funding and low salary. As many readers are aware, most research laboratories in US medical schools are staffed by researchers born outside of this country. Fortunately for us, many of these researchers opt to stay and enrich this country with their skills. In contrast to the sad situation here, many other wealthy nations have increased their research funding, resulting in a significant proportion of new research coming from these nations. A random review of abstracts selected for presentation at the national American Heart Association meetings in Orlando in November 1997 revealed that 47% of all accepted abstracts originated from countries other than the United States, about 11% from Japan, and 9% from Germany. Of the US abstracts (53% of total), an individual born outside the United States was the first author on 45%! This is clear evidence that a US-born individual is unlikely to enter a career in biomedical research.

We as a nation have to seriously consider whether we want to continue to be leaders in biomedical research. Besides national prestige, research in biomedical sciences has direct economic implications. Haven’t we learned enough from losing a significant market share in automobiles, computers, televisions, and electronics to other countries over the last 2 decades? I implore all readers of this editorial to remind their Congressional representatives of the need to significantly increase funding for biomedical research. I also look forward to readers’ comments, preferably by email.

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What Happens in the Dead Sea?

Oxygen therapy works miracles in the hypoxic patient through multiple mechanisms. The first mechanism acts directly on gas exchange, resulting in an increase in arterial partial O₂ pressure that improves O₂ saturation (SaO₂) and O₂ content (CaO₂) of arterial blood. The second relaxes the pulmonary vascular bed, thus decreasing pulmonary vascular resistance and improving right ventricular performance. The third mechanism relieves shortness of breath by decreasing the hypoxic drive from the carotid body.

Conventionally, O₂ therapy is implemented by giving patients an increased fraction of inspired O₂ concentration (FIo₂); an alternate approach is to increase environmental pressure, thus giving patients a higher partial pressure of inspiratory O₂ (Pİo₂). Increased Fİo₂ is widely used in daily patient care. On the contrary, hyperbaric oxygenation remains mostly in the realm of underwater medicine, the exclusive turf of Navy submarine medical personnel. In civil medicine, indication of hyperbaric oxygenation is limited to acute CO poisoning or similar conditions that render hemoglobin transiently ineffective for O₂ transport. Here one is dealing with a truly hyperbaric intervention, with several atmospheres of pure O₂ required to load enough O₂ on the plasma by physical solution in order to satisfy survival basal metabolism.

In this issue of CHEST (see page 571), Kramer and coworkers have made the first attempt to manipulate environmental pressure for O₂ therapy in the common hypoxemia of COPD by conducting a rehabilitation of a group of Jerusalem patients in the Dead Sea. Their approach has several original features. It is equipment-free, taking advantage of the special topography of the Dead Sea as the lowest area on earth. The investigators have used a very modest degree of hyperbaric intervention, the pressure differential between Jerusalem and the Dead Sea being approximately 100 mm Hg. Thus, their mini-hyperbaric approach is safe from most of the usual hyperbaric complications.

Despite the fact that the Pİo₂ increase was merely 21 mm Hg (equivalent to what is achieved by O₂ at 1 to 2 L/min at sea level), their results in the Dead Sea were more than impressive. Shortness of breath was relieved, albeit at the price of a mild CO₂ retention. Arterial O₂ markedly increased, as did work capacity and maximum O₂ consumption (V̇O₂).

The V̇O₂ increase seemed to exceed what would be expected from the increase in SaO₂ and CaO₂ alone. This suggests that maximum cardiac output increased. That the O₂ pulse increased beyond what would be expected from the SaO₂ and CaO₂ increase alone is consistent with an increase in stroke volume being the mechanism for the increase in maximum cardiac output. It appears that a better filling of the left ventricle was observed from better pulmonary venous return, thanks to improved pulmonary vascular resistance.

The simplicity of the investigation by Kramer and colleagues, of course, did not allow certain confirma-
tion of the mechanism responsible for the observed results. Nevertheless, their data still suggest an impressive advantage of the Dead Sea treatment over conventional O\textsubscript{2} therapy of equal intensity. An increase in P\textsubscript{IO\textsubscript{2}} of 21 mm Hg achieved by conventional O\textsubscript{2} supplementation at the rate of 1 to 2 L/min would not have yielded the same results. In fact, many of the studied patients had been on chronic O\textsubscript{2} supplementation in Jerusalem before the Dead Sea sojourn.

