High-Altitude Pulmonary and Cerebral Edema

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"In the next few hours his breathing became progressively more congested and labored. He sounded as though he were literally drowning in his own fluid, with an almost bubbling sound as if breathing through liquid. This occurred eight hours after onset of symptoms, 24 hours after arrival at 16,700 feet, and six days after leaving sea level."

"We had ridden mules to about 15,000 feet. After resting a day I went up to 17,400 feet and arrived in a tired but otherwise clear condition. The following morning I was comatose and my comrades carried me back down. I was comatose for approximately a day and a half, and subsequently was evacuated to the Army hospital. I did not have the usual warning signs, i.e. lung fluids and coughing."

MAKE no mistake: high-altitude sickness can strike anyone rash enough to go too high too fast. Even the experienced are not immune. High in the cold thin air, lack of oxygen exacts a price from everyone — and some pay heavily. But only recently have we begun to appreciate the importance of the problem — not only to climbers, but to travellers, soldiers, or anyone rash enough to rush to altitude.

The story of altitude sickness — including pulmonary edema, cerebral edema, retinal hemorrhage, and other less dramatic but unpleasant symptoms such as nausea, vomiting, weakness and headache — goes back only a few decades. In a sense acute altitude sickness is a modern disease — a disease of "progress", because only recently has it been possible for large numbers of people to go rapidly from low to high altitude. And it is the speed of ascent that determines the severity of the illness.

Granted that Acosta described the headache, nausea, vomiting and nosebleeds which occurred 400 years ago when Spanish Conquistadors climbed the Mexican volcanoes to get sulfur. Even before then Hannibal's men had trouble on the high Alpine passes — although there is no mention of whether or not his elephants were also affected! Almost all of the early Alpine climbers described symptoms on top of the highest peaks. Indeed not to mention the headache and nosebleed was almost a confession that one had not reached the summit! In 1925 Barcroft, that most delightful and observant mountaineering physiologist, described vividly and comprehensively the course of mountain sickness in the Andes, bringing up to date the encyclopedic observations by Paul Bert in 1876, and by Mosso twenty years later. Most observers commented that even healthy and experienced climbers had symptoms above 12,000 feet if they went up too fast.

We seem to have outgrown some of our susceptibility — or at least to have raised our ceiling. Nowadays many thousand people of all ages and degrees of fitness go rapidly from sea level to ski and climbing centers and within a day are skiing or climbing energetically above 12,000 feet — usually with only mild symptoms. But when the symptoms do occur, they can be severe:

"Twenty-four hours after leaving sea level he was skiing hard at 10,000 feet, despite headache, shortness of breath, and cough which increased for the next two days. On the night of the third day after leaving sea level his cough worsened, he had tightness in his chest and bubbling moisture in his lungs. He felt he might die of suffocation and was hospitalized and treated for asthma, growing steadily worse until the correct diagnosis of pulmonary edema was made. By the third hospital day he had recovered, and on the fifth day after hospitalization he returned to cautious skiing."

"Dr. B. began skiing hard at 8500 feet two days after leaving sea level, and within a day was short of breath and had a cough producing pink frothy sputum . . . Three days after reaching altitude he was semiconscious, extremely ill, and unable to walk. By the time he was taken down he was apparently dying. In hospital at 5000 feet, the diagnosis of pulmonary edema was easily made, treatment started, and he was discharged, well, on the fourth day . . . Three months later, taking four days to go from sea level to 8500 feet, he again developed such severe symptoms that he required helicopter evacuation; his pulmonary edema was again treated resulting in prompt and rapid recovery."

"For three days after flying from 2000 to 10,000 feet he carried 40 to 50 pounds progressively higher. On the fourth day, while climbing to 14,000 feet, he became very weak, developed a cough with profuse bloody sputum, and felt bubbling in his lungs. He was helped down to 10,000 feet where he improved after 24 hours of rest and medication, but remained weak and semi-comatose, requiring emergency helicopter evacuation. Both lungs were filled with pulmonary edema, and he had retinal hemorrhages in both eyes. He recovered rapidly without special treatment at 2000 feet."

