Respiratory Diseases in Relation to Changes in Atmospheric Pressure

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ABSTRACT

In this paper are reviewed the present status of respiratory diseases in relation to high and low altitude environments.

High Altitude Sickness

INTRODUCTION

Mountain sickness occurs in both an acute form and a chronic form. Only recently has this been studied to any degree; however, the first description was by a Peruvian in 1897. In 1937, Hurtado described a case of pulmonary edema in an Indian who became acutely ill after returning home from sea level to the high altitude. In 1945, a 39 year old man was examined who had developed pulmonary edema after going to a height of 11,550 feet. In 1949, a 29 year old man was described who died from an acute pulmonary illness in La Oroya, Peru, at an elevation of 12,250 feet. Lundberg, in 1952, described several cases of acute pulmonary edema. Mountain sickness or high altitude pulmonary edema occurs in individuals who quickly go from sea level to altitudes of 9,000 to 15,000 feet. Natives who have lived at high altitudes for a considerable time and then visit at sea level are most susceptible when they return to their mountain homes. Young children are also particularly susceptible under these circumstances. Most episodes in mountaineers occurred on their initial exposure to high altitude without proper acclimatization time. Men living at high altitudes have a higher total blood volume and a greater proportion of pulmonary blood volume than that present in sea level inhabitants. Persons going to high altitudes tend to develop greater blood volume in the pulmonary bed. Houston had reported mountaineers who have described cases of rapid death attributed to pneumonia. This occurred in healthy persons who were engaged in strenuous exercise over 14,000 feet. Death in 12 to 24 hours resulted if the symptoms were severe and remained untreated.

The period of acclimatization should be gradual in going to different altitudes and should last from 3 to 7 days. Persons who develop high altitude pulmonary edema have stayed at sea level any where from two days to two months. There seems to be an individual predisposition to high altitude pulmonary edema. During acclimatization, undue physical exertion should be avoided for the first 48 hours. Mountain sickness can be divided into two types, the
acute form or Soroche and the chronic form or Monge's disease. The onset of early symptoms varies from 6 to 36 hours after reaching a high altitude area. Most patients develop symptoms in the first 24 hours. The symptoms come on either during sleep or prolonged exercise. Infection is not a problem in the patient with the occurrence of high altitude pulmonary edema.

**Symptoms and Findings**

The early symptoms consist of headache, restlessness, increasing dyspnea, dry cough, palpitations, precordial discomfort, and nausea. Severe respiratory distress develops after several hours. This is followed by dyspnea, wet cough, foamy sputum, cold perspiration, anxiety, diffuse muscular and joint aching, thirst and sensory disturbances. Marked dyspnea may develop when lying down and is accompanied by cyanosis in severe cases. Most of the patients have marked facial pallor. On physical examination, the lungs are full of wet rales. Tachycardia is present and there is hypotension. Most cases have leukocytosis. Electrocardiographic findings are the same as in acute right ventricular strain. The average sinus tachycardia of 124 beats per minute was usually present. The tracing tends to show elevated P-waves and depression of the T-wave. After recovery, the electrocardiogram returns to normal and is similar to the tracings of other persons living at high altitude.

X-ray findings are usually that of spotty, mottled edema scattered through the lung fields with a tendency for the apices and bases to be free. The X-ray findings usually clear after 48 hours of treatment and the patients are clinically improved before the X-ray picture clears. The pulmonary artery may be prominent during an attack; however, the heart size remains unchanged.

The chronic form of mountain sickness occurs in Indian natives who develop an intolerance to high altitudes. This condition was first described in 1924 and the pathology has been described only recently. A 48 year old woman who lived at 14,300 feet developed a syndrome of right cardiac insufficiency, anasarca, and prominent cyanosis. Before her death, her hemoglobin was 21.6 g and the red blood count was 7.42 million. The anatomic findings of importance included: (1) severe right heart hypertrophy, (2) the right ventricle weighing 67 percent of the total ventricular weight, (3) a marked degree of muscularization of the peripheral pulmonary arteries, (4) multiple fresh and partially organized thrombi were present in the medium sized and smaller peripheral arteries and arterioles of the lung and (5) a moderate degree of muscular hypertrophy of the intermediate and proximal pulmonary arteries was noted. The pathogenesis of high altitude pulmonary edema has been studied for many years with gradual increase in the amount of available information.

**Etiology**

The cause is apparently a change in hemodynamics of the pulmonary vascular bed. Patients have pulmonary hypertension, but normal left atrial pressure. Hultgren has reported normal pulmonary capillary pressure and, therefore, it is considered that a contributing factor is vasoconstriction at a precapillary level. Increased capillary permeability must still be considered another contributory factor. This may aid the development of hyaline membrane described in the autopsied cases. The degree of anoxemia is more marked in newcomers to high altitude than in regular residents. The development of pulmonary edema while sleeping is considered a postural effect. Hurtado also noted a fall in the arterial oxygen while the patients were sleeping at high altitude. The increase in pulmonary blood volume is probably also a factor. The changes producing high altitude pulmonary edema appear to be a
magnification of the hemodynamic changes noted during rapid exposure to low temperature and hypoxia.

