only occasionally. The puzzle has many pieces, such as the general health of the individuals, the intake of salt and water, underlying pathologic conditions (three persons developing severe high altitude pulmonary edema at only 2,900 meters [9,514 feet] were found to have previously unsuspected congenital absence of one pulmonary artery), or individual idiosyncrasies. Hultgren et al found by studying six persons who previously had several bouts of high altitude pulmonary edema that their pulmonary arterial pressures rose to much higher levels than the normally elevated pressures usually found at altitude, even though none of the six subjects showed evidence of high altitude pulmonary edema at the tested altitude of 4,600 meters (15,092 feet).

Hultgren et al and Scoggin et al have made impressive cases. What might be some of the clinical implications? Obviously, one must warn the victim of high-altitude pulmonary edema about the need for extra precautions when returning to altitude after a brief stay lower down, and one should probably caution persons who have had more than one or two bouts to be particularly careful on subsequent visits to altitude. Are there some lessons for persons at sea level, too?

Thousands of persons are severely hypoxic at sea level, due to pulmonary insufficiency from one cause or another; in effect, they live at altitude, often higher than 3,500 to 4,500 meters (11,483 to 14,784 feet) in terms of oxygen in the blood. Occasionally, these persons must be given therapy with supplemental oxygen, bringing them, as it were, down to low altitude. Do they become more vulnerable to pulmonary or cerebral edema when treatment is stopped? Acute pulmonary edema after near drowning or near fatal heroin overdosage or during and after prolonged shock is not uncommon and may be due to acute hypoxia like that during rapid ascent; but in some instances, pulmonary edema appears when the crisis seems over, after therapy has been halted. Are these experiences equivalent to edema developing on reascent after a period in an environment with normal oxygen? Recently, it has been shown that arterial oxygen saturation falls sharply during sleep, probably due to disturbed breathing patterns. Well known is the exacerbation of symptoms during the night and in the early morning among patients with chronic pulmonary conditions. Is the sudden infant death syndrome, which is remarkably more common during sleep, in any way similar to high altitude pulmonary edema after reascent? If so, could some of the preventive measures used by climbers benefit the sick? Therapy with acetazolamide (Diamox), nikethamide (Coramine), or medroxyprogesterone 17-ace-

tate (Provera) has been proven of value to mountaineers; will these medications help the hypoxic ill patient or infants vulnerable to sudden infant death syndrome as well? Would the ability of phenytoin sodium (Dilantin) to stabilize membranes decrease the migration of salt and water and thus diminish high altitude pulmonary edema, cerebral edema, and acute mountain sickness? Should such medicines be recommended to those returning home after a stay at low altitude? The study by Hultgren and Marticorena and many other studies at altitude should provoke new thoughts about persons with a lack of oxygen at sea level, as well as those going to high altitude.

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Tachycardia Induced by Atrial Pacing

Tachycardia induced by atrial pacing is widely employed in the evaluation of patients with pain in the chest. Tachycardia is a simple method of increasing left ventricular work and producing myocardial ischemia. Evidence of myocardial ischemia can be monitored easily in the catheterization laboratory by assessing electrocardiographic, hemodynamic, and myocardial metabolic changes. Furthermore, the ischemic state induced by atrial pacing disappears rapidly when sinus rhythm is restored, thus providing a safety factor unavailable in other forms of stress (eg, exercise or infusion of isoproterenol). Atrial pacing does not replace exercise stress tests for the evaluation of patients with ischemic heart disease. Instead, atrial pacing is a supplementary technique that makes it possible to examine left ventricular hemodynamics, coronary blood flow, and myocardial metabolic function be-
fore, during, and after the induction of the ischemic state.

Pain in the chest can be induced by rapid atrial pacing in most patients with typical angina pectoris. In patients with coronary arterial disease, Sowton et al. found a "threshold" for angina that could be consistently reproduced by pacing the right atrium.

ST-segment changes on the electrocardiogram during pacing are often difficult to interpret (because of the pacing artifact); however, ST-segment depression that persists after the termination of pacing is abnormal and is usually the manifestation of myocardial ischemia.

Brachial arterial pressures change little during atrial pacing in response to an increase in heart rate; however, in patients who develop angina pectoris during pacing, a slight increase in mean brachial arterial pressure is noted. In 1970, Conti et al. noted an increase in mean coronary arterial pressure (measured in the orifice of the coronary artery) in most patients during myocardial ischemia produced by atrial pacing. In this issue of Chest (see page 381), Loeb and his colleagues convincingly show an elevation of arterial systolic pressure during atrial pacing; this elevation occurred only in the presence of coronary arterial disease and was nearly always associated with clinical, electrocardiographic, and metabolic evidence of myocardial ischemia. Loeb et al. report that the rise in arterial systolic pressure frequently paralleled the laboratory signs of myocardial ischemia and was apparent before significant thoracic discomfort occurred. They appropriately interpret this observation to mean that it was unlikely that the rise in arterial systolic pressure was the result of pain in the chest.

Left ventricular end-diastolic pressure generally decreases during tachycardia induced by atrial pacing in normal subjects. Left ventricular end-diastolic pressure usually returned to normal within three to four beats of the resumption of sinus rhythm. A similar response was noted in patients with coronary arterial disease in whom angina pectoris did not occur during atrial pacing and in whom there were no metabolic abnormalities to suggest the presence of myocardial ischemia. In contrast, patients with coronary arterial disease who developed angina pectoris and metabolic evidence of myocardial ischemia during atrial pacing usually had no change in left ventricular end-diastolic pressure during pacing-induced tachycardia prior to the onset of angina. In these same patients, with the onset of angina pectoris, the left ventricular end-diastolic pressure rose slightly, and it rose markedly in the period immediately after pacing. The pathophysiology of this marked elevation of left ventricular end-diastolic pressure after pacing is not clear. It may be due to decreased ventricular diastolic compliance during ischemia. Regardless of the mechanism, it seems that elevation of left ventricular end-diastolic pressure during pacing is another marker of myocardial ischemia.

The advantages of tachycardia induced by atrial pacing over other forms of stress are as follows: (1) no effort is required on the part of the patient (thus, patients with physical limitations can undergo stress); (2) the procedure is safe because stress can be terminated abruptly; (3) the level of stress is reproducible in the same patient; and (4) hemodynamic and metabolic studies can be done during the stress.

The disadvantages of tachycardia induced by atrial pacing are as follows: (1) pacing-induced tachycardia generally does not increase cardiac output and systolic blood pressure to the same degree as exercise, and, in addition, venous return is usually reduced (thus, ventricular volume is decreased); these effects of pacing-induced tachycardia may make it difficult to produce myocardial ischemia, except at very high heart rates; (2) the ECG is often difficult to interpret during pacing because of interference by the pacing artifact; and (3) the procedure requires catheterization of the right heart.

In summary, tachycardia induced by atrial pacing is, as Balcon et al. have said, "a way of exercising the heart without exercising the patient." It is particularly useful for evaluating pain in the chest in patients suspected of having ischemic heart disease. The development of typical angina and hemodynamic, metabolic, and electrocardiographic changes of myocardial ischemia during pacing-induced tachycardia provide physiologic evidence that a given coronary arterial narrowing is responsible for the pain in the chest. The observation reported by Loeb et al. in this issue of Chest provides an additional hemodynamic marker of myocardial ischemia during pacing-induced tachycardia.

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