The exact relationship between the mild headache, nausea and vomiting, and the accumulation of fluid in the lungs (pulmonary edema) or the staggering gait, severe headache, and rapid onset of unconsciousness (cerebral edema) is not clear. Our modern concept is that these and other symptoms are not directly due to oxygen lack but represent individual variations in the response to the bodily changes which oxygen lack inspires, since most of the symptoms of altitude sickness take hours or even days to develop. These changes — known as acclimatization — are profound and dramatic, and not completely understood. Occurring in hours, days, and some even taking weeks, they make up an integrated pattern which tends to bring the oxygen in the lungs closer to that prevailing at sea level, despite the lower oxygen pressure at altitude.

The healthy person who goes to altitude slowly adjusts; the changes take place at a slow, orderly, and for the most part asymptomatic pace. Thus the Himalayan climber can reach 28,000 feet or higher without oxygen, and without pulmonary or cerebral edema, though he will be weak, have headache, and lose his sea-level mental and physical agility. By contrast the aviator who flies — again without oxygen — from sea level to 15-18,000 feet in an hour or so will be light headed, dizzy, confused, and perhaps unconscious, all with a pleasant euphoria, similar incidentally to acute alcoholism! He does not develop pulmonary or cerebral edema, because he is incapacitated too fast!

Pulmonary edema hits the individual who reaches 10-14,000 feet by jeep, plane or mule, within a few days of leaving sea level, and then climbs higher without pause for acclimatization:

> *"The number of cases on Mt. Kenya has been steadily increasing: there have been four confirmed cases so far this year (November 1971). A 17-year-old school boy died in hospital after being carried off from 14,000 feet where he had been active for three days . . . Ten days later the secretary of the mountaineering club developed a dry cough during the night at 15,000 feet, and walked with difficulty down to 13,500 feet. The next day he collapsed and a doctor diagnosed pulmonary edema. He was carried off rapidly and recovered after three days in hospital . . . As far as we know there has not been a single case on Kilimanjaro which is almost 2000 feet higher. We believe the reason is the speed of ascent: on Kilimanjaro one starts walking at 5000 feet and most people take three days to get to 15,000 feet."*

> *"This case was noteworthy because he developed symptoms at such a low altitude (9000 feet) . . . He developed headache and light headedness . . . that night his breathing became bad and he could feel water in his lungs; his respiration tripled and he became cyanosed and edematous. The physicians who saw him after evacuation said he was still dyspneic and x-rays showed interstitial congestion in the lungs. The cardiac silhouette was normal suggesting that rather than acute cardiac failure this was strictly pulmonary edema. We have seen one or two of these cases each year, the most frequent cause being a lowgrade respiratory infection preceding the hike."*

Although some climbers undoubtedly developed pulmonary or cerebral edema many years ago, it was not until the mid-fifties that pulmonary edema was identified as a major risk of going to altitude rapidly, and several case reports were published in Spanish by Peruvian physicians. Then in December 1958 a healthy athlete became acutely ill during a ski trip across a 12,000-foot pass in Colorado. He was carried down on New Year's Eve by a slightly hung-over rescue party and in hospital was found to have, not the pneumonia anticipated, but classical pulmonary edema. Without obvious cause such as heart or lung disease, the cause of the edema puzzled consultants. The great cardiologist Paul Dudley White suspected altitude and urged that the case be published, which brought a flood of correspondence from around the world. The boy recovered within a few days and returned to skiing and climbing without trouble. This was the first note of the condition in English.

In the last ten years many papers have been published defining the now clearly recognized syndrome of high-altitude pulmonary edema, but its exact cause is still mysterious. The largest experience with acute altitude sickness came during the Sino-Indian war in 1962, when Chinese troops, thoroughly acclimatized by many months of residence above 16,000 feet on the Tibetan plateau, easily overran the Indian forces, the majority of whom were flown from the low plains to the high mountains in hours or days. Indian physicians have published papers describing several thousand cases of the different types of altitude sickness.

