Corticosteroids and Respiratory Muscles

Does It Matter?

Corticosteroids in high doses and for prolonged periods are frequently used in the treatment of many diseases, including chronic respiratory disorders.1,2 Unfortunately, steroids have a potential to cause several major side effects, including myopathy.3 Typically, steroids affect muscles that are less active, that are proximal, and that contain a predominance of type IIb, or fast glycolytic, fibers—characteristics not found in respiratory muscles, particularly the diaphragm.4 Nevertheless, steroid-induced myopathy of the respiratory muscles has been suggested, although its prevalence and clinical importance remain controversial.

In several animal models, corticosteroids have been shown to produce a myopathy of the respiratory muscles, which impacts on their function.5,6 However, extension of this information to humans is fraught with problems due to interspecies differences in steroid sensitivity and steroid metabolism and the excessive dosing typically used in animal models. In humans, various case reports have suggested that corticosteroids can produce an acute myopathy in the respiratory muscles,6 can cause prolonged respiratory muscle dysfunction following simultaneous administration with paralytic agents,7 and may induce respiratory muscle dysfunction in respiratory patients undergoing chronic steroid therapy.8 On the other hand, the results of clinical trials specifically designed to examine the impact of prolonged steroid use on respiratory muscle function have, in general, been negative.9

Unfortunately, several potential problems limit interpretation of the findings from the case reports and even the clinical trials. In most instances, respiratory muscle pathology was not assessed, with conclusions regarding the presence of a myopathy inferred from tests of respiratory muscle function. In addition, patients had underlying pulmonary diseases, which may have altered respiratory muscle function, independent of any effect of steroid therapy. Furthermore, most studies looked solely at respiratory muscle strength, without measuring muscle endurance or other aspects of respiratory muscle function. Finally, low doses of corticosteroids were typically used in the clinical trials.

In an attempt to address some of these issues, Weiner et al in this issue of Chest (see page 1788) report the effect of oral corticosteroids on respiratory muscle strength and endurance. Although not a blinded, controlled trial, the sole intervention likely to have impacted on respiratory muscle function in these patients was the corticosteroid therapy. It is noteworthy that they used corticosteroid doses prescribed for many acute and even chronic conditions and studied patients with no underlying pulmonary or neuromuscular disease and normal baseline respiratory muscle function, eliminating some of the problems described above. They observed moderate reductions in strength and striking reductions in endurance, which took several months to reverse following discontinuation of steroids. Of interest, they found that respiratory muscle endurance was affected earlier and more severely than strength, a finding that has been noted in in vitro animal experiments10 and that suggests that factors other than muscle atrophy with secondary

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weakness must also be present to explain the reduced endurance.

Thus, examination of respiratory muscle strength alone may be inadequate in assessing muscle dysfunction in steroid-induced myopathy of the respiratory muscles, and other factors, such as respiratory muscle endurance, may need to be measured. Clearly, additional information needs to be obtained and questions need to be answered. However, the possibility that corticosteroids can induce myopathy of the respiratory muscles and respiratory muscle dysfunction needs to be recognized and should be considered in all patients receiving steroids. This is of particular importance in pulmonary patients, in whom the potential benefits of corticosteroids on lung function may be overshadowed by their negative effect on respiratory muscle function. If unrecognized, these changes could lead to additional complications and mismanagement of patients.

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The Pendulum and the Arterial Line

In the United States, funding is currently derived from various sources and is spent according to the perceived needs of the hospital board in a market-driven system. More technology is available on average in US hospitals. The arterial line is part of that technology, but what are its positive or negative attributes and can the patients stand the cost? The current emphasis on cost containment and measuring “outcomes” for all of our procedures will certainly target the arterial line for close scrutiny. The intensive care unit is but not immune from any analysis of cost effective care.

The arterial line, if properly used, with good patient selection, is an asset. However, those who will measure the risk/benefit for this invasive device may well challenge some current practices. It is not a complex task for critical care physicians, nurses, and respiratory therapists to formulate a continuing quality improvement monitor that requires justification for insertion and length of stay. We have just such a respiratory therapy protocol at our institution. Our respiratory therapists are credentialed for arterial line placement and recertified and observed every year. Each line is monitored through a continuing quality improvement document. In 1991 and 1992, the therapists have placed 785 lines with three treated bloodstream infections using the same criteria as outlined by Gronbeck and Miller in this issue of Chest (see page 1716). In 1989 and 1990, nine bloodstream infections were noted with no protocol in place. Physician placement has occurred in 311 lines in 1991 and 1992, usually in the operating room or surgical intensive care unit.

In addition, our protocol called for health care providers to justify lines in place for more than 96 h. The only major disappointment in the Gronbeck/Miller data was that 40 percent of their lines were in place more than 96 h (clinically needed?). Some justification responses that could not be upheld by a risk/benefit analysis of outcomes included the following: (1) the ICU policy includes arterial lines on all patients on ventilators; (2) the physician will order so many laboratory tests that the arterial line is convenient for phlebotomy; (3) the phlebotomy is necessary for legal reasons; and (4) “the site looks good.”

There will be an increasing role for all physicians and physician extenders to take an active role in curtailing these types of “straw man” arguments. Three giant steps have already been taken. Drs. Smoller and Kruskall from Harvard Medical School noted that patients in the ICU had more blood drawn (944 ml—almost 4 U) and had blood drawn more often (four times per day) with arterial lines in place. They warned that evaluation of test-ordering patterns can