sis but, in their article, there are many other reasons for developing lactic acidosis. There is no precise presentation of hemodynamic data. The cardiac index is quite low. The blood pressure is stable only with the administration of dopamine, epinephrine, and nitroprusside infusion after disconnection of the bypass machine. In fact, this critically ill patient probably developed lactic acidosis during extracorporeal circulation (bicarbonate level, 19 mmol/L [19 mEq/L]), whereas epinephrine therapy was not begun. In their article we do not know if it is a cardiac output (liter per minute minus one [L/min \(^{-1}\)]) or a cardiac index (liter per minute per square meter [L/min/m\(^2\)]) and the patient has a 2-m\(^2\) surface. If pressure in the right side of the heart is about 10 mm Hg, on interpretation peripheral resistance is at 2800 or 1333 dynes/cm\(^2\)/s \(^{-1}\). We cannot calculate important data such as the difference between arterial and venous oxygen content (C [a-v] \(O_2\)), calcium dioxide, or peripheral oxygen extraction based on the data. It is not possible in these circumstances to exclude a type A lactic acidosis, especially after cardiac surgery, usually done with bypass hemodilution that could be associated with a poor oxygen extraction.

The patient had a non-insulin-dependent diabetes mellitus and was obese. In this circumstance, insulin resistance is very common and the high serum glucose level was probably due to poor serum glucose control and high postoperative stress.

Based on the available data, lactic acidosis may be due to epinephrine or other drug infusion, or to poor perfusion related to low cardiac output. Regression of lactic acidosis occurred after improvement of hemodynamic data and bypass-related disturbances.

**Jean-Michel Guerin, MD Philippe Meyer, MD Yasmina Habib, MD Paris**


**In Reply.—** We thank Dr Guerin and associates for their interest in our article. We can reassure the reader that the patient's cardiac index was 2.0 L/min/m\(^2\)—an excellent value for an elderly patient after cardiac bypass and who had not received pressors. We emphasized in our report that the pressors were administered empirically, not because of hypotension, oliguria, or any other evidence of poor tissue perfusion. Thus, the scenario for type A lactic acidosis is conspicuously absent. A serum bicarbonate level of 19 mmol/L (19 mEq/L) is insufficient to support a diagnosis of lactic acidosis, and may reflect the mild hyperlactatemia that accompanies general anesthesia.\(^1\)

We have never seen insulin resistance of this severity in a diabetic patient, obese or otherwise, except in the rare circumstances of insulin\(^2\) or insulin receptor\(^3\) antibodies. The temporal relationship between insulin resistance and epinephrine administration is compelling, and the insulin resistance waned rapidly and concurrently as the epinephrine infusion rate was decreased.

It is not clear what is meant by "bypass related disturbance." The hemodynamic data provided indicate that the patient was not hypotensive, oliguric, or hypoxicemic at any time. Thus, we believe that this patient's lactic acidosis and insulin resistance were specifically induced by epinephrine.

**Michela Caruso, MD John M. Miles, MD Rochester, Minn**


**Pacing in Left Bundle-Branch Block During Swan-Ganz Catheterization**

**To the Editor.—** We read with interest the recent article by Morris and colleagues\(^1\) that reported a fairly low rate of developing complete heart block (CHB) during Swan-Ganz (SG) catheterization in patients with left bundle-branch block (LBBB). We feel that this is an important finding since several sources, including recent advanced cardiac life support procedural guidelines, recommend prophylactic placement of a temporary transvenous pacemaker prior to pulmonary artery catheterization.\(^2\)\(^3\) Although CHB and hemodynamically significant bradycardia during this procedure may be uncommon, its occurrence can be quite dangerous and even catastrophic. There are several additional points that should be mentioned. The data presented by Morris and associates emanate from SG procedures in a critically ill population. It should be emphasized that these data may not be applicable to patients with more extensive conduction system disease, eg, patients with LBBB and a history of syncope and patients with LBBB undergoing permanent pacemaker implantation or diagnostic catheterization of the right side of the heart with a hard wire. The authors correctly do not make these extrapolations.

During SG catheterization in critically ill patients with LBBB, two additional options merit consideration. First, pacing can be accomplished, if necessary, noninvasively by an external transthoracic pacing device.\(^4\) In patients with LBBB, we routinely have the external pacemaker paddles near the chest prior to catheterization of the right side of the heart, and this can be applied and rapidly activated during the procedure if CHB or hemodynamically significant bradycardia develops. Second, we frequently use a balloon-tipped catheter with a pacing-infusion port in the right ventricle. With the pace port SG catheter in place, a reliable transvenous pacemaker in the right ventricle can be rapidly inserted, if necessary. In patients with LBBB that is of new onset or of indeterminate age, particularly during acute myocardial infarction, we routinely have the external pacer correct positioned for immediate activation and an SG with a pacing wire through the right ventricular port would be used. In our experience, this type of pacemaker is reliable (at least for the short term), does not significantly prolong the SG procedure, and is not associated with a higher incidence of complications than with routine SG catheterization alone. In addition, placing the pacing wire via the SG route does not require the same degree of training or skill as placing a hard, transvenous pacing wire, and could easily be accomplished by internists skilled in SG placement. When pacing via the SG pace port catheter, we recommend checking pacemaker thresholds at least twice daily. In patients with LBBB, manipulation of any SG catheter should be performed, as much as possible, while the catheter tip is in the right atrium; catheter manipulation in the right ventricle should be avoided.