If — as seems likely from our present knowledge — many of the symptoms of altitude sickness are related, then do they have a common cause? The answer is a speculative yes. Our current information suggests that rapidly developing oxygen lack stimulates a series of hormone changes involving angiotensin, renin, aldosterone, and the antidiuretic hormone of the pituitary. The interplay of these hormones spurs striking shifts of fluids and electrolytes between cells and intercellular space and circulating blood which in turn affect kidney function and edema formation. The production of hemoglobin and red cells is increased. The cardiac rate and output increase. Respiratory exchange increases, resulting in an increase in the oxygen available through lung to blood, but a concomitant decrease in carbon dioxide — which among other things regulates the acidity of the blood. The decrease in carbon dioxide stimulates changes in the acid/base balance of the blood which are reflected by the cerebro-spinal fluid, and water and electrolytes shift within the brain itself. New vascular beds open, permeability of small capillaries may increase, allowing water and salts or even blood to escape (retinal hemorrhage) and the blood pressure changes in specific organs such as lungs — where the pulmonary artery pressure rises to high levels.

These intricate relationships are integrated so that one affects the others, and — if given sufficient time — the result is an accommodation to the lowered oxygen pressure which enables the acclimatized man to function almost normally at altitude up to 18,000 feet, though above 18,000 feet he is, as George Mallory put it, "a sick man walking in a dream" however long he may stay and however well he may acclimatize.

But our present concern is with the increase in pulmonary and cerebral edema among mountaineers. Though the numbers are not precisely known, it seems likely that 30 to 40 cases occur in North America each year, and twice as many more in other mountainous areas. About 20% die, usually because of slow recognition and treatment. All could be prevented.

The symptoms of pulmonary edema are: increasing weakness, shortness of breath greater than expected, irritative cough frequently with bloody sputum, and bubbling noises in the chest often audible to the victim and his companions. Breathing becomes more labored, the frothy sputum increases, pulse rate and temperature usually rise, the victim becomes unconscious, and may die within hours unless vigorously treated.

The most important treatment of pulmonary edema is to get down! Oxygen is often dramatically helpful. But even with oxygen the victim should be taken down as soon, as far, and as fast as possible. Medication is somewhat effective: the treatment of choice at this time is Furosemide (Lasix 40 to 120 mg by mouth or intravenously), which causes a great increase in urine and usually improves edema rapidly. This diuresis can lead to dehydration and shock, and therefore — paradoxical as it may seem — a high fluid intake is mandatory, including juices, tea, water, but relatively little salt. At this time no one is quite sure whether or not digitalis is helpful; morphine may be used, as it is in cardiac failure, but is probably unwise. Intravenous or rectal aminophyline, priscoline, and other unusual drugs are seldom advisable or necessary. The essence of treatment is early recognition — or at least suspicion — rapid descent, and diuretics.

> *"During the first six days after landing at 10,000 feet he carried loads to 16,000 feet in stages taking about two days for each successive 2000 feet. He seemed a bit slower than the rest and had a tremendous headache; there was more cyanosis of the lips . . . On the sixth day while packing to 16,000 feet he began to have double vision, spots before the eye and to hallucinate . . . He was promptly helped down to 10,000 feet, given a diuretic, and recovered a few days after plane evacuation".*

Cerebral edema — much rarer but more dangerous — usually begins with severe headache, double vision, hallucinations, disorientation, and

progresses to rapid loss of consciousness, paralysis, coma and death. It too can develop in a few hours. One healthy young man flown to 17,500 feet from sea level was acutely ill in 24 hours and comatose in 36; he recovered immediately on descent. Cerebral edema usually responds well to dexamethasone or betamethasone — both adrenal steroids — given by intramuscular or intravenous injection, but they take several hours to be effective, and they are not a substitute for prompt descent as soon as the condition is first suspected.

From what has been written above, it should be clear that prevention of altitude sickness is most desirable, and prevention depends largely if not entirely on slow ascent. Few healthy individuals develop symptoms below 10,000 feet, whereas most individuals will be sick above 15,000 feet if they go up rapidly. It is quite safe for most mountaineers to reach 10,000 feet within hours from sea level, and after a day or two at that altitude to climb to 14,000 feet at the rate of 1,000 feet or less each day. Above 14,000 feet, 500 vertical feet per day is fast enough. And above 18,000 feet all rules are off: the individual must find his own pace.

As every climber knows thirst is a usual companion in the mountains. At high altitude water is particularly important because increased breathing in the cold dry air sucks water out of the body, as does insensible perspiration. Add to this the slight nausea and loss of appetite so common at altitude, and the difficulty of melting snow to make every drop of water and you have the basic reasons for dehydration. Even at altitudes below 10,000 feet it is desirable to keep up a high fluid intake, but above 12,000 or 14,000 feet water is much more important.

