our ignorance of the cause. Cryptogenic fibrosing alveolitis, thus, became an accepted term in Europe and the British Isles. However, because the term interstitial pulmonary fibrosis is so entrenched, it is unlikely to be totally superseded by fibrosing alveolitis, even though this may be more accurate.

What then are we to do? Purists will not accept the term Hamman-Rich syndrome. Our colleagues across the Atlantic would prefer cryptogenic fibrosing alveolitis. However, on the North American continent, idiopathic pulmonary fibrosis continues to remain popular.

Here is an observation worth pondering. While going through William Osler's textbook of medicine, I came across the following paragraph:

In one of Charcot's cases. . . . death occurred about three months and a half after the onset of the acute disease and the lung was two thirds of the normal size, grayish in color, and hard as cartilage. In the only case of the kind which has come under my observation the patient died about a month from the onset of the chill. The lung was uniformly solid and grayish in color. Microscopically these areas showed advanced fibroid changes and great thickening of the alveolar cells . . .

This, indeed, is a perfect description of interstitial pulmonary fibrosis by Osler, more than 50 years before the publication of Hamman and Rich, from the John Hopkins Hospital. By calling idiopathic pulmonary fibrosis (cryptogenic fibrosing alveolitis) Osler-Charcot disease, we not only will be clearing up the semantic confusion, but also will be awarding credit to those who observed and described this entity first. I suggest that the entity, which has roamed so long in search of a title, be now duly crowned as Osler-Charcot disease.

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References

William Tell and Technology*

A 14th century legend recounts that William Tell was forced to shoot an apple from atop his son's head when he refused to pay homage to the governor. A man of great skill with nerves of steel, Tell was very much like our modern bronchoscopists. Armed with 20th century lances and as much concern for their patients as Tell had for his son, they also challenge the status quo. In this issue (see page 573) Schwartz et al describe the piercing of a bronchogenic cyst using sophisticated radiologic targeting to guide an endoscopically directed needle.

Bronchogenic cysts, like apples, are round structures usually found in the periphery of the tracheobronchial tree. About one-third are found near the major branches in the mediastinum. They may even fall off of the tree and land in the pericardium, esophagus, or the vertebral column. In infancy and childhood, these cysts commonly present with symptoms or signs of respiratory distress, dyspnea, or chest discomfort due to the mass effect. In adults, they are often asymptomatic, although they may present with infection, hemorrhage, perforation and, rarely, malignant degeneration.

Prior to the development of computerized axial tomography (CAT scanning) and the transbronchial needle, these lesions were suspect on conventional tomography because of their spherical shape, homogeneous density and position in the chest. The absence of more refined radiographic and endoscopic techniques led to the performance of major surgical procedures to obtain a diagnosis and occasionally to relieve symptoms. Combining the computerized axial tomogram with the transbronchial needle aspiration technique appears to be an ideal solution to the diagnostic dilemma posed by these congenital lesions. The low density readings often, but not always, found with CAT scanning raises our level of suspicion that we are seeing a bronchogenic cyst. Because these lesions seldom are malignant, it is probably unnecessary that they be removed unless symptoms are present or cytologic aspirates show malignant cells.

The uses of the transbronchial needle aspiration technique are expanding, as evidenced by the report of Schwartz et al. In the future, the transbronchial needle aspiration technique may serve not only our diagnostic needs, but may also be therapeutic. If a large enough volume of fluid can be removed from a bronchogenic cyst via needle aspiration, then symptoms related to the mass effect may disappear and surgery may be avoided. We must await reports of the feasibility of using the needle aspiration technique in the pediatric population.

The use of these new techniques requires the skill of a William Tell and intimate knowledge of the situation in which it is used. The analogy with William Tell must end here, for Tell promised that if he failed with his first arrow, the second would pierce the heart (of the governor).

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Sodium Bicarbonate during CPR
Does It Help or Hinder?*

It has been well established that acidemia prior to onset of cardiopulmonary arrest prognosticates unsuccessful resuscitation.1 The assumption that correction of acidemia by infusion of sodium bicarbonate improves the outcome is not documented. To the contrary, there is increasing evidence that this may be counterproductive.2-4 However, this may not necessarily apply to patients in whom acidemia follows the onset of arrest with cessation of ventilation and systemic perfusion. In this setting of catastrophic cardiac arrest, rapid onset of acidemia may be related to hyperventilation with hypercapnia and the accumulation of lactic acid due to critical reduction in oxygen delivery to tissues.

However, during cardiopulmonary resuscitation, increases in arterial PaCO2 are uncommonly observed during the initial 30 minutes when the patient is externally ventilated. To the contrary, arterial blood gas analysis usually discloses respiratory alkalosis. Under experimental conditions in the mechanically ventilated dog and pig, there is no significant acidemia during the initial ten minutes of CPR.5-7 Comparable observations have been made in human patients after witnessed cardiac arrest.8 Nevertheless, current practices support the routine administration of 1 mEq/kg of sodium bicarbonate as part of the initial life-support intervention and half of the initial dose at intervals of ten minutes.9 This intervention accounts for iatrogenic increases in plasma levels of sodium, hyperosmolality of plasma, and metabolic alkalosis. Both iatrogenic alkalosis and the hyperosmolar state following cardiopulmonary resuscitation have been associated with high mortality.10,11

More recently, the demonstration of marked hypercapnia in mixed venous blood and the paradox of arterial respiratory alkalosis and venous respiratory acidosis during the initial minutes of cardiopulmonary arrest and CPR have been confirmed in both experimental animal and human subjects.7-9 The administration of bicarbonate to experimental animal subjects further augments the severity of respiratory acidosis in mixed venous blood and, therefore, in tissue acidosis.12 In contrast, after the administration of the non-carbon dioxide generating organic buffer tromethamine, increases in the pH of both mixed venous and arterial blood are observed with a decrease rather than an increase in carbon dioxide tension in venous blood.

Finally, current studies indicate that the rationale of alkalization during cardiac arrest must be critically examined in terms of ultimate benefit. Bicarbonate-induced alkalosis, hypernatremia, hyperosmolality, and mixed venous hypercapnia are more likely to compromise rather than improve ultimate survival.10,11 Objective evidence fails to securely establish that beneficial effects of alkali, especially sodium bicarbonate, outweigh these risks.

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