inability to perform low-level exercise is associated with increased postoperative morbidity and mortality. It has long been recognized that bedrest can produce deconditioning and can impair aerobic performance. The results of the study by Girish et al remind us that inactivity due to obesity or other medical conditions that limit mobility can have a similar deconditioning effect and thereby can increase the risk of postoperative morbidity.

Third, Girish et al found no difference in postoperative morbidity between patients undergoing upper abdominal and thoracic operations. It is obvious why a thoracic incision would impair chest wall excursion and would predispose the patient to pulmonary complications. However, it is less readily apparent why surgery that does not directly involve the diaphragm would be associated with increased morbidity. The diaphragm is an integral part of the chest wall apparatus, and upper abdominal surgery clearly is associated with impaired diaphragmatic movement and decreased transdiaphragmatic pressure generation. The mechanism of this impairment is unclear, but the consequence is increased cardiopulmonary morbidity compared to that in patients undergoing lower abdominal operations.

The ideal screening test should be simple, inexpensive, and widely available. Stair climbing meets these criteria and, even in the new millennium, remains a valid technique to screen for cardiopulmonary fitness. Patients who are able to climb five flights may be deemed suitable candidates for major surgery without further evaluation. Those patients who cannot or will not tolerate this level of exertion should be evaluated by directed techniques such as an echocardiogram or pulmonary function tests to more precisely determine the etiology and extent of their impairment.

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Emergency Department Visits in Asthma

Should All Be Prevented?

Each year, despite a wide and efficient array of asthma treatments, patients with asthma make approximately 1.8 million visits to emergency departments (EDs), with African Americans accounting...
defines undertreatment. This lack can be absolute or inferred source of care. The lack of steroid medication primary-care physician, viewed the ED as their pre-
cian involvement, because 69% of patients visiting the ED of Harlem Hospital Center, despite having a
ED visits because their asthma is very severe. In a population with different racial and economic
alties coping measures.8
To prevent most ED visits, we should apply a more aggressive treatment in patients with severe
asthma than indicated in the Guidelines for Diagnos-
isis and Treatment of Asthma.9 Also, we should improve self-care and bring together patients, phy-
jury, lack of a written asthma action plan, and various psychosocial attitudes interfering with avoid-
ance coping measures.8
The comprehensive Guidelines for the Diagnosis and Treatment of Asthma, which were published by
an expert panel,9 are essential reading for physicians involved in asthma care. Some have objected to their complexity10 or to the fact that the classification of asthma severity is not evidence-based.11,12 Because of their complexity, the primary-care physician may need to discuss with an allergist or pulmonologist some of the recommendations, in particular those pertaining to severe asthma.
At times, the classification of asthma severity in four steps9 seems disconcerting. At each step, there are characteristic clinical and physiological features that are assumed to occur simultaneously, but a footnote indicates that they may also overlap. I just saw a patient with continuous symptoms (severe persistent asthma) requiring daily use of a short-
acting inhaled β2-agonist and limitation of his activity (moderate persistent asthma), but with a peak expi-
atory flow of 80% of predicted (mild persistent asthma). According to the guidelines, the highest step defines the severity, in this case, the severe persistent step. However, the step would remain the same if all, not only part, of the manifestations would fit the criteria of severe persistent asthma. Most importantly for patients with severe asthma, the response to treatment is not used to stage severity of the disease. In the patient mentioned above, inhaled steroids could be started or doubled if already prescribed, but if the patient was already taking these drugs, he should probably go directly to the ED and be hospitalized. Not surprisingly, two stud-
ies, one carried out in a selected asthmatic population (Ford et al) and the other in a general asthmatic population,13 could not validate the parallelism between symptoms or between symptoms and FEV1 changes.13 Thus, the same step may be associated with a different constellation and intensity of symp-
toms, as well as different response to treatment (from none to some); it may require different new recommenda-
tions. Equivalent changes in peak expi-
atory flow and FEV1, which are tests of different physiologic sensitivity, could indicate different levels of severity. Also, the presence of wheezing and dyspnea at rest, symptoms with the highest positive predictive value for asthma,14 may have a different connotation in different patients. For instance, the attacks may appear spontaneously, without an obvious removable trigger, or may follow exposure to an avoidable allergic or nonallergic trigger. Their treat-
ments should be different, not the standard treat-
ment of any severe, persistent asthma.

The compact presentation of the guidelines did not allow the panel to point out the three key goals in the prevention and treatment of severe asthma exacerba-
tions: the improvement should be substantial, rapid, and monitored. This means therapy with more short-acting β2-agonists than had been inhaled be-
fore and, unless the exacerbation is mild, adminis-
tration of systemic steroids. The mode of adminis-
tration of the former should be by jet nebulization because equipotent doses delivered via metered-
dose inhaler are inconvenient for the patient during moderate and severe exacerbations. Jet nebuliza-
tion is recommended on page 38 of the guidelines, in the section on “home management of asthma exacerbations” on page 60, but not on page 62. The dose and dosing interval are not addressed. The recom-

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ing since it depends on many factors (eg, past exacerbations or baseline treatment). In moderate-to-severe exacerbations, the 2 mg/kg dose may work faster and more efficiently than the commonly recommended dose of 60 mg/day. At these steps, starting with high doses of inhaled steroids and then stepping down works faster than the step-up strategy. This approach is recommended by the guidelines (page 44), but the doses listed are half the equivalent of the 1,600 μg budesonide dose recently found to be effective in patients with poorly controlled asthma. It is also surprising that in patients with severe asthma, the guidelines recommend therapy with high-dose inhaled steroids (> 600 μg budesonide) but not systemic steroids.

