tinued. But there is now a clear need for more decisive action on control measures and the Occupational Safety and Health Act of 1970 sets the stage for such action.

Application of the technics of industrial hygiene must be accelerated. The present threshold limit value for asbestos should be lowered far below some recent proposals. National administrative mechanisms to initiate and implement action are available — the recently established National Institute for Occupational Safety and Health and the National Advisory Committee on Occupational Safety and Health foremost among them. The Secretary of Labor, under the aforementioned Act, is charged with the setting of standards and has the right of entry into and inspection of places of employment. Thus, enforcement and regulatory mechanisms as well as advisory bodies have been established by law. Among the many problems that they face, asbestos should have high priority. Technologic developments are needed in many areas: sophisticated dust control; better and harmless asbestos substitutes; and improved monitoring of both the workers and their environment. Adequately devised tax policies could provide incentives for industry to clean the air and protect the workers. Such policies, by generating action in each plant where a risk exists, can rapidly and considerably amplify efforts on the national scale.

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ONE PRICE OF ACROPHILIA

MAN'S view of mountains has varied over the centuries. To the ancient Greeks and many Asiatics, high mountains were the inviolable abode of the gods; Renaissance man feared them as the home of demons. Ruskin praised their beauty from below, and 19th-century alpinists sought peace in walks and climbs among them. The current fevered century regards mountains as challenges to be overcome: courtship of beauty has been replaced by conquest of a foe . . . and a new "disease" has resulted.

In this impatient and highly competitive society it is not surprising that so many climbers hurry to "bag" their peaks. Not only are their acrobatics more incredible, but the speed with which ever higher altitudes are reached is quickening. Whereas Victorians experienced the same symptoms of mountain sickness as had been described in the 15th century, today's climbers have added a new entity to mountain sickness — high-altitude pulmonary edema.[1-3]

This "physiologic disease" is directly caused by hypoxia, and probably by hypoxia alone. The clinical and radiologic picture is classic and similar to edema of other causes. High pulmonary-artery pressures have been demonstrated[4] in individuals brought to hospital shortly after onset of severe high-altitude pulmonary edema (HAPE), developing as low as 11,000 feet. But why do not equally high pressures cause edema in other persons resident at altitude,[5] or in some patients with chronic heart or lung disease? Pulmonary hypertension is obviously not the sole cause. No chemical abnormalities have been identified. Hypovolemia and tissue dehydration contrast sharply with the waterlogging of the lungs: is the condition due to too great a shift of blood from peripheral to pulmonary circulation, perhaps resulting from peripheral vasoconstriction?[4] Cardiac output is reduced or normal, left atrial and pulmonary wedge pressures are generally low.[4,6] It is postulated that precapillary arteriolar constriction[4,7] in the lungs produces the condition — but how? The influence of local humoral changes and the contribution of the hypothalamus may be considerable — but have not yet been fully defined.[8] Hypoxia is known to increase capillary permeability, as may be manifest by the occurrence of retinal hemorrhage during hypoxia from any cause,[8] but the definitive studies remain to be done.

Whatever the pathogenesis, the condition has become increasingly common among people who go too high too fast, before the normal and marvelous acclimatization process can protect. The modern climber is usually in a hurry, flying from home to mountain base and thence rapidly by small plane to a landing above 10,000 feet. In the next few days he climbs — usually with a heavy pack — faster than adaptation can keep up, and trouble begins. His tickling cough becomes productive — often of frothy, bloody sputum. His companions hear his gurgles during sleep, he weakens and sinks to semi-consciousness sometimes in a few hours. Unless energetically treated early, he is likely to die.

Of course, not every dyspneic, coughing climber has pulmonary edema. Pneumonitis, pericarditis, hypertensive heart failure and other conditions probably account for half or more of the cases hastily called "pulmonary edema." As one examines the incidence among mountain climbers, one must be careful not to confuse the record by inaccurate diagnosis.

HAPE is a preventable disease — not an accident. Although physical conditioning does not prevent, a leisurely ascent does. The climber who