human immunodeficiency virus, means that condoms should be used with high-risk partners. But the findings reported by Hubacher et al. should reassure clinicians and women alike that copper IUDs, which are by far the most common type of IUD used in the United States and around the world, are not a threat to the health or future fertility of the women who use them, including those without children.

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THE ACUTE RESPIRATORY DISTRESS SYNDROME, MECHANICAL VENTILATION, AND THE PRONE POSITION

THE acute respiratory distress syndrome is a devastating, often fatal inflammatory condition that probably affects more than a million patients throughout the world each year.[1] Since its description over 30 years ago,[2] this syndrome has claimed the attention of clinicians and researchers because it is associated with an extremely high mortality rate and is difficult to treat, and because the pathophysiology of the disorder is unique.

The common denominator in the acute respiratory distress syndrome is hypoxemia. Virtually all affected patients require mechanical ventilation to improve arterial oxygenation and minimize the energy costs of breathing. Ironically, the focus on normalizing blood gases may have inadvertently contributed to the high mortality rate because of the large tidal volumes delivered during mechanical ventilation. Twenty years ago, tidal volumes of 15 to 20 ml per kilogram of body weight were commonly used in patients receiving mechanical ventilation for the acute respiratory distress syndrome in an attempt to achieve normal values for the partial pressure of carbon dioxide and oxygen saturation. However, experimental data have shown that ventilatory strategies that overdistend parts of the lung or allow the lung to cycle repeatedly between a collapsed state and an open state can lead to injury — so-called ventilator-induced lung injury.[3,4] Patients with the acute respiratory distress syndrome are particularly susceptible to this form of injury, because the disorder causes the collapse or consolidation of large regions of the lung, often leaving only a small percentage of the lung available for ventilation.[5] Hence, a tidal volume that might not overdistend the lung in a normal person could lead to regional overdistention and thus cause ventilator-induced lung injury in a patient with the acute respiratory distress syndrome.

The clinical effects of ventilator-induced lung injury may extend beyond the lungs. The majority of patients with the acute respiratory distress syndrome die not from hypoxemia but from multiple-organ failure.[6] The mechanisms leading to multiple-organ failure are probably multifactorial, but there is evidence that lung injury caused by mechanical ventilation can result in the release of several mediators, including proinflammatory cytokines.[7] These mediators, as well as endotoxin or bacteria, may enter the systemic circulation[8-10] and cause organ dysfunction and, ultimately, multiple-organ failure.[11] In other words, ventilator-induced lung injury, not hypoxemia, may be the primary cause of death in many patients with the acute respiratory distress syndrome. There are data that support this idea. Last year, a consortium sponsored by the National Institutes of Health (NIH) reported that reducing the tidal volume from 12 to 6 ml per kilogram decreased mortality by 22 percent among patients with the acute respiratory distress syndrome.[12] The implication of this finding is that by inducing iatrogenic lung injury during mechanical ventilation, clinicians have inadvertently been contributing to the high mortality associated with the syndrome.

How can clinicians improve oxygenation in patients with the acute respiratory distress syndrome

while limiting ventilator-induced lung injury? One approach that has gained popularity is to place patients in the prone position. Findings dating back to the mid-1970s[13] indicate that the prone position improves oxygenation in about 70 percent of patients with the acute respiratory distress syndrome,[14] and more recent data suggest that it may also limit ventilator-induced lung injury.[15] In this issue of the *Journal,* Gattinoni et al. report the results of a large, multicenter study of the effects of the prone position in patients with the acute respiratory distress syndrome.[16] The study showed that there was no significant difference in mortality between patients randomly assigned to placement in the prone position and those assigned to conventional treatment but that the patients assigned to the prone position had a significant improvement in the partial pressure of oxygen. These findings are reminiscent of the results of the NIH consortium, which reported that the ratio of the partial pressure of oxygen to the fraction of inspired oxygen for the first several days was lower in the group treated with a tidal volume of 6 ml per kilogram than in the group treated with a higher tidal volume, yet mortality was lower in the first group.[12] These results highlight the importance of focusing on important clinical outcomes such as mortality when assessing the benefits of a treatment, rather than on intermediate physiological markers such as hypoxemia.

One question that arises from the study by Gattinoni et al. is why there was no decrease in mortality among the patients assigned to placement in the prone position, given the strong rationale underlying the use of this position.[17] One explanation is that prone positioning does not decrease ventilator-induced lung injury, but there are a number of other possible explanations. First, the study may not have had adequate statistical power. The authors studied more than 300 patients, but this number may have been too small, given the imprecision inherent in the diagnosis of the acute respiratory distress syndrome, the heterogeneity of the underlying diseases that confer a predisposition to the syndrome, and the lack of uniformity among other interventions used, apart from mechanical ventilation. Second, patients were placed in the prone position for an average of only 7.0 hours per day. Thus, the patients were exposed to the potentially injurious effects of mechanical ventilation in the supine position for more than 70 percent of each day. Third, the authors limited the use of the prone position to 10 days, which may have been too short a period for any significant long-term benefit to occur. Indeed, the authors reported a trend toward a decrease in mortality at 10 days in the prone group; this effect was not apparent by the time of discharge from the intensive care unit. Clearly, future trials should examine the efficacy of maintaining the prone position for longer periods.