**PATHOLOGICAL FINDINGS**

The pathology of acute high altitude pulmonary edema has been reported by Arias-Stella\(^2\) with a mortality rate of 12.7 percent in a series of 86 cases. The autopsy findings described cyanosis of the face and anterior aspects of the thorax and hands. The trachea and bronchi were full of abundant seromucous secretion which was sometimes blood tinged. The lungs were heavy and did not collapse when the thorax was opened. There was abundant white, pink and foamy fluid exuding from the lungs. The right ventricular wall was thickened. Microscopically, alveolar edema alternated with emphysema in all lobes of the lung. Alveolar hyaline membranes were present as well as recent thromboses which were frequently seen in precapillary septal vessels. The pulmonary artery had thickening of the media and peripheral arterioles showed frequent muscularization. Cellular elements were very scanty in the edema fluid of the alveoli. The hyaline membranes were histochemically similar to those seen in hyaline membrane disease or in placental fibrinoid. These patients were not affected by primary cardiac nor pulmonary disease.

Children living at high altitudes have evidence of right ventricular hypertrophy by vectorcardiogram. An accidentally killed child showed, at autopsy, right ventricular hypertrophy and pulmonary arterioles with the prominent muscular wall.\(^{29}\) Sime et al reported right heart catheterization in 32 healthy children living at a high altitude of over 14,000 feet. The degree of arterial unsaturation in these children was the same as in adults at that altitude. Pulmonary wedge and right atrial mean pressures were normal. The child living at high altitude tends to maintain a pulmonary arterial structure of fetal type.

Hultgren gave the normal pulmonary arterial pressure as 13.8 ± 1.9 mm Hg.\(^{13}\) He considered that high altitude pulmonary edema was still of debatable etiology in 1971. Four hemodynamic abnormalities were cited which are consistently observed: (1) elevated pulmonary arterial pressure, (2) normal or decreased pulmonary artery wedge pressure, (3) an excessive degree of arterial desaturation, not corrected by 100 percent oxygen and (4) a normal or decreased cardiac output. Hultgren studied a group of individuals with right heart catheterization performed first at sea level and then, several weeks later, repeated at high altitude. The studies at high altitude were done following rapid ascent; expired samples of air and blood were examined at the same time. Exposure to high altitude produced a remarkable increase in the pulmonary artery pressure. Pulmonary artery pressure was considerably increased over wedge pressure, whereas at sea level they had been equal. Hultgren considered that there was a 3 to 5 fold increase in pulmonary vascular resistance. Increased oxygen inhalation partially relieved the elevated pulmonary artery pressure. During exercise, while breathing oxygen in high concentration, pulmonary artery pressure was significantly decreased. The stroke volume of cardiac output was reduced but the heart rate was increased at high altitude. Hultgren concluded that the patients who were prone to develop high altitude pulmonary edema tend to develop grossly abnormal pulmonary hypertension.\(^{13}\) This was due to increased pulmonary vascular resistance since pulmonary blood flow and pulmonary capillary wedge pressure were normal. The nature of the increased resistance is not known definitely. A portion of the resistance can be accounted for by hypoxic pulmonary vasoconstriction.

The development of the widespread fibrin deposits may also contribute to the development of edema. The peripheral
venous constriction has been shown to be exaggerated in persons susceptible to high altitude pulmonary edema.

**Treatment**

Treatment consists of prompt administration of oxygen by mask and bed rest. It is imperative that there be as little delay in treatment as possible. Supportive treatment with digitalis preparations and diuretics has not proven beneficial. Occasionally, if it is feasible, the patient must be removed to a lower altitude. The pulmonary congestion is usually gone in 24 to 48 hours and progress examination several weeks later reveals no evidence of residual symptoms nor signs.

**Decompression Sickness**

**Introduction**

Decompression sickness has been recognized for many years. Robert Boyle, in 1670, first recorded gas bubbles in tissues of animals. These were demonstrated by placing small animals in a decompression chamber and withdrawing the air. Decompression is also known as caisson disease and dysbarism. Workers call the disease the bends, the staggers or the chokes. The condition can develop during a transition from a high pressure to normal pressure or from normal pressure to low pressure such as encountered in high altitude flying. There are two types of the bends: Type 1, better designated as mild, and Type 2, designated as serious. The respiratory form is known as the chokes, the neurological form as the staggers and a circulatory manifestation has been known as the shocks. Minor effects, such as debilitating fatigue, rash, pruritus and paresthesias, may be experienced. The manifestations of decompression sickness are similar in the diver, the aviator and the tunnel worker, although the aviator is more prone to develop the chokes. Most of the symptoms develop within one hour after decompression. The most characteristic symptoms are pain, paresthesias, muscular weakness, rash and pruritus, vertigo, visual disturbances, paralysis, and dyspnea. A paradoxical response may occur with rapid deterioration during the recompression period. The pathogenesis of the condition develops from too rapid decompression with the development of intravascular gas bubbles. It has been noted, experimentally, that partial or complete constriction of muscular arteries following gas injection lasts several minutes. Next there is then a resulting vasodilatation and increased blood flow. The presence of gas micro-nuclei within the blood is recognized. These may be produced in areas of reduced hydrostatic pressure owing to muscular contraction. Workers regularly employed seem to develop acclimatization, and the frequency of bends decreases. This suggests the possibility that micro-nuclei may be dissipated by repeated pressure-decompression cycles. Several authors conclude that there is a large element of chance in the development of decompression sickness. Occurrence of pulmonary fat embolism is also observed with decompression. It is doubtful that this observed fat embolism is of serious consequence. Recent studies have shown that decompression sickness may occur in workers exposed to relatively low pressures in the range of 11 to 16 psi.