Under conditions of iso-P\textsubscript{IO\textsubscript{2}}, mini-hyperbaric O\textsubscript{2} therapy was obviously better than conventional O\textsubscript{2} therapy. What makes mini-hyperbaric treatment better than conventional O\textsubscript{2} therapy is puzzling. Was it because, by being truly continuous and sustained, the (modest) increase in P\textsubscript{IO\textsubscript{2}} during the Dead Sea sojourn was more effective than conventional O\textsubscript{2} therapy in relieving pulmonary hypertension? Or was it because the inspired gas composition remained unchanged, with F\textsubscript{IO\textsubscript{2}} remaining at 21%, so that only P\textsubscript{IO\textsubscript{2}} was increased?

The work of Kramer and colleagues raises the need for further studies to address numerous new questions. First, will their findings be duplicated in larger patient samples and more various shades of COPD? If so, can any new therapeutic modalities be derived from the Dead Sea experience?

The report by Kramer and colleagues should not be interpreted merely as proof that the Dead Sea therapy is more advantageous than conventional O\textsubscript{2} therapy for COPD patients. Not everyone is blessed with a Dead Sea in his or her backyard. If no general applications of the findings can be conceived and developed to benefit patient care elsewhere, the observation would remain merely a curious phenomenon related to the very special topography of a very unique land.

Fortunately, the Dead Sea environment (in terms of barometric pressure and temperature) can be artificially recreated without significant difficulty. The pressure differential between Jerusalem and the Dead Sea is rather modest, essentially in the vicinity of 100 mm Hg. The practical implication is that mini-hyperbaric hospital wards capable of withstanding a comparable pressure differential are not beyond the reach of current technology; much higher pressure differentials have been achieved routinely in airline passenger compartments.

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Right Ventricular Function in COPD

Can It Be Assessed Reliably by the Measurement of Right Ventricular Ejection Fraction?

Pulmonary hypertension (PH) is a frequent complication of advanced COPD. It is defined by a resting mean pulmonary artery pressure (PAP) >20 mm Hg. In COPD, PH is generally mild to moderate, with PAP (when measured in a stable state of the disease) most often ranging between 20 and 35 mm Hg,\textsuperscript{1,2} in contrast to the gross increases in PAP seen in primary pulmonary hypertension (PPH). This is confirmed by the results of Vizza and colleagues, which appear in this issue of CHEST (see page 576). They have investigated patients with advanced pulmonary disease who had been evaluated for lung transplantation, the majority of whom were COPD and \( \alpha_1 \)-antitrypsin (\( \alpha_1 \)-AT) emphysema patients. Pulmonary hemodynamics could be measured in 156 COPD and 54 \( \alpha_1 \)-AT emphysema patients; this group indeed represents one of the largest series in the literature. The mean (±SD) PAP was, respectively, 26±7 and 25±6 mm Hg, which is in very good agreement with earlier studies.

Thus, PH is not severe in most COPD cases, which are very different from cases of left heart and congenital heart disease, pulmonary thromboembolic disease, and particularly PPH, where PAP is usually >50 mm Hg; this is confirmed by Vizza and colleagues, since the average PAP in their group of 50 PPH patients is 58±21 mm Hg, very similar to the results of the National Prospective Registry study.\textsuperscript{3} It must be remembered that if PAP is slightly to moderately elevated in the COPD patient at rest, it may increase markedly and sometimes abruptly during exercise, acute respiratory failure,\textsuperscript{4,5} and REM sleep.\textsuperscript{6} These acute increases could favor the development of right heart failure.

According to the classical conception, PH leads to right ventricular hypertrophy, the so-called “cor pulmonale,”\textsuperscript{7} to right ventricular enlargement, which includes both hypertrophy and dilatation, and finally to right ventricular failure. However, the possible occurrence of right heart failure in COPD has been denied by some authors, who argue that the level of PH in most COPD cases is too modest to induce “true” ventricular failure.\textsuperscript{8} Indeed, peripheral edema is not synonymous with right heart failure in COPD patients, but on the other hand, objective signs (including hemodynamic data) of right heart failure