army. During all that time, the aforementioned complaints persisted. At the beginning of June, he made ten dives in five days to a maximum depth of 30 meters. At the beginning of July, he was examined at a medical center because of upper respiratory infection. Nothing exceptional was reported, and he was given antibiotics and symptomatic medications. The next day, while instructing at a diving course, he swam a distance of 50 meters underwater, breathholding, as an example to the trainees. During July 1983, he made about 25 dives to depths between 3 and 50 meters. On July 20, when he was diving, he touched a grey sponge with his right arm. The next day he felt hangover, mild fever, and had dermatitis on his arm. He referred to the local hospital. On examination, he looked generally in a good condition. There was no dyspnea, tachypnea, or cyanosis. The blood pressure was 130/80 mm Hg; pulse rate, 80 per minute and regular; and axillary temperature was 37°C. In the chest wall examination, there was hyperresonance in percussion of the right lung, and in auscultation, there were markedly diminished breath sounds on the same side. A chest roentgenogram showed right pneumothorax (Fig 1). An intercostal tube was inserted. The lung was fully expanded and the intercostal drain was pulled out a day later. All his complaints disappeared.

After going over the details the diver was asked to bring the minographic roentgenogram taken on April 19. This roentgenogram clearly shows a pneumothorax (Fig 2). This pneumothorax is identical to the one observed on July 21 (Fig 1) and was misdiagnosed at the time it was taken.

DISCUSSION

A history of spontaneous pneumothorax is a contraindication for diving. Most of these patients suffer from pulmonary pathology, usually subpleural emphysematous blebs or bullae. The recurrence rate is high: 33 percent in five years according to Edmonds et al., an average of 20 percent to 30 percent according to Kizer or 50 percent according to Saunders and Ingram. This high recurrence rate is the main reason for prohibiting further diving. The possibility of diving after traumatic pneumothorax is in dispute. Some authors would disqualify such a diver for good. Others would let him dive after complete recovery, normal chest roentgenogram, and probably, normal results on pulmonary function tests.

At any event, there is total undoubted agreement that no one should be allowed to dive while he has a pneumothorax.

This diver made many dives with a pneumothorax present for five months. Coughing, even heavily, is not a common cause of pneumothorax. Immediately afterwards, this diver had chest pain and permanent discomfort while breathing. This favors the diagnosis that he had this pneumothorax since his fit of coughing on February 20. He is athletic and well built and seems to have very good lung function, so the pneumothorax bothered him substantially only during hard physical effort such as running. His excellent physical capacity is proved by his ability to lift heavy weights and swim a distance of 50 meters underwater while breath holding. While he was diving, the air bubble between the pleurae became smaller according to Boyle's law, so the deeper he went, the better he felt. During the flight, he felt much more short of breath. This is because the aircraft is pressurized only to 0.8 atmospheres, so that the pneumothorax probably increased in size.

The pneumothorax is clearly proved by the chest roentgenogram taken on April 19 (Fig 2). However, this was a minograph, taken and examined simultaneously with dozens of others, so it seems that it was not examined too carefully. The final diagnosis was delayed until July 21. The pneumothorax in Figure 1 is very much like the one in Figure 2.

It is quite uncommon for a pneumothorax to exist for such a long time. Usually the air in the pleural space will be reabsorbed spontaneously, since the sum of the partial pressures of gases in the pleural space (ie, air = 760 mm Hg) is greater than the sum of the partial pressures of gases in the end-capillary blood due to low end-capillary PO2. The rate of reabsorption of air from a pneumothorax without intervention is about 1.25 percent of the total roentgenographic area per day, so a moderate size pneumothorax may take four to six weeks to resolve.

Pneumothorax bears a great risk for divers. For this reason, the roentgenograms taken routinely for them must be examined very carefully.

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Hypotension and Sinus Arrest with Nifedipine in Pulmonary Hypertension*

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An adverse reaction to the acute administration of sublingual nifedipine is reported. Subsequent reversal was by intravenous administration of calcium chloride.