Workers who are to be examined for work in compressed air must be carefully examined according to requirements set up by various state agencies. Constant facilities for recompression and for first aid treatment must also be available 24 hours a day.

Behnke has noted that there is a marked increase in susceptibility to decompression sickness between the ages of 18 and 28 years. In addition, susceptibility to the illness increases with increase in amount of body fat. Serious problems may develop from pulmonary abnormalities. These include congenital cysts, scar tissue, vesicles and emphysematous bullae. Their presence...
may result in air embolism or pneumothorax. The workers must also be screened carefully to rule out those with obstructive lung disease. The intravenous bubbles contain mainly nitrogen, but occasionally they may contain oxygen.\textsuperscript{19}

Evaluation of the auditory tubes and auditory acuity is important.\textsuperscript{4} During compression, it is necessary to resort to voluntary procedures, such as the Valsalva maneuver, swallowing and yawning, to equalize pressure on both sides of the tympanic membrane. Upper respiratory tract infections impair the ability to accommodate to pressure changes. Dysbarism has produced little disturbance in hearing within the speaking range. This disease rarely causes deafness. Upper respiratory and nasal allergies predispose the workers to aerotitis.\textsuperscript{4} Experimentally, there has been a rather marked degree of hemoconcentration during decompression.

**Etiology**

Despite the evidence that intravascular bubbles are the initiating cause of decompression sickness, complete protection is provided by preoxygenation to affect nitrogen removal prior to rapid altitude ascent. Other associated complicating factors are fat embolism, liberation of proteolytic enzymes, potassium ions and peptides.\textsuperscript{4} Additional changes noted are vasoconstriction, loss of circulating plasma, hemoconcentration and the development of shock.

**Pathological Findings**

Aseptic necrosis of bone has been a serious and disabling complication of decompression sickness. Among the workers who built the East River Tunnel, there were 20 deaths. All the men in that construction were experienced workers.\textsuperscript{4} A marked increase in decompression time with resulting shortening of work hours has resulted in elimination of the serious disabilities. Tables from the United States Navy give much more decompression time than the tables from the British Ministry of Labor. The states of Washington and California give even greater length of times for decompression. The serious aseptic necrosis of bone is seen particularly about the heads of the humerus and femur. These bones, if involved, show areas of density in the head, neck and medullary shafts. Juxta-articular involvement leads to serious crippling complications. The adoption of the new decompression tables, as proposed by Sealey, has effectively eliminated this serious complication of compressed-air workers. The possible occurrence of a chronic neurological defect is extremely rare.

A decompression and prevention of decompression sickness are not necessarily related entirely to the physical condition of the worker. Haldane had proposed that the gas could be transported in a state of supersaturation in the circulating blood to the lungs. This probably represented the body’s tolerance of “silent” gas bubbles.\textsuperscript{4} Slow decompression, 10 to 15 minutes per foot of ascent, allows a low pressure head for diffusion of gases from tissues and subsequent transport in the blood. Transfer of oxygen from blood to the tissues results in a space or oxygen window, through which the inert gases can be transported from the tissues to the lungs. The inhalation of pure oxygen at elevated pressures is extremely useful in allowing this window to open.\textsuperscript{4} Behnke has proposed that prolonged residence in compressed air, not exceeding 14 psi, would solve most of the medical problems of this condition. A holding facility with pressures no higher than 12 psi would permit workers to leave after a relatively short period of time, if necessary. This system of control of decompression sickness, however, must also meet social obligations of the workers. Campbell has reported that certain agents, including fat scattering agents, narcotics, autonomic depressants and diuretics, are effective in preventing severe decompres-
Another possibility is that protective properties of these agents may be related to sludging of blood as described by Behnke. It is believed that the action of oxygen is that of a mechanical gradient allowing a more expedient elimination of nitrogen from the blood and tissues. Campbell and Spencer demonstrated that oxygen and theophylline synergistically enhance the elimination of nitrogen. During decompression, fluid replacement, sedation and cardiac supportive measures may be required. Aereolembolism should be treated by immediate recompression and oxygen.

References