Pericardial effusions are often hemorrhagic, especially with the large number of patients now undergoing hemodialysis for end-stage renal failure. In the performance of pericardiocenteses in such patients, it is often difficult to determine from appearance alone the source of fluid initially obtained. Even pericardial effusions with a hematocrit level of 10% or less can suggest an intravascular source on gross examination, especially when the patients involved are known to be anemic. Some urgency exists to perform such procedures expeditiously, and laboratory delay in the reporting of hematocrit determinations is variable. Although hemorrhagic pericardial fluid may often not clot on standing, if sufficient protein is present, such may not be the case. Conversely, whole blood from patients on dialysis or with other clotting abnormalities may remain liquid on standing after removal.

When contacting the left ventricle the exploring needle will usually transmit pulsations easily detected by the physician. When the left ventricle is penetrated, bright blood is usually obtained and also easily observed as pulsatile in nature, indicating the location. However, blood emerging from the right ventricle, even when the pressures within are abnormally elevated, may drip slowly from the pericardial catheter because of damping during transmission. The low oxygen content may also cause confusion with a peripheral venous source or hemorrhagic pericardial/pleural effusion. As has been noted by others,[2] the right ventricle may be penetrated without perception of resistance by the operator or the warning of extrasystoles.

Many of these confounding variables were demonstrated in the patients we report. We were especially misled in patient 1 by his improvement in symptoms and the unexplained rise in systemic blood pressure while he was actually undergoing a significant phlebotomy. The drop in central venous pressure initially observed was obviously caused by hypovolemia rather than relief of cardiac tamponade. Chandraratna et al.,[3] in their two-dimensional study of 16 patients undergoing pericardiocentesis with the assistance of contrast echocardiography, emphasized visualization of the pericardial needle during performance of the study. Because of the potential for laceration of the heart associated with the presence of a needle, we recommend rapid withdrawal of the needle and advancement of the flexible Teflon sheath as soon as fluid is obtained. Echocardiographic contrast can then reveal the site of the catheter tip, and contrast remaining within the catheter lumen renders it echogenic, whereas heretofore we have been unable to visualize this particular catheter on numerous attempts.

Once a catheter is located within the right ventricle, we believe that surgical intervention is the wisest course because of some tendency of this chamber to leak after such penetrations, at least experimentally.[4] In patient 2 it was only the critical nature of the patient's status that impelled us to withdraw the catheter from the right ventricle to the pericardial space under emergency conditions, fortunately without harm. Finally, although not previously demonstrated, we believe that echocardiographic contrast injections performed in association with attempted pericardiocenteses may prove helpful in differentiating sampling from associated pleural effusions versus pericardial effusions. Loculations of pericardial effusions may also potentially be demonstrated with this technique.

In conclusion, contrast echocardiography can prove extremely helpful in providing rapid and accurate assessments in the performance of pericardiocenteses when hemorrhagic aspirates are obtained and when the right ventricle has unknowingly been entered.

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Exercise and heart rate variability

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The analysis of heart rate variability provides noninvasive information about the autonomic regulation of the heart and may provide important diagnostic and prognostic information in clinical situations such as myocardial infarction.[1] Heart rate variability has been shown to be affected by aerobic physical training.[2-4] In this study we compared patients primarily engaging in aerobic exercise (cyclists) versus those primarily engaging in isometric training (weight lifters) to assess the effect of different types of exercise on 24-hour heart rate variability.

Methods. Subjects were <40 years of age and included cyclists (VO2 >55 cc/kg/ml, $n = 12$), weight lifters (>5 hours of lifting weights per week, $n = 10$), and a sedentary control group ($n = 10$). Each subject underwent maximal upright bicycle testing with continuous electrocardiographic monitoring and direct gas exchange measurement for de-

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AM HEART J 1996;131:825-7.

