right ventricular afterload by administration of inhaled NO (10 to 20 ppm). NO inhalation failed to improve her clinical condition. In fact, we observed a rapid decline in oxygen saturation determined by pulse oximetry and confirmed by measurement of arterial blood gases (Table 1). The observed decline in oxygen saturation could not be attributed to the admixture of NO in N₂ because this has no effect on FIO₂ in our delivery system. After cessation of NO inhalation, there was a rapid, initial improvement of oxygenation, although complete recovery to baseline values took several hours. As no improvement was obtained with thrombolytic therapy, and the patient deteriorated further, embolecomy was performed. Following surgery, oxygenation and hemodynamics improved dramatically. Neurologic examination, however, revealed brain death due to prolonged periods of hypoxia. Postmortem examination revealed right ventricular hypertrophy of the heart, widespread PE, and cerebral edema without cerebral hemorrhage.

In this patient, inhaled NO worsened gas exchange, while rapid improvement of oxygenation after cessation of drug administration was demonstrated. We speculate that the intrinsic pulmonary vasodilating effects of NO undermine a delicately compensated Va/Q equilibrium. As in COPD, this will lead to an increase in shunt or ventilation/perfusion mismatch with worsening of oxygenation as a result. Whether this phenomenon only occurs in long-standing pulmonary diseases as COPD and recurrent PE remains unclear. This case report emphasizes once again that NO is a powerful but potentially dangerous drug that should be used with caution.

To the Editor:

We read with interest the clinical case reported by Dr. Tulleken and colleagues on the use of 10 to 20 ppm of inhaled nitric oxide (NO) in order to reduce right ventricular afterload in a 20-year-old woman hospitalized with severe respiratory distress, shock, and anuria after a history of attacks of shortness of breath for more than 6 months' duration. Following administration of NO, the authors observed a decrease in peripheral oxygen saturation and PaO₂. Oxygen desaturation without improvement in pulmonary artery hemodynamics may be unfavorable during the acute phase of pulmonary embolism. On the other hand, however, it is well known that a decrease in PaO₂ often accompanies an increase in pulmonary vascular perfusion and cardiac output in this special pathophysiologic setting. Therefore, oxygen desaturation per se does not necessarily reflect a negative effect of a specific treatment strategy with regard to the main target of treatment in acute life-threatening pulmonary embolism, right ventricular performance. To decide whether changes in oxygen saturation are associated with positive or negative net effects, it is necessary to measure pulmonary hemodynamics, cardiac output, mixed venous partial pressure of oxygen (PVO₂), and/or right ventricular function. Dr. Tulleken and coworkers used inhaled NO to reduce right ventricular afterload but did not report the effects of inhaled NO on pulmonary hemodynamics and cardiac output.

In an animal model of acute pulmonary microembolism induced by the injection of microspheres, we observed the positive effects of 40 to 80 ppm of inhaled NO on pulmonary hemodynamics. In contrast to results observed with other diseases where inhaled NO was used, PaO₂ and PVO₂ did not improve. This model differs from clinical thromboembolism, and caution should be taken to extrapolate the results directly to the clinical situation. However, the conclusions drawn from this experimental approach were supported by two further reports on the successful use of inhaled NO in patients suffering from acute pulmonary thromboembolism. In one case, a 74-year-old woman developed a right-to-left shunt through a patent foramen ovale following embolization. Inhalation of 25 ppm NO completely abolished right-to-left shunting, as documented by transesophageal echocardiography, and led to a decrease in mean pulmonary artery pressure from 41 to 36 mm Hg without changing mean arterial pressure. Moreover, NO inhalation resulted in an increase in PaO₂ from 61 to 133 mm Hg, and PVO₂ from 34 to 41 mm Hg. Thus, inhaled NO has the potential to reduce pulmonary artery pressure selectively following acute pulmonary embolism in human subjects. This can be associated with an increase in oxygen delivery and oxygen saturation. The pathophysiology of gas exchange following acute pulmonary embolism, however, is complex, and the spectrum of abnormalities depends on various individual factors. Therefore, the effects of inhaled NO on PVO₂ can be unpredictable in an individual patient.

In the present case, chronically recurrent pulmonary embolism occurred, which led to right ventricular hypertrophy. The pathophysiology of this entity differs from acute pulmonary thromboembolism with regard to the extent of ventilation-perfusion mismatch and shunt formation. It is thus possible that the observed oxygen desaturation, which has also been reported after the administration of inhaled NO in patients with COPD, might preferentially occur in patients with pre-existing respiratory disorders like COPD and recurrent pulmonary embolism.