How can clinicians use the results of this study to improve patient care? Taken at face value, the findings do not support the use of the prone position in all patients with the acute respiratory distress syndrome. However, a post hoc analysis by Gattinoni et al. showed that placing patients in the prone position reduced mortality at 10 days in the quartile of patients who were the most ill. Post hoc analyses are notoriously unreliable, but this finding is intriguing. In caring for a patient with the acute respiratory distress syndrome (as with all patients), the clinician has to weigh the potential benefits of any therapy against its risks, often in the absence of definitive evidence. This is particularly true in the intensive care unit, where many patients have complex disease processes. I believe that for the most severely ill patients, if there are no contraindications, it is now reasonable to use ventilation at a low tidal volume with the patient in the prone position, for several reasons: the biologic rationale for using the prone position is strong; major complications, as ascertained in this study, appear to be limited; the costs are minimal; and there is evidence, albeit weak at present, that mortality is decreased in the subgroup of patients who are most severely ill. I do not mean to imply that the prone position should be the standard of practice or that there is no need for a definitive study. Indeed, the results of this study underscore the need for an investigation addressing the hypothesis that the prone position is beneficial when used for longer periods and for the most severe cases of the acute respiratory distress syndrome.

These are exciting times for intensivists caring for patients who require mechanical ventilation. Basic research on the mechanisms of ventilator-induced lung injury has led to the development of ventilatory strategies that have been shown to decrease mortality in randomized clinical trials. Future studies that identify patients at risk for ventilator-induced lung injury, test new ventilatory strategies, and identify the underlying molecular mechanisms of the lung injury will dramatically alter our approach to mechanical ventilation.

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The Jugular Venous Pulse and Third Heart Sound in Patients with Heart Failure

In my recent book on the cardiovascular physical examination, Braunwald wrote, "Intelligent selection of investigative procedures from the ever-increasing array of tests now available requires far more sophisticated decision-making than was necessary when the choices were limited to electrocardiography and chest roentgenography. The clinical examination provides the critical information necessary for most of these decisions."[1] The article by Drazner et al.[2] in this issue of the *Journal* is a step in the right direction and will help refocus attention on proper technique for the bedside examination of the heart and circulation.

In 1867, Potain described the wave forms of the internal jugular vein.[3] In 1902, Mackenzie established the jugular venous pulse as an integral part of the cardiovascular physical examination,[4] and in 1928, Wiggers wrote that the jugular venous pulse might be useful in the interpretation of dynamic events in the heart.[5] In 1956, Wood argued, "Precise analysis of the cervical venous pulse and measurement of the height of each individual wave with reference to the sternal angle is not only possible at the bedside but highly desirable."[6] Despite these important insights, bedside interpretation of the jugular pulse has lagged behind auscultation as a clinical skill.[7] The ill-defined term "jugular venous distention" is still used, despite the fact that it lacks specificity as an index of either right atrial wave form or right atrial pressure.

Mackenzie provided nomenclature that is still in use.[4] He called the two wave crests A and V and described them as follows:

> There are two rises in the auricular pressure curve, a large and a small one, with of course two falls. The first rise in pressure immediately precedes the rise in ventricular pressure. It can only be due to the systole of the auricle. Immediately after the auricle ceases to contract, there is a great fall (*x*) in the pressure due to the diastole of the auricle. The auriculo-ventricular valves being closed, the blood pouring into the auricles from the veins, the pressure gradually rises, producing the second small wave in the curve. This wave is terminated by the opening of the auriculo-ventricular valves at the beginning of ventricular diastole. When the pressure becomes lower in the ventricles than in the auricles, the valves open and the contained blood passes through, reducing the auricular pressure, and causing the second fall, *y*. After this the pressure slowly rises by the accumulation of blood in both chambers, until it is suddenly increased by the next auricular systole.

Figure 1 shows a normal jugular venous pulse.

Proper examination of the jugular venous pulse requires a bed or examining table that permits controlled adjustment of the patient's trunk above the horizontal plane.[7] The examination should begin with a 30-degree angle of elevation, with subsequent adjustment of the trunk to the angle that achieves the maximal visible oscillations of the right internal jugular vein. The patient's head should be turned slightly to the right to avoid compression of the internal jugular by the overlying sternocleidomastoid muscle. The external and internal jugular veins are both examined — the external for the mean right atrial pressure, and the internal for both wave form and pressure. The nonpulsatile external jugular vein may not be visible unless it is mechanically distended by digital compression at the base of the neck. The examining room should be darkened, and a light beam (from a pocket flashlight) directed tangentially to highlight the fluctuations of the right internal jugular pulse. The examiner's left hand should direct the light source while the thumb of the right hand palpates the left carotid pulse as a reference for timing, as Mackenzie recommended.[4] Heart sounds can be used as an alternative reference, which was Potain's recommendation.[3] What the eye perceives is a series of gentle, undulating crests and troughs. Attention should be focused on the nonpulsatile external jugular vein and then on the heights of the internal jugular A and V waves, in centimeters, above the sternal angle.[7]

Potain attributed the third heart sound to the sudden cessation of distention of the ventricle in early diastole and offered advice on how best to elicit this