0002-8703/96/$5.00 + 0 **4/4/68324**

Table I. Group characteristics

	Cyclists	*Weight lifters*	*Control group*	*p Value*
n	12	10	10	
Age (yr)	28.9 (2.9)	26.9 (2.1)	27.6 (3.3)	NS
Height (cm)	174.3 (1.8)	179.5 (2.6)	177.2 (2.0)	NS
Weight (kg)	68.5 (2.0)	85.2 (5.1)	81.5 (5.0)	NS
DBP (mm Hg)	74.0 (1.8)	77.0 (2.1)	77.8 (2.6)	NS
SBP (mm Hg)	121.2 (4.4)	130.0 (3.7)	129.8 (3.8)	NS
HR preexercise (beats/min)	64.9 (1.7)	67.5 (2.1)	72.9 (2.9)	NS
LV mass (gm)	186.6 (17.6)	202.9 (15.1)	167.5 (4.7)	NS
Fractional shortening (%)	35.5 (3.2)	33.2 (2.3)	35.4 (1.9)	NS
VO2max (ml/kg/min)	65.5 (1.8)	44.7 (2.4)†	34.2 (1.6)†	*<0.01 †<0.05

DBP, Diastolic blood pressure; *SBP*, systolic blood pressure; *HR*, heart rate; *LV*, left ventricular.
*$p < 0.01$, difference between cyclists and weight lifters.
†$p < 0.05$, difference between weight lifters and controls.

Table II. Heart rate variability

	Cyclists	*Weight lifters*	*Sedentary*	*p Value*
Time domain				
SD R-R	104.8 (11.4)	87.4 (13.3)	80.6 (11.4)	NS
Mean R-R (msec)	839.0 (52.1)	788.2 (56.7)	754.6 (39.6)	NS
Frequency domain				
Total power	11189 (2726)	8706 (2412)	6815 (1603)	NS
Low power (0.04-0.15 Hz)	2456 (484)	2995 (872)	1968 (444)	NS
High power (>0.15-0.4 Hz)	1562 (608)	1805 (709)	821 (323)	NS

termination for VO2max, had two-dimensional echocardiography performed for determination of left ventricular mass,[5] and had 24-hour ambulatory monitoring. Subjects did not exercise during the monitoring period and were instructed not to have cardiotonic beverages (e.g., coffee, cola). Heart rate variability analysis in time and frequency domain was performed on the 24-hour ambulatory monitoring tapes (Del Mar Avionics spectra scan model 563 Holter analysis system, Irvine, Calif.). The entire 24-hour period was evaluated; heart rates between 40 and 110 beats/min were accepted. Results are reported as mean ± SEM. Statistical analysis was performed with one-way analysis of variance. When significance was obtained, Sheffe's test was used to look for pair-wise differences.

Results. Cyclists, weight lifters, and control groups had similar baseline characteristics (Table I), including preexercise heart rate and blood pressure. The combined athletic group (weight lifters + cyclists) had significantly greater left ventricular mass ($p < 0.05$) than the sedentary control group. Cyclists had significantly higher VO2max than weight lifters ($p < 0.01$), and weight lifters had significantly higher VO2 max than the sedentary control group ($p < 0.05$) (65 + 1.6 ml/kg/min vs 44.7 + 2.4 ml/kg/min vs 34.2 + 1.6 ml/kg/min, respectively). No difference was seen between groups with respect to either time domain or frequency domain variables (Table II). A significant correlation was found between VO2max and the standard deviation of the R-R interval ($r = 0.41$, $p = 0.02$), but no significant correlation was seen between VO2 max and indexes from frequency domain analysis.

Comment. Significant chronic exertion often has an effect on the heart.[6, 7] Previous studies have shown that heart rate variability is affected by degree of aerobic fitness,[2-4] which was confirmed in the time domain in our study. This study was designed to determine whether performing primarily isometric exercise (such as weight lifting) or primarily dynamic exercise (such as cycling) affects heart rate variability. Our data show that the mean R-R interval did not differ between groups over a 24-hour period. These data suggest that except for aerobic conditioning, the training effect in our athletes did not affect the heart rate or heart rate variability during activities of daily living. Thus a patient's overall degree of chronic physical activity rather than the type of exercise usually performed requires consideration when doing heart rate variability analysis. Further studies of the effect of training status on heart rate variability in an older population should be done to determine whether the results are similar in a population more likely to have heart disease.