Part of the acclimatization process is the loss of bicarbonate in the urine; the kidney must maintain a fairly good level of urinary output to do this efficiently. At the same time there are many hormone changes, and shifts of water within the body, which can lead to curious and apparently contradictory conditions. For example, it is not uncommon to find swelling of the face or ankles (edema due to fluid accumulation) in an individual who is seriously dehydrated and has a low circulating blood volume because his fluid intake has not kept up with urine output. One might think that in pulmonary edema — where an excess of fluid accumulates in the lung — the body would have too much water, but the reverse is true; dehydration is the rule in patients with pulmonary edema.

Consequently, it is crucial that high-altitude climbers drink more liquids than they feel like taking — two to three quarts per day is a minimum. It seems increasingly clear that failure to take in fluids increases significantly the signs and symptoms of mountain sickness. One is not likely to take too much!

Much has been written about the use of protective medication. Diamox (acetazolamide) has become popular as a chemical method of avoiding altitude illness. It is effective, and it seldom does harm. Its

value lies in its action as a carbonic anhydrase inhibitor, and not as a diuretic. Our present knowledge — admittedly imperfect — suggests that two or three Diamox tablets on each of the two days before and for one or two days after rapid ascent to above 10,000 feet reduces the symptoms and signs of altitude illness significantly. Larger doses are not helpful. Taking Diamox for more than three or four days is not beneficial, and may even be harmful, because the enzymes which counteract Diamox may actually increase the altitude problems. At the same time, even more water should be taken, because of the mild diuretic action of Diamox. It is important to note that Lasix (furosemide) which is not a carbonic anhydrase inhibitor but is a strong diuretic, is actually dangerous rather than beneficial in preventing altitude illness. Lasix, in combination with a great deal of water, is good *treatment* for pulmonary edema, but in our opinion a poor *preventive.*

Our current culture is oriented toward pills for everything, and it is understandable that the man in a hurry will turn to Diamox in an attempt to make his hurried climb free of symptoms or danger. So be it. I think it preferable to climb more slowly, but if this is impossible, keep a high fluid intake and take Diamox — together these are remarkably effective.

Addendum: A very large literature on altitude illness has accumulated. For the scientifically minded reader, a few of the better and most recent articles are noted below.*

SELECTED BIBLIOGRAPHY ON ALTITUDE SICKNESS

**Acute Mountain Sickness,* Singh, I., Khanna, P.K., Srivastava, M.C., Lal, M., Roy, S.B., Subramanyan, C.S.V., New Eng. Jour. Med. 280, 175, 1969. (The authors report clinical observations on 1925 individuals who experienced one or another form of acute mountain sickness in the Himalayan Sino-Indian war of 1962. The relationships between acute mountain sickness, cerebral and pulmonary edema and antidiuresis are discussed. Various forms of treatment are evaluated. This is by far the largest series of cases yet reported.)

Body Hydration and the Incidence and Severity of Acute Mountain Sickness, Aoki, V.S., and Robinson, S.M., Jour. Appl. Physiol. 31, 363, 1971. (Twelve subjects were exposed to 14,000 feet altitude for two days while given (a) placebo, (b) vasopressin or (c) furosemide. Hydration was determined by body weight, plasma volume and electrolytes, and end expiratory gas samples were also measured; severity of acute mountain sickness was determined by standard questionnaires. There appeared to be little relationship between hydration and symptoms.)

**Water Metabolism in Humans During Acute High Altitude Exposure,* Hrzywicki, H.J., Consolazio, F., Johnson, H.L., Nielsen, W.C., and Barnhart, R.A., Jour. Appl. Physiol. 30, 806, 1971. (Two groups of six men were studied before and after six days at 14,000 feet. Total body water was significantly decreased; extra-cellular water increased; and intra-cellular water decreased. Heavy physical exertion was performed before and during the altitude exposure; hypo-hydration and diuresis occurred, suggesting that water loss may have been an adaptive mechanism to acute altitude exposure.)

Mountain Sickness: A Cerebral Form. Fitch, R.F., Ann Int. Med. 60, 871, 1964. (A case report of severe cerebral edema occurring at 16,400 feet during a mountain expedition is presented with a review of relevant literature.)

