Alcohol and the Lung

It has long been suspected that ethanol may have deleterious effects on the lung, resulting in chronic pulmonary dysfunction. Individuals addicted to alcohol appear to be especially prone to respiratory infections, which are uncommonly severe and associated with unusual organisms. Dissuading people from the concept of alcoholic lung disease as a distinct entity has been the tendency to attribute lung disease in alcoholics to cigarette smoking, aspiration and general debilitation. However, there has been some evidence that alcohol per se can affect both metabolic and ventilatory functions of the lung.

Green and Kass\(^1\) in 1964 demonstrated that alcohol impaired bacterial clearance in experimental animals, while Guarneri and Lauretti\(^2\) showed that this effect was associated with impaired macrophage mobilization. In addition, alcohol in high concentration has been shown to impair mucociliary function.\(^3\)

Extrapolating from the effects of alcohol on hepatic lipid metabolism, Wagner and Heinemann\(^4\) hypothesized that similar alterations might occur in the lung. Working with rats, they demonstrated that prefeeding with alcohol interfered with incorporation of precursors into lung phospholipid. Whether such changes were associated with diminished surfactant production, or impaired lung function was not investigated.

In 1967, Burch and DePasquale\(^5\) took cognizance of the fact that alcohol metabolites accumulated in the lung and suggested the possibility of alcoholic lung disease as an analogy of alcoholic cardiomyopathy. In response to their editorial, Rankin et al\(^6\) performed spirometric tests on alcoholic patients and found a high incidence of obstructive disease which they attributed to smoking. Banner\(^7\) and Emirgil et al\(^8,9\) also found a high incidence of obstruction, but suggested that not all of the observed findings could be attributed to smoking. Asthmatic patients occasionally relate their attacks to alcoholic beverage consumption. Breslin et al\(^10\) attributed this effect to an immediate hypersensitivity reaction to congeners in alcoholic beverages since they could not document any effects from pure ethanol. Geppert and Boushey,\(^11\) however, demonstrated bronchospasm due to pure ethanol in a nonasthmatic woman. The mechanism of this effect could not be demonstrated.

Characteristic of the pulmonary dysfunction in alcoholics is diffusion impairment which cannot be explained solely on the basis of smoking.\(^7,8\) This diffusion impairment persists for at least three weeks following withdrawal from alcohol, but is less evident in patients who have remained sober for greater than six months.\(^9\) In the latter group, it is not known whether the diffusion abnormality resolves or whether this group represents a separate population who do not develop diffusion impairment.

In the present issue of Chest, Peavy et al (see page 488) have identified an acute effect of alcohol on diffusing capacity. The mechanism of the diffusion impairment is unexplained, but the authors suggest that the decrease in diffusion may be related to competitive inhibition of a specific carbon monoxide-oxygen carrier, cytochrome P-450. In support of their hypothesis the authors cite their previous work in lung and placenta, where they demonstrated saturation kinetics for CO transfer,\(^12\) a disproportionately high rate of diffusion for oxygen as compared to inert gases,\(^13\) and impairment of diffusion by drugs which interact with cytochrome P-450.\(^12\)

In support of such a mechanism for the effects of ethanol, it would have been helpful if the authors would have related the degree of diffusion impairment to the blood alcohol level both during the phases of absorption and elimination and to the effects of alcohol on the membrane and blood components of diffusion. If indeed such a mechanism for O\(_2\) transfer exists, it would be relevant to docu-
ment the clinical significance in patients, particular-
ly those with pre-existing diffusion impairment.

Even if facilitated transport of O₂ exists, competi-
tive inhibition would not account for the chronic
diffusion impairment noted in alcoholic patients,
since the diffusion defect persists despite sobriety
and is associated with mechanical changes in the
lung.⁷

To sort out the complex relationships of ethanol
ingestion and lung dysfunction, additional work
needs to be done in both experimental animals and
man to define the structural, biochemical and func-
tional sequelae of alcohol abuse.

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Heat Stroke

Modern Approach to an Ancient Disease

Heat stroke was originally described more than
2,500 years ago.¹ Although the clinical entity of
heat stroke has been recognized for many years, the
mortality remains significant—varying from 10 to 80
percent. Both the young and old are at risk. "Exer-
tion-induced" heat stroke is typically a disorder of
the young and healthy athlete or military recruit
training in a hot and humid environment. Classical
heat stroke is commonly a disorder of the elderly
occurring during environmental heat waves. These
latter individuals often have pre-existing disease
(atherosclerotic heart disease, diabetes mellitus, or
alcoholism) or are receiving medication (pheno-
thiazines, anticholinergics, sedatives, or diuretics)
known to predispose to the development of heat
stroke.

Heat stroke must be recognized as a medical
emergency. Inappropriate delay in diagnosis or
treatment may lead to death or irreversible organ
damage. Heat stroke should be suspected in any
patient with mental status changes accompanying
heat stress. The clinical criteria include exposure to
high environmental temperatures, rectal tempera-
ture exceeding 40.6⁰C (105⁰F), hot and dry skin,
and central nervous system disturbances.² The diag-
nosis of heat stroke should be made liberally as all
criteria may not be fulfilled.

Sprung et al.²⁸ and Costrini and associates⁶ have
recently described the hemodynamic and metabolic
alterations of heat stroke which are important for an
understanding of the disorder, its complications, and
treatment. Increased heart rate and cardiac output
and decreased systemic vascular resistance are nor-
mal circulatory adjustments required to dissipate
the heat load imposed by increased environmental
temperatures. These are the hemodynamic changes
that are most commonly observed in patients with exer-
tion-induced heat stroke.³ In contrast, most elderly
patients suffering from classical heat stroke manifest
a decreased cardiac output and increased peripheral
resistance.³ The etiology of these circulatory altera-
tions are not fully understood. When stressed by
heat, an elderly individual may be unable to com-
 pense with tachycardia and decreased systemic
resistance, and therefore, be predisposed to the de-
velopment of heat stroke. Circulatory failure in el-
derly patients has been shown to be secondary to
peripheral pooling of blood or hypovolemia.³ Most
patients require large volumes of intravenous fluids
to reverse their hypovolemia. Despite the inappro-
priately low cardiac output, patients do not develop
pulmonary edema if they are carefully monitored
during fluid resuscitation. Patients with bedside
right-sided heart catheterization have not manifest-
ed myocardial insufficiency or increased pulmo-
nary vascular resistance which are previously re-
ported causes of circulatory failure.²

Normal resting subjects will hyperventilate in re-