If inhaled NO is used in patients suffering from pulmonary embolism, monitoring of pulmonary hemodynamics and cardiac output and/or assessment of right ventricular function by echocardiography should be performed in addition to blood gas analysis to calculate the net effects of this experimental thera-

Table 1—Arterial Blood Gases During Mechanical Ventilation in Patient With Massive PE

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.24</td>
<td>7.21</td>
<td>7.21</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>66.0</td>
<td>73.5</td>
<td>72.8</td>
</tr>
<tr>
<td>PaO₂</td>
<td>76.5</td>
<td>61.5</td>
<td>68.3</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>28.20</td>
<td>28.60</td>
<td>28.80</td>
</tr>
<tr>
<td>SaO₂</td>
<td>92.00</td>
<td>85.00</td>
<td>89.00</td>
</tr>
</tbody>
</table>

* A, 15 min before NO inhalation; B, during 15 min of NO inhalation (10 to 20 ppm); and C, 15 min after cessation of NO inhalation.

REFERENCES


Jacob E. Tulleken, MD, PhD
Jan G. Zijlstra, MD, PhD
Kim Evers, MD
Tjip van der Werf, MD, PhD
University Hospital Groningen, the Netherlands

Chest 1/112 / 1 JULY, 1997 297
peutic approach on oxygen delivery and right ventricular performance. This may allow us to decide appropriately whether an individual patient will benefit from inhaled NO.

Berrad W. Böttiger, MD  
Johann Motsch, MD  
Department of Anesthesiology  
Joachim Dörسام, MD  
Department of Urology  
Ulf Miek, MD  
André Gries, MD  
Jörg Weimann, MD  
Eike Martin, MD  
Department of Anesthesiology  
University of Heidelberg  
Germany

References
5 Blanch L, Baigorrí F, Fernandez R, et al. Efecto vasodilata-
7 Dantzer DR, Bower JS. Alterations in gas exchange follow-

Warn Asthmatics of Scuba Diving Risks

To the Editor:

I am writing to ask that very clear information and warnings be given to people with asthma (both newly diagnosed and chronic) concerning the risk of scuba diving. This concern stems from my husband’s recent and sudden death at the age of 47 from an asthmatic reaction subsequent to a relatively shallow scuba dive in Cancun, Mexico.

My husband was certified as a scuba diver in 1969 while in college. He had dived several times, but not for approximately 20 years. Apparently, certified divers are expected to know their own health risks and recertification and updating are generally not required, as they were not, in this case. Few questions are asked or warnings issued.

In my husband’s case, the dive instructor did indicate that asthma created some risk. However, because my husband had dived previously with no problem, while an asthmatic and while using inhalers, and was enough of a risk-taker to consider diving in the first place, he underestimated the degree of risk.

I am asking here that all physicians who treat people with asthma—and perhaps other lung diseases—tell their patients the degree of risk that scuba diving entails. The chronicity of asthma should be indicated as an additional risk. Even certified divers who have asthma should be asked to consult with their doctor prior to any dive. The standard waiver/release indicating that diving entails risk may be perceived as pro forma.

Information about diving with asthma, if communicated regularly between physician and patient, may save lives or avert the tragedy of injury. Similarly, information could be included in the literature accompanying asthma inhalers.

Ginger E. Benlifer, PhD  
Found Ridge, NY

Wisdom in Video-Assisted Cardiac Surgery

What Can or Should Be Done?

To the Editor:

We read with great interest the recent paper by Tsai and colleagues (December 1996)1 reporting their preliminary experience with the application of video-assisted techniques in reparative mitral valve surgery. We were impressed with the technical abilities of this group of enthusiastic surgeons. Nevertheless, we would like to express our concern regarding their patient selection and the conclusions of the report.

There is no doubt that the principle of minimizing the surgical incision has become widely accepted, and this has recently found its extension into the field of cardiac surgery.2-5 Yet, in cardiac surgery, there are as yet no data that confirm that smaller incisions are synonymous with less morbidity. All that can be said from the experience to date is that, in combination with the use of cardiopulmonary bypass, median sternotomy is likely to be associated with a higher morbidity than a lesser incision, but this remains to be substantiated.

Realizing this, if video-assisted cardiac surgery were to be an advancement on established practice, it would have to be advantageous in terms of patient outcome. Moreover, the safety of these innovative approaches must be addressed inasmuch as surgical precision and clinical outcome cannot be sacrificed for the yet-doubtful pledge of benefits ascribed to reduced short-term morbidity.

In the report of Dr. Tsai and colleagues,1 four very ill patients in need of emergency reparative mitral valve surgery were selected. One cannot help immediately noticing the exceptionally prolonged cardiopulmonary bypass perfusion time, with a mean of 222 min (of particular significance is patient 4, who underwent mitral valve thromboectomy requiring 320 min of cardiopulmonary bypass perfusion). Furthermore, the need for deep systemic hypothermia and hypothermic fibrillatory cardiac arrest in isolated mitral valve surgery is by no means standard practice. Bearing these issues in mind, one is obliged to question the wisdom of subjecting critically ill patients to “experimental” surgery, and to question whether these patients fared better than those undergoing conventional surgery. Yet, the paper by Tsai et al1 offers no data to support their actions, since it neither presents the patients’ postoperative progress nor demonstrates any other advantage of the video-assisted approach.