Circulatory Dynamics during High Altitude Pulmonary Edema. Penaloza, D., and Sime, F., Amer. Jour. Cardiol. 23: 369, March 1969. (Cardiac catherizations were done on two young males who developed pulmonary edema on return to 14,200 feet after a brief sojurn at sea level. Severe hypoxemis, marked pulmonary artery hypertension and increased pulmonary vascular resistance were associated with low cardiac output and low pulmonary wedge pressure. Pulmonary hypertension was significantly reduced after breathing 100% oxygen. The authors suggest that arteriolar constriction at the precapillary level contributes to the condition but cannot explain the rapid occurrence of edema in the absence of elevated pulmonary wedge pressure.)

**High Altitude Pulmonary Edema.* Singh, I., Kapila, C.C., Khanna, P.K., Nanda, R.B., and Rao, B.D.P., Lancet 229 (Jan 30, 1965) (Clinical observations are reported on 332 men who developed pulmonary edema at altitudes above 11,000 feet following rapid ascent. The condition occurred in 15.5% of persons reaching altitude for the first time, and in 13.0% of those returning after a stay at low altitude. A schema is presented suggesting that pulmonary edema, cerebral edema and acute mountain sickness are various reactions to the same underlying physiologic response to hypoxia, and a number of mechanisms are proposed. Treatment with aminophylline, atropine, morphine and digoxin is discussed; the latter apparently ineffective. Failure of oxygen to relieve hemoglobin desaturation is noted.)

**High Altitude Pulmonary Edema.* Hultgren, H.N., Spickard, W.B., Hellriegel, K., and Houston, C.S., Medicine: 40: 289, September 1961. (This early article discusses eighteen patients with pulmonary edema studied at 12,000 feet in the Peruvian Andes and notes thirteen suggestive cases occurring in mountaineers. The literature to date is reviewed and mechanisms examined; acute left ventricular failure was considered the most likely cause despite the absence of left atrial enlargement, but treatment of cardiac failure was not of clear value. Fifteen of the eighteen persons studied in the Andes developed the condition after re-entry to altitude to which they were fully acclimatized, raising the possibility that such persons are more vulnerable.)

Acute Pulmonary Edema: Pitfalls in Diagnosis and Treatment. Altschule, M.D., Medical Counterpoint: July 1969. (the author presents a variety of causes for pulmonary edema and points out the efficacy or failure of various treatments in relation to the underlying physiologic mechanisms involved. He notes that pulmonary edema is not invariably or even consistently associated with left ventricular failure.)

**One Price of Acrophilia.* Houston, C.S., New Eng. Jour. Med. 285; 1318, 1971. (The rising incidence of pulmonary edema among mountain climbers is noted and the importance of adequate fluid intake, slow ascent, and prompt treatment is reviewed. The relationship to retinal hemorrhage from hypoxia is mentioned.)

**Acute Pulmonary Edema of High Altitude.* Houston, C.S., New Eng. Jour. Med. 263: 478, 1960. (A case of acute pulmonary edema occurring in a healthy skier at 12,000 feet is reported, together with brief reviews of four similar but less well documented cases among mountaineers. Though the mechanism was unknown, the combined effects of anoxia, cold, and exertion were suspected as causative. This is the first report of high altitude pulmonary edema published in English.)

**Effect of Acetazolamide on Acute Mountain Sickness.* Forwand, S.A., Lansdowne, M., Follansbee, J.N., and Hansen, J.E., New Eng. Jour. Med. 279, 1, 1968. (In a double blind study, either placebo or acetazolamide was given to 43 subjects rapidly transported to 12,800 feet for five days. Significant reductions were observed in the most prominent symptoms of acute mountain sickness, but the mechanism of action was not identified.)

**Control of Acute Mountain Sickness.* Gray, G.W., Bryan, A.C., Frayser, R., Houston, C.S. and Rennie, I.D.B., Aerospace Medicine 42:81, 1971. (Clinical trials with placebo, acetazolamide and furosemide were conducted on subjects rapidly transported to 17,500 feet. Acetazolamide conferred significant benefit whereas furosemide appeared to exacerbate symptoms.)