We may prevent numerous ED visits, but many asthma specialists, including myself, feel that even the best treatment may leave some patients with poorly controlled asthma. Different asthma phenotypes with different types of inflammation might respond differentially to steroids. This poorly controlled subset and two additional subsets, one presenting with severe exacerbations and fragile general health and the other with a history of near-fatal asthma attacks (ie, high CO2 and/or intubation), should be directed to the ED.

Fatal and near-fatal exacerbations of asthma are frequent, but not invariably, associated with recent severe asthma, poor medical compliance, previous hospital admissions, and eventual intubation. Importantly, fatal or near-fatal asthma may have a rapid or a slow onset. These risk factors need to be promptly recognized. For the following reasons, the treatment needs to start immediately (particularly in rapid-onset exacerbations), aggressively, and in the ED: (1) death in asthma is due to asphyxia, and O2 treatment, closely monitored, may be life-saving; (2) the administration of β2-adrenergic drugs by continuous jet nebulization requires ECG monitoring in many hospitals; (3) the blood concentration of steroids peaks earlier if given IV rather than orally; and (4) the patients with severe asthma are not only unstable from a respiratory standpoint but also tend to have many comorbid conditions.

These examples show that even detailed guidelines cannot cover all situations and need clarification by a specialist. The current trend is to exclude the primary-care physician from the hospital and to replace him/her with a hospitalist, brought in primarily to discharge patients as fast as possible. This will leave the primary-care physician with mostly noninteractive sources of information (eg, books, articles, and guidelines).

In addressing asthma education, the guidelines advance an idealized proposal based on team effort with the principal clinician introducing the key educational message. However, today, at least in some parts of this country, patient education is assigned to registered nurses and respiratory therapists. They may not share with the treating physician the content of their message and inadvertently may contradict the physician’s recommendations. I will always remember Max Samter, the renowned allergist and editor of clinical immunology textbooks, who in the early 1970s periodically organized educational sessions at his home in Evanston, IL. I am sure that his patients, many of them from Chicago’s west side, where the University of Illinois Hospital is located, would have never considered the ED as their main source of care.

Identifying areas that can be improved reflects an optimistic attitude. Today, we have the means to treat severe asthma, safely, quickly, and selectively, and we also can enjoy what we are doing. To instill a perennial value in this attitude, I will borrow two Latin precepts and paraphrase another: *primum nil nocere* (first do not harm), *qui celer dat bis dat* (who gives fast gives twice), and *qui amat artem amat asthmann* (who loves the [medical] art loves asthma).

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Predicting Extubation Failure

Is It in (on) the Cards?

Determining readiness for liberation from mechanical ventilation (weaning) and the optimal technique to facilitate the process for patients who prove more difficult to wean is of considerable clinical relevance. Yet, once mechanical ventilation is no longer required, the clinician must address the separate question of whether or not the patient can tolerate removal of the endotracheal tube (eg, extubation). The process and outcome of extubation has received increasing attention among clinical investigators. Recent work clearly demonstrates that liberation and extubation are discrete processes with distinct pathophysiologic causes and unique outcomes.

Unsuccessful extubation (the need for reintubation) occurs in up to 20% of patients within 24 to 72 h of planned extubation. Factors that appear to increase the risk are the type of patient (eg, medical ICU), age > 70 years, higher severity of illness at weaning onset, use of continuous IV sedation, and possibly a longer duration of mechanical ventilation prior to extubation. Studies demonstrate that unsuccessful extubation is associated with increased hospital mortality especially for general surgical and medical patients. In addition, unsuccessful extubation significantly prolongs the duration of mechanical ventilation, ICU and hospital stay, and need for tracheostomy. The etiology of unsuccessful extubation influences outcome, with mortality lowest for airway problems (upper-airway obstruction, aspiration, excess pulmonary secretions) and highest when reintubation results from other reasons. Possible explanations to explain the high mortality seen with unsuccessful extubation include a sicker patient population, direct complications of reintubation, the adverse effect of prolonged mechanical ventilation, or clinical deterioration between extubation and reintubation. In contrast, Coplin et al recently demonstrated that brain-injured patients experiencing a potentially unnecessary delay in extubation experienced higher mortality and longer ICU stay when compared to patients expeditiously extubated.

Given the risks associated with extubation delay and those of unsuccessful extubation, what should an “acceptable” unsuccessful extubation rate be? Cardinal and colleagues recently addressed this issue by constructing a decision analytic model. These investigators found that there is no fixed acceptable probability of unsuccessful extubation. Of the factors studied, the rate of improvement in the patient’s condition (eg, the change in probability of tolerating extubation) over time had the greatest influence on the decision to extubate. When the rate of improvement was high, the best approach was to continue mechanical ventilation unless the probability of unsuccessful extubation was very low (eg, < 5%). Conversely, when there was little or no chance for further improvement, the best decision was extubation.

The frequency of reintubation and the adverse impact on outcome indicate that accurate prediction of extubation outcome is potentially important. Currently, clinicians often simultaneously assess patient readiness for liberation and extubation by conducting a spontaneous breathing trial (SBT) after the patient has demonstrated clinical recovery and his/her condition is hemodynamically stable. The optimal pre-extubation mode of ventilation (continuous positive airway pressure, T-piece, or pressure support) and duration of the SBT (30 to 120 min) has not yet been identified. Nevertheless, when extubation occurs without an SBT, the reintubation rate is prohibitively high.

Can physiologic measurements further improve