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Dynamic left ventricular outflow tract obstruction as a complication of acute myocardial infarction

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Acute myocardial infarction is associated with several well-recognized complications, including mitral regurgitation and ventricular septal defect, both of which have clinical symptoms of hypotension and a new systolic murmur. In this report we present two patients noted in a 9-month period who had clinically significant dynamic left ventricular outflow tract obstruction as a consequence of ischemia and/or infarction in the left anterior descending coronary artery distribution. In each case, hypotension and a transient systolic murmur were noted. In neither case was there evidence of a hypertrophic cardiomyopathy before the acute event. In patient 1, resolution of the outflow tract obstruction was documented by Doppler echocardiographic techniques after resolution of wall motion abnormalities. The initial clinical impression in this case was that of ventricular septal defect or mitral regurgitation in association with acute myocardial infarction. In patient 2, myocardial infarction was an unexpected finding in a patient with presumed sepsis. Echo recognition of dynamic left ventricular outflow tract obstruction in these situations allowed appropriate avoidance of inotropic agents and resolution of the acute clinical syndrome.

Patient 1. A 78-year-old woman with a history of hypertension and recurrent endometrial adenocarcinoma was admitted to the hospital with nausea and vomiting 2 days after taking a course of adriamycin. Vital signs included a temperature of 97.7° F, pulse of 111 beats/min, blood pressure of 131/99 mm Hg, and respirations of 24 breaths/min. The lungs were clear to auscultation. The cardiac examination demonstrated normal first and second heart sounds; no murmur was noted. One month earlier, a baseline echo had demonstrated mild left ventricular hypertrophy but otherwise normal chamber sizes and normal left ventricular systolic function. There were no segmental wall motion abnormalities. The aortic valve was focally thickened without significant stenosis. The peak velocity in the left ventricular outflow tract was 1.7 m/sec by Doppler. Six days after admission the patient had dyspnea, weakness, and persistent nausea. Physical examination revealed rales in the basilar half of the lungs bilaterally. The cardiac examination revealed an S_3 gallop and a new grade III/VI systolic ejection murmur at the left sternal border and apex. Hypotension developed, with a blood pressure of 70/40 mm Hg, and the patient was noted to have an elevated temperature of 100.8° F. She was transferred to the intensive care unit for treatment of presumed sepsis. Hypotension did not respond to fluid administration, and dopamine was begun. A 12-lead electrocardiogram demonstrated Q-waves in leads V_1 and V_2, with 3 mm horizontal ST depression in leads V_3 through V_5. Serial enzymes were negative for acute myocardial infarction. An echo compared with the study performed 1 month earlier demonstrated new areas of akinesis in the apex, distal septum, and anterior walls consistent with anteroapical myocardial infarction. Hyperkinesis of the basilar myocardial segments was noted in association with prominent systolic anterior motion of the mitral valve and a left ventricular outflow tract gradient of 80 mm Hg. The spectral Doppler profile was consistent with dynamic outflow tract obstruction. Dopamine was decreased, and the patient's blood pressure gradually returned to normal. Twenty-four hours later the murmur had diminished to grade I/VI and was not noted on subsequent physical examinations. The patient died 45 days later from underlying malignancy.

Patient 2. A 79-year-old woman with a history of near syncope was referred to the University of Michigan for electrophysiologic study. The basic study was unremarkable, and there was no evidence of conduction abnormality or inducible supraventricular or ventricular arrhythmia. As part of an aggressive arrhythmia provocation protocol, an isoproterenol infusion was begun. Shortly afterward, severe retrosternal chest pain developed. A 12-lead electrocardiogram (Fig. 1, right) revealed a current of injury in the anterior precordial leads consistent with impending anterior myocardial infarction. Immediate cardiac catheterization was performed, and coronary arteriography demonstrated a 90% stenosis of the proximal left anterior descending coronary artery. This stenosis was successfully reduced to 30% with balloon angioplasty. The patient tolerated the procedure and was transferred to the coronary care unit in good condition. Chest pain and ST-segment elevation rapidly resolved, and creatine phosphokinase peaked at 272 IU with an MB fraction of 39 ng/ml at 24 hours. Eighteen hours after the acute event, hypotension developed and a grade 3/6 systolic murmur was noted at the apex and left sternal border. The differential diagnosis

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AM HEART J 1996;131:827-30.

0002-8703/96/$5.00 + 0 **4/4/70 065**