Deep venous thrombosis (DVT) is a recognized complication in people ‘deviating from a good state of health’. In the general population epidemiologists find an annual frequency of DVT of 160/100,000, with 70 (about 44%) developing pulmonary embolism (PE). The figures change dramatically when we look at hospital patients, particularly those with orthopedic surgery, those in ICUs, but even those on internal wards. In the latter, the ones with the highest risk are stroke patients, who develop DVT in more than 56%. Acute myocardial infarction (22%) and other medical conditions (17%) follow at some distance. The mortality rate from PE in stroke is 19% (1 in 1 in DVTs) and 10% in postinfarction patients (half of those with DVT). These figures contrast considerably with the mortality from PE in general surgery patients, which is only 4% [1].

The prerequisite of PE is peripheral venous thrombosis. The risk of the latter increases with immobilization, age over 65, multimorbidity and tissue damage at any site of the body. These risk factors certainly coincide in surgery, ICU and stroke patients, but (with exemption of major tissue damage) also in patients with exacerbation of severe COPD. In an analysis of the PIOPED study population, 108 patients with COPD were investigated according to their propensity to PE. About one fifth were eventually diagnosed with PE, who did in no way behave differently from the majority of COPD patients who had no PE (21 with, 87 without PE). There were 35 patients with moderate to severe airflow obstruction, and of these 2 (6%) had PE [2].

In a more recent analysis of the PIOPED study [3] the risk of dying within 1 year was almost 2-fold higher among 399 patients with PE plus COPD (24/45, 53%) than in those who did not have COPD (71/354, 20%). However, the risk associated with COPD seemed to be much less in comparison from the majority of COPD patients who had no PE (21 with, 87 without PE). There were 35 patients with moderate to severe airflow obstruction, and of these 2 (6%) had PE [2].

I would suggest the following conclusions:

1. COPD patients develop DVT at about the same rate as other medical ICU patients.
2. The incidence of submassive PE in COPD patients is not different from that in patients without COPD, but the proof of true PE in COPD is extremely troublesome and – in general – will not massively influence the management of these patients.
3. COPD patients who suffer PE are at increased risk of immediate and 1-year mortality, but risk factors of other underlying diseases (e.g. neoplasm, infection, cardiac disease) are of greater importance.
Identifying those with DVT and pulmonary hypertension by noninvasive, reliable measures (D-dimer, sonography, echocardiography) could be of help for a well-balanced anticoagulant prophylaxis or even therapy.

References

Respiration 1998;65:172

Are Sleep Studies Always Needed before Treating Sleep-Disordered Breathing?

Sleep has a number of beneficial effects, none of them are particularly useful for breathing. The activity of upper airway dilators decreases, the accessory muscles of respiration and the intercostal contract less forcefully, airway resistance increases, and sensitivity of respiratory neurons to various chemical and reflex stimuli diminishes. All of this leads to a fall in alveolar ventilation relative to metabolic rate and rises in PCO₂ and falls in arterial PO₂. Generally in healthy people these changes are not very great. Even the brief periods of hypopnea, apnea and periodic breathing that can occur during sleep are usually of little clinical significance unless there are associated cardiac arrhythmias or daytime drowsiness. But, in those with neuromuscular diseases, or respiratory illness, the adverse effects of sleep on breathing can lead to acute or chronic respiratory failure.

Barthlen in her paper in ‘Respiration’ [1] correctly points out that sleep-related respiratory disorders can produce sufficiently severe and/or prolonged hypoxia to lead to polycythemia, heart failure, and hypertension. She recommends that polysomnographic evaluation and other studies performed by a sleep specialist be undertaken in patients with neuromuscular disease who have symptoms suggesting respiratory distress during sleep. Barthlen recommends treatment by noninvasive ventilatory support, continuous positive pressure ventilation, bilevel positive airway pressure, or IPV for those who have sleep-disordered breathing.

Drs. Ishikawa and Bach [2] point out that intermittent positive pressure ventilation using a molded plastic mouthpiece with a lip seal can be more effective than nasal and oral-nasal methods of noninvasive ventilation in many patients with neuromuscular diseases, a conclusion which they have documented in a study of over 250 patients with a variety of muscular and neuromuscular disorders. In addition since inadequate cough frequently complicates these diseases, Ishikawa and Bach emphasize the importance of making provisions for expiratory muscle weakness. They insist that these patients frequently do need sophisticated sleep studies for the noninvasive support of breathing. Rather noninvasive ventilation should be tried and its effects on gas exchange be monitored. They make a persuasive argument for their viewpoint.

In this era where the costs of health care delivery are being scrutinized everywhere it would be worthwhile to see if sleep studies are in fact cost-effective in patients with neuromuscular disorders. The data may already exist in some laboratories of some respiratory group and just need to be analyzed.

References
1 Barthlen GM: Nocturnal respiratory failure as an indication of noninvasive ventilation in the patient with neuromuscular disease. Respiration 1997; 64(suppl):35–38.

Neil S. Cherniack, Newark, N.J., USA