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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^{-5}/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 tissue 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.

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1. Caruso M, Orszulak TA, Miles JM: Lactic acidosis and insulin resistance associated with epinephrine administration in a patient with non–insulin-dependent diabetes mellitus. *Arch Intern Med* 1987;147:1422-1424.

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 hypoxemic at any time. Thus, we believe that this patient's lactic acidosis and insulin resistance were specifically induced by epinephrine.

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JOHN M. MILES, MD
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1. Stjernström H, Jorfeldt L, Wiklund L: The influence of abdominal surgical trauma upon the turnover of some blood-borne energy metabolites in the human leg. *J Parent Ent Nutr* 1981;5:207-214.
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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 correctly 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

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IN PNEUMONIA...
TIMENTIN®
sterile ticarcillin disodium and clavulanate 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:

Bacterial Septicemia: including bacteremia, caused by β-lactamase producing strains of *Klebsiella* spp., *E. coli*, *Staphylococcus aureus* and *Pseudomonas aeruginosa* (and other *Pseudomonas* species).

Lower Respiratory Infections: caused by β-lactamase producing strains of *Staphylococcus aureus*, *Hemophilus influenzae* and *Klebsiella* spp.

Bone and Joint Infections: caused by β-lactamase producing strains of *Staphylococcus aureus*.

Skin and Skin Structure Infections: caused by β-lactamase producing strains of *Staphylococcus aureus*, *Klebsiella* spp., and *E. coli*.

Urinary Tract Infections (complicated and uncomplicated): caused by β-lactamase producing strains of *E. coli*, *Klebsiella* spp., *Pseudomonas aeruginosa* (and other *Pseudomonas* species), *Citrobacter* spp., *Enterobacter cloacae*, *Serratia marcescens*, and *Staphylococcus aureus*. While TIMENTIN is indicated only for the conditions listed above, infections caused by ticarcillin susceptible organisms are also amenable to TIMENTIN treatment due to its ticarcillin content. Therefore, mixed infections caused by ticarcillin susceptible organisms and β-lactamase producing organisms susceptible to TIMENTIN should not require the addition of another antibiotic.

Appropriate culture and susceptibility tests should be performed before treatment in order to isolate and identify organisms causing infection and to determine their susceptibility to TIMENTIN. Because of its broad spectrum of bactericidal activity against Gram-positive and Gram-negative bacteria, TIMENTIN is particularly useful for the treatment of mixed infections and for presumptive therapy prior to the identification of the causative organisms. TIMENTIN has been shown to be effective as single drug therapy in the treatment of some serious infections where normally combination antibiotic therapy might be employed. Therapy with TIMENTIN may be initiated before results of such tests are known when there is reason to believe the infection may involve any of the β-lactamase producing organisms listed above; however, once these results become available, appropriate therapy should be continued.

ON ADMISSION

Based on the *in vitro* synergism between TIMENTIN and aminoglycosides against certain strains of *Pseudomonas aeruginosa*, combined therapy has been successful, especially in patients with impaired host defenses. Both drugs should be used in full therapeutic doses. As soon as results of culture and susceptibility tests become available, antimicrobial therapy should be adjusted as indicated.

CONTRAINDICATIONS: TIMENTIN is contraindicated in patients with a history of hypersensitivity reactions to any of the penicillins.

WARNINGS: SERIOUS AND OCCASIONALLY FATAL HYPERSENSITIVITY (ANAPHYLACTOID) REACTIONS HAVE BEEN REPORTED IN PATIENTS ON PENICILLIN THERAPY. THESE REACTIONS ARE MORE LIKELY TO OCCUR IN INDIVIDUALS WITH A HISTORY OF PENICILLIN HYPERSENSITIVITY AND/OR A HISTORY OF SENSITIVITY TO MULTIPLE ALLERGENS. THERE HAVE BEEN REPORTS OF INDIVIDUALS WITH A HISTORY OF PENICILLIN HYPERSENSITIVITY WHO HAVE EXPERIENCED SEVERE REACTIONS WHEN TREATED WITH CEPHALOSPORINS. BEFORE INITIATING THERAPY WITH TIMENTIN, CAREFUL INQUIRY SHOULD BE MADE CONCERNING PREVIOUS HYPERSENSITIVITY REACTIONS TO PENICILLINS, CEPHALOSPORINS, OR OTHER DRUGS. IF AN ALLERGIC REACTION OCCURS, TIMENTIN SHOULD BE DISCONTINUED AND THE APPROPRIATE THERAPY INSTITUTED. **SERIOUS ANAPHYLACTOID REACTIONS REQUIRE IMMEDIATE EMERGENCY TREATMENT WITH EPINEPHRINE, OXYGEN, INTRAVENOUS STEROIDS, AND AIRWAY MANAGEMENT, INCLUDING INTUBATION, SHOULD ALSO BE PROVIDED AS INDICATED.**

PRECAUTIONS: While TIMENTIN possesses the characteristic low toxicity of the penicillin group of antibiotics, organ system functions should be assessed periodically during therapy.