Nifedipine has been proposed as therapy for both primary and secondary pulmonary hypertension. Because of

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the variable response in individual patients with pulmonary hypertension, the short-term effects of several vasodilators, including nifedipine, are routinely tested in our catheterization laboratory prior to institution of long-term therapy.

**CASE REPORT**

A 52-year-old white woman was initially admitted for evaluation of asymptomatic electrocardiographic right ventricular hypertrophy. The patient's only significant medical history was that of biopsy-proven alcoholic cirrhosis. Examination revealed a right ventricular heave, loud pulmonic second sound, splenomegaly, edema, and ascites. Arterial blood gas revealed a PaO$_2$ of 66 mm Hg, PaCO$_2$ of 34 mm Hg, and a pH of 7.44. Cardiac catheterization (Table I) revealed normal right atrial, right ventricular end-diastolic, pulmonary artery wedge, and left ventricular end-diastolic pressures. Pulmonary artery pressure and arteriolar resistance were markedly elevated. Cardiac output was reduced. There was no evidence of intracardiac shunting. Pulmonary arteriography did not show evidence of pulmonary emboli. Left ventricular and coronary angiography were normal. Hemodynamics did not improve after oxygen inhalation, intravenous phentolamine, or hydralazine.

The patient returned 30 months later with increasing dyspnea, peripheral edema, and ascites. Radionuclide angiography demonstrated an enlarged and poorly contracting right ventricle. Repeat catheterization (Table I) revealed the pulmonary artery pressures to be unchanged from the first study, but the right ventricular end diastolic (22 vs 6 mm Hg) and right atrial (18 vs 2 mm Hg) pressures were higher, suggesting the development of pulmonary hypertension. The systemic arteriovenous oxygen difference (9.3 vs 8.5 volume %) and the cardiac index (1.3 vs 2.3 L/min/m$^2$) had worsened from the previous study. Systemic vascular resistance was elevated at 2,157 as was pulmonary vascular resistance at 1807 dyne/sec/cm$^5$. Resting hypoxemia had worsened with PaO$_2$ of 45 mm Hg, PaCO$_2$ of 26 mm Hg, and pH of 7.46.

At the time of sublingual nifedipine (10 mg) administration, femoral artery pressure was 110/70, and the patient was in sinus rhythm. Ten minutes later the sinus rate slowed from 85/minute to 72/minute. An ectopic atrial pacemaker was followed by a junctional escape rhythm (63/minute). With this rhythm, the cardiac index and blood pressure fell (Table I). Intravenous administration of atropine (1.4 mg) resulted in transient (30 second) reappearance of sinus rhythm with an increase in blood pressure. When the junctional rhythm returned, blood pressure and cardiac index fell further (Table I).

Intravenously administered calcium chloride (500 mg) produced an increase in systemic pressure and cardiac index within five minutes despite persistence of a junctional escape rhythm and no change in heart rate (Table I). A further 500 mg of calcium chloride was followed by reappearance of an ectopic atrial rhythm at essentially the same rate as the junctional rhythm (68/minute vs 66/minute). Arterial pressure and cardiac index rose further (Table I). Within several hours, normal sinus rhythm returned and blood pressure rose to baseline levels.

**DISCUSSION**

Nifedipine provoked hypotension and sinus arrest in this patient with pulmonary hypertension. The negative inotropic action of nifedipine is suggested by a rise in the right-sided filling pressure at a time when the pulmonary artery pressure had fallen and the resistance to flow, as measured by the pulmonary resistance, had remained essentially unchanged. Although hypotension as a consequence of myocardial dysfunction has not been reported with nifedipine, such a response is not surprising in that nifedipine, like other calcium channel blockers, decreases in vitro the rate of rise and strength of contraction of isolated atrial, ventricular, and vascular smooth muscle. In contrast, in vivo, these effects have not been apparent, and the lack of negative inotropism has been attributed to sympathetic nervous system reflex response to the peripheral vasodilation and lowering of blood pressure induced by nifedipine. The manifestation of right ventricular dysfunction may have been a result of adding a negative inotropic agent to an already critically compromised right ventricle, which, in the baseline state, was under maximal sympathetic stimulation.

