her blood pressure and hematocrit level progressively dropped. A CT scan revealed a large subcapsular HH (Fig 1) and hemoperitoneum. An angiographic study did not reveal any vascular malformation, and the hepatic artery was embolized. On the second day after admission, she showed clinical data that suggested intra-abdominal rebleeding. A laparotomy was performed and the hepatic artery was bound. The patient died of cardiogenic shock.

Case 2

A 57-year-old man was admitted with an anterior AMI. His physical examination, and laboratory and coagulation studies were normal. He was given anisylolated plasminogen streptokinase-activated complex (APSAC), 30 IU, and aspirin. Twenty-four hours later, the patient complained of abdominal pain and subsequently developed a hypovolemic shock. An abdominal ultrasound study showed the presence of free intra-abdominal liquid. A laparotomy disclosed a large HH and blood in the peritoneal cavity. Because of incoercible hepatic bleeding, liver packing was performed. Hepatic angiography showed no evidence of hepatic hemangioma. The patient gradually improved and was discharged from the hospital.

In previous reports,4,5 liver hemangioma or hepatic trauma were reported as conditions that could have facilitated liver bleeding in patients with TT, but in the cases presented here, there were no liver malformations or hepatic traumas.

In summary, hepatic bleeding caused by TT in patients with AMI is very exceptional but should be investigated in patients with unexplained gastrointestinal symptoms or hypovolemic shock following thrombolysis.

ACKNOWLEDGMENT: The authors thank Dr. Raúl de la Fuente-Fernández for his critical review of the manuscript.

REFERENCES

Cardiopulmonary Effects of Laparoscopic Surgery, Revisited

To the Editor:

In a well-written article, Sharma et al (September 1996)1 reviewed the cardiopulmonary effects of laparoscopy. However, there are two additional studies of respiratory mechanics of interest to your readers.

One study measured airway flow and airway and esophageal pressures of anaesthetized/paralyzed, tracheally intubated patients during mechanical ventilation.2 Measurements were made in the

Figure 1. Abdominal CT scan revealing a large hepatic subcapsular hematoma.