We feel that the use of an external transthoracic pacemaker and an SG with a pacing port should be strongly considered for patients with LBBB.

(Continued on page 984.)
sterile ticarcillin disodium and clavulane potassium for Intravenous Administration

BRIEF SUMMARY OF PRESCRIBING INFORMATION

INDICATIONS AND USAGE: TIMENTIN® is indicated in the treatment of infections caused by susceptible strains of the designated organisms in the conditions listed below:

Bacteriologic diagnosis, including cultures, by β-lactamase producing strains of Staphylococcus aureus, and Pseudomonas aeruginosa and other Pseudomonas species.

COMMENTS: TIMENTIN is contraindicated in patients with a history of hypersensitivity to cephalosporins.

ADMINISTRATION:

AMINOGLYCOSIDES: Intravenous administration is recommended. Intramuscular, subcutaneous, and oral administration are not recommended.

ADMINISTRATION OF OTHER ANTIBIOTICS: Other antibiotics used concurrently with TIMENTIN should be administered in appropriate dosage. Concomitant therapy with aminoglycosides is not recommended. 

POISONING FROM TIMENTIN: Exposure to TIMENTIN by the oral route or parenteral route should be treated with supportive measures. No specific treatment is known for TIMENTIN poisoning other than the administration of carbon dioxide to hasten the elimination of the drug from the body.

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of which are undergoing catheterization of the right side of the heart for hemodynamic monitoring.

CARL J. LAVIE, MD
BERNARD J. GERSH, MB, ChB, DPHIL
Rocheester, Minn


In Reply.—Drs Lave and Gersh have addressed several important issues regarding our study.1 They suggest that in patients with left bundle-branch block (LBBB), extensive symptomatic conduction disease or the use of rigid catheters pose additional risks for developing catheter-induced complete heart block. We did not have the data to support or refute this contention. It is noteworthy that, in our study, the incidence of catheter-induced complete heart block in patients with LBBB was extremely low, despite the fact that these patients were critically ill. The majority of patients in our study had acute myocardial infarction, congestive heart failure, and/or cardiogenic shock. It is not clear if the risk for catheter-induced complete heart block in patients with LBBB is greater with severe conduction disease than with cardiogenic shock and/or ongoing myocardial ischemia.

We agree with Drs Lave and Gersh that recent refinements in external transthoracic temporary cardiac pacing2 provide an excellent method for rapidly initiating cardiac pacing, should catheter-induced complete heart block develop. Although external transthoracic pacing can be initiated immediately, there is failure to capture in approximately 20%.2 Thus it is imperative that the capability for transthoracic pacing be immediately available. The transthoracic pacing wire can be inserted through the introducer sheath or vein used for the initial pulmonary artery catheterization, or through a pacing-infusion port in the pulmonary artery catheter itself, as suggested by Drs Lave and Gersh. However, since catheter-induced complete heart block frequently occurs when the catheter tip enters the right ventricle initially, the pacing-infusion port of the pulmonary artery catheter may not be in a position that provides a significant advantage for positioning the pacer wire into the right ventricle. Finally, catheter-induced heart block will often resolve spontaneously by withdrawing the offending catheter. If the patient develops a transient catheter-induced complete heart block, it may be more prudent to place a transvenous pacemaker first, before the diagnostic pulmonary artery catheterization.

DIAGNOSTIC VALUE OF THE MEDICAL HISTORY

To the Editor—I read "The Diagnostic Value of the Medical History" in the November issue of the Archives with interest.1 I am not surprised that faculty and residents alike claim a high interest in the medical history. I am afraid that they are just spouting the party line. Their actual skill in, use of, and trust of the medical history may be far from what they claim.

After having observed over 500 clinical interviews, I am still impressed with the low general level of skill. But no one ever claims to doubt the value of the process; to do so would be equivalent to an admission of incompetence. I recall one resident whom I observed for a month while attending on the medical service at our local university hospital. The resident was egregiously incompetent at relating to people. We had several patients sign out against advice while he was on our service. I observed him interrupt a patient with "I have to see you in before you go home," several times of a personal nature. He was unable to hear his patients, insistently controlled all interviewing with a series of questions that the patient could only answer "yes" or "no," and reliably alienated his patients. He easily ignored my comments about routes to improvement. Then, at the end of the month, he told me, over a cup of coffee, that he was "ever grateful that he had mastered history and physical examination skills at a very early stage," since they stood him in such good stead during his residency. I was struck speechless.

DENISE MORRIS, MD
SAN DIEGO


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Editor's Correspondence