A Hypothesis Regarding the Pathophysiology of Acute Mountain Sickness. Hansen, J.E., and Evans, W.O., Arch. Env. Health: 21:666, 1970. (Acute mountain sickness is a syndrome of unknown etiology which occurs, after a time lag, following abrupt exposure to oxygen lack and which includes a characteristic spectrum of symptoms. The author has collected animal and human evidence to suggest that a causal relationship exists between brain compression and symptoms, and advances several theories as to mechanism.)

Neurologic Manifestations of Chronic Pulmonary Insufficiency. Austen, F.K., Carmichael, M.W., and Adams, R.D., New Eng. Jour. Med. 257:579, 1957. (Three cases of headache, papilledema and impaired consciousness attributed to the hypoxia and hypercapnia of pulmonary insufficiency are presented and the mechanism discussed.)

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Ocular Changes in Pulmonary Insufficiency. Spalter, H.F. and Bruce, G.M., Tr. Am. Acad.Ophthal and Otol. 661, July 1964. (Sixteen patients with pulmonary insufficiency due to cystic fibrosis of the pancreas are reported and compared with another group of patients with chronic pulmonary insufficiency from other causes. The retinopathy is attributed by the authors to carbon dioxide retention and acidosis; the literature is reviewed and photographs of the retinal pathology presented.)

**Retinal Hemorrhage at High Altitude.* Frayser, R., Houston, C.S., Bryan, A.C., Rennie, I.D., and Gray, G., New Eng. Jour. Med. 282, 1183, 1970. (Nine out of twenty-five individuals taken to 17,500 feet were found to have retinal hemorrhage. Of seventeen of these persons who went up rapidly six had hemorrhage; of eight who climbed slowly to altitude, three showed hemorrhage. Incidence of retinal hemorrhage was unrelated to any other symptoms of altitude sickness, but seemed to be reduced in the nine individuals premedicated with acetazolamide. This is the first report of retinal hemorrhage in healthy individuals exposed to high altitude hopoxia.)

The Response of the Retinal Circulation to Altitude. Frayser, R., Houston, C.S., Gray, G., Bryan, A.C., and Rennie, I.D., Arch. Int. Med. 127, 708, 1971. (Studies of individuals rapidly and slowly taken to 17,500 feet showed that retinal blood flow increased by 89% over control values within two hours, and by 128% over control after four days following rapid ascent. Retinal blood flow increased by 105% over control values in acclimatized subjects. Both arterioles and venules show increased diameter and tortuosity beginning a few hours after arrival at altitude.)

Pulmonary Edema of High Altitude. Viswanathan, R., Jain, S.K.,and Subramanian, S., American Review of Resp. Dis. 100; 342, 1969. (In a series of three reviews the authors examine the production of edema in animals, the clinical and hemodynamic features, and the pathogenesis, and conclude that the condition is due to abnormal hypoxic response in susceptible individuals who manifest a genetically determined condition by greater than normal precapillary vascular resistance in lungs, leading to pulmonary artery hypertension. The mechanism whereby edema results is not explained.)

Haemodynamic Studies in High Altitude Pulmonary Edema. Roy, S.B., Guleria, J.S., Khanna, P.K., Manchanda, S.C., Pande, J.N., and Subha, P.S. British Heart Journal 31; 52, 1969. (Six individuals who developed pulmonary edema at altitudes above 13,000

feet were thoroughly studied at 12,000 feet in the Himalayas; all showed elevated pulmonary artery pressure but normal pulmonary capillary, left atrial, and ventricular filling pressures. Cardiac output was reduced in three subjects; pulmonary blood volume was above normal in two.)

The Preterminal Arterioles in the Pulmonary Circulation of High Altitude Natives, Recavarren, S., Circulation 33; 177, February 1966. (Quoting the work of others, the author speculates that increased pulmonary artery muscularization correlates, in individuals fully acclimatized by long residence at altitude, with the susceptibility to pulmonary edema by increased capillary hydrostatic pressure, increased capillary permeability, and points out that normal capillary pressures have been observed after edema has appeared.)

High Altitude Pulmonary Edema, Menon, N.D., New Eng. Jour. Med. 273: 66, 1965. (The author reports his clinical observations of 101 individuals developing pulmonary edema above 11,000 feet in the Himalayas. The condition was twice as common among re-entrants as among first arrivals; it was not increased by exertion or infection. Intravenous digitalization produced prompt improvement suggesting that some myocardial failure was present.)