Bleeding manifestations have occurred in some patients receiving β-lactam antibiotics. These reactions have been associated with abnormalities of coagulation tests such as clotting time, platelet aggregation and prothrombin time and are more likely to occur in patients with renal impairment. If bleeding manifestations appear, TIMENTIN treatment should be discontinued and appropriate therapy instituted.

TIMENTIN has only rarely been reported to cause hypokalemia. Periodic monitoring of serum potassium may be advisable in patients receiving prolonged therapy.

Pregnancy (Category B). Reproduction studies have been performed in rats given doses up to 1050 mg/kg/day and have revealed no evidence of impaired fertility or harm to the fetus due to TIMENTIN. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

DOSAGE AND ADMINISTRATION: TIMENTIN should be administered by intravenous infusion (30 min.). Usual recommended dose for systemic and urinary tract infections for average (60 kg) adults is 3.1 Gm TIMENTIN (3.1 Gm vial containing 3 Gm ticarcillin and 100 mg clavulanic acid) given every 4 to 6 hours. In urinary tract infections, a dosage of 3.2 Gm TIMENTIN (3.2 Gm vial containing 3 Gm ticarcillin and 200 mg clavulanic acid) given every 8 hours is adequate. Please see official package insert for details on dosages for other patients, including those with renal insufficiency, and directions for use.

SUPPLIED: 3.1 Gm and 3.2 Gm Standard Vials; 3.1 Gm and 3.2 Gm Piggyback Bottles; 31 Gm Bulk Pharmacy Package; 3.1 Gm ADD-Vantage™ Antibiotic Vial

1 Data on file, Medical Department, Beecham Laboratories. 7548/E-BS
2 In retrospective reviews of (1) 228 pneumonia patients enrolled in comparative studies; and (2) 181 patients with lower respiratory tract infections enrolled in multicenter TIMENTIN clinical trial protocols, who would appear to be candidates for DRG 79 (respiratory infection + inflammation, age > 69, and/or C.C.).

*Due to susceptible organisms. *In vitro* activity does not necessarily imply *in vivo* efficacy.

†Clinical response defined as cured or improved. Clinical cure defined as complete resolution of all presenting signs and symptoms by the end of therapy. Improvement defined as substantial diminution in the severity of presenting signs and symptoms. Bacteriologic response defined as elimination of initial pathogen during therapy and for the duration of follow-up or unavailability of culture material.

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(Continued from page 981.)

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

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1. Morris D, Mulvihill D, Lew WYW: Risk of developing complete heart block during bedside pulmonary artery catheterization in patients with left bundle-branch block. *Arch Intern Med* 1987;147:2005-2010.
2. Kimbiris D, Dreifus LS, Linhart JW: Complete heart block occurring during cardiac catheterization in patients with preexisting bundle-branch block. *Chest* 1974;65:95-97.
3. Kaye W: Invasive monitoring techniques, in McIntyre KM, Lewis AJ (eds): *Textbook of Advanced Cardiac Life Support*. Dallas, American Heart Association, 1983, pp 165-196.
4. Zoll PM, Zoll RH, Falk RH, et al: External noninvasive temporary pacing: Clinical trials. *Circulation* 1985;71:937-944.

In Reply.—Drs Lavie 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 Lavie and Gersh that recent refinements in external transthoracic temporary cardiac pacing[2] 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 transvenous pacing be immediately available. The transvenous 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 Lavie 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.

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1. Morris D, Mulvihill D, Lew WYW: Risk of developing complete heart block during bedside pulmonary artery catheterization in patients with left bundle-branch block. *Arch Intern Med* 1987;147:2005-2010.
2. Zoll PM, Zoll RH, Falk RH, et al: External noninvasive temporary cardiac pacing: Clinical trials. *Circulation* 1985;71:937-944.

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 an intake history 11 times for phone calls, several 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 all 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.

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