Nifedipine also caused sinus arrest. Again, based on in vitro studies, this finding is not surprising. Nifedipine can slow the spontaneous sinus rate and decrease the amplitude of the sinus node potential. In contrast, in the non-anesthetized awake dog, it produces an increase in heart rate, and electrophysiologic studies in humans have shown a decrease in sinus node recovery time and an increase in sinus node firing of 20 to 30 percent. Here again, an already stimulated sympathetic system may have negated the reflex tachycardia usually seen.

Intravenous administration of calcium chloride promptly increased blood pressure, even before restoration of atrial activity. This response is compatible with an improvement in myocardial function, or less likely, a reversal of vasodilation. Experimentally, calcium chloride has been shown to reverse both the myocardial depression and vasodilation produced by nifedipine. Atrial activity further augmented cardiac performance. Calcium chloride therapy has been reported to be effective in cases of verapamil intoxication.

This case emphasizes the potential of nifedipine to have deleterious hemodynamic and electrophysiologic effects, at least in the setting of pulmonary hypertension and right ventricular dysfunction.

<table>
<thead>
<tr>
<th>Table I—Cardiac Catheterisation*</th>
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<tr>
<td><strong>State</strong></td>
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<td>Rhythm/rate</td>
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<tr>
<td>CO</td>
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<tr>
<td>PA Sys/Dias/Mean</td>
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<td>PAR</td>
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<td>SVR</td>
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<td>*CaCl is intravenous calcium chloride; CO, cardiac output (L/min); Dias, diastolic; EAR, ectopic atrial rhythm; FA, femoral artery pressure (mm Hg); JER, junctional escape rhythm, Min, minutes; N, nifedipine; NSR, normal sinus rhythm; PA, pulmonary artery pressure (mm Hg); PAR, pulmonary vascular resistance (dynes-sec-cm$^{-5}$); RA, mean right atrial pressure (mm Hg); and Sys, systolic.</td>
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ventricular dysfunction. It also strongly suggests that, as with verapamil, these effects may be reversed by calcium chloride. We recommend that patients be tested carefully in the hemodynamic laboratory before prescription of this agent for treatment of pulmonary hypertension.

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Biofeedback and Hypnosis in Weaning from Mechanical Ventilators*


Weaning patients from mechanical ventilation can be hindered by both physical and psychologic factors. Biofeedback has been used successfully as an adjunct in difficult weaning problems. We have used a combination of hypnosis and biofeedback to wean a patient with neurologic disease who previously failed weaning by standard procedures. A 30-year-old woman with respiratory failure secondary to multiple sclerosis with transverse myelitis was given eight sessions of biofeedback over 12 days in which the movements of her chest wall, as monitored by magnetometers, were displayed on an oscilloscope. The patient was praised for targeted respiratory rate, amplitude, and rhythm. These sessions included hypnosis in which the patient was given suggestions of well-being and that she could breathe as she had five years earlier. In this manner the patient was successfully weaned. Respiratory biofeedback and hypnosis appear to be useful adjuncts in weaning patients from ventilators.

Weaning patients from mechanical ventilators can be difficult. Both physiologic and psychologic factors can hinder the weaning process. An increasing number of medical problems have been helped by biofeedback, eg, tension headache, migraine, and Raynaud's disease. Patients given visual or auditory feedback of normally unconscious physiologic processes often have been able to gain control over these processes. Corson et al reported two cases of paralyzed patients who were dependent on mechanical ventilation. They viewed their tidal volume and respiratory rates via an oscilloscope connected to transducers on the chest wall or to a pneumotachometer, thus obtaining information about breathing movements usually provided unconsciously via sensors in the upper airways and chest wall proprioceptors. The patients were trained to approach normal respiratory rates and normal tidal volumes. Both were successfully weaned after this intervention. Prior to the use of biofeedback, standard medical and psychologic techniques had been unsuccessful.

Yarnal et al reported that a less formal biofeedback procedure has been helpful in weaning a wide variety of patients from ventilators. Hackett and Cassem state that hypnosis has been useful in weaning patients from ventilators, probably by alleviating the anxiety associated with the weaning process.

The following is a case report in which biofeedback and

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