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ACUTE LEFT VENTRICULAR FAILURE

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Acute left ventricular failure, characterized by an agonizing struggle for air, is terrifying to the patient, family, and physician. The seizure is distinctive, therefore most contemporary descriptions are similar. Probably the first account of this syndrome was that of Aretaeus of Cappadocia.[1] His words paint a picture that has been little improved upon in the 2000 years since his death. In patients with paroxysmal nocturnal dyspnea, "the cheeks are ruddy; eyes protuberant, as if from strangulation; . . . voice liquid and without resonance; a desire of much and of cold air; they breathe standing, as if desiring to draw in all the air which they possibly can inhale; and, in their want of air, they also open the mouth as if thus to enjoy the more of it; pale in the countenance, except for the cheeks, which are ruddy; sweat about the forhead and clavicles; cough incessant and laborious; expectoration small, thin, and cold, resembling the efflorescence of foam; neck swells with the inflation

of the breath; the praecordia retracted; pulse small, dense, compressed; and if these symptoms increase, they sometimes produce suffocation, after the form of epilepsy."

Following in the footsteps of Aretaeus, many students of heart disease (White,[2] Weiss,[3] Ernstene,[4] Blumgardt,[5] Smith,[6] Robb,[3] Wolferth,[7] Harrison,[8] Prinzmetal,[9] Christie,[10] Hope[11]) have written of the mechanism and treatment of this syndrome in recent years.

The large number of reports belies the relative infrequency of this state, but, on the other hand, emphasizes its impressive nature. With the exception of 250 patients reported by Palmer and White,[2] 82 by Weiss and Robb,[3] and 40 by Ernstene and Knowlton,[4] most other reports include few patients.

The term "acute left ventricular failure" indicates that the left ventricle is subjected to disproportionate strain because of hypertensive vascular disease, coronary artery disease, or valvular defect. Thus, in Weiss and Robb's[3] report of 82 cases, 53 per cent had hypertension, 20 coronary sclerosis, 20 syphilitic aortitis, 6 rheumatic heart disease. Ernstene and Knowlton[4] observed a similar distribution.

Few manifestations of underlying cardiac disease more certainly herald early death, particularly when the attacks occur more often than once a day and are associated with gallop rhythm or pulsus alternans.[2] The serious import of the syndrome is indicated by the average duration of life after the first attack. Palmer and White[2] found that of 250 patients, 59 were dead within the first six months, and an additional 69 within the first two years.

It seems worthwhile to re-examine the published evidence and add to it our experience gathered in the past years at the Indianapolis City Hospital and more recently at Cleveland Clinic.

Analysis of Patient Records

Twenty patients with acute left ventricular failure were observed (table). Seven of the 20 had essential and 13 malignant hypertension. One patient had six attacks and finally died of a cerebral hemorrhage. Most of them had only one or two attacks, and one was immediately fatal.

Four patients while ambulatory were observed to have paroxysmal auricular tachycardia at some time during the course of the disease. The records of 10 others showed the occurrence of extrasystoles frequently enough to have impressed the patient or the examiner. No irregularities were recorded in the remaining 6.

The average duration of life after the first attack was 1.3 years. Only 2 patients are still alive—1 with malignant hypertension has lived thirty-

six months and 1 with essential hypertension has lived eighteen months following such symptoms.

Treatment

The table indicates the type of treatment used to control the attacks. The usual management follows:

1. The nurse or relatives are instructed to assist the patient to a sitting or, if possible, standing position.
2. Sixteen milligrams of morphine sulfate is administered subcutaneously or, if possible, intravenously.
3. If bronchospasm is particularly severe, 0.5 Gm. of aminophylline in 10 cc. of saline solution given intravenously will often produce striking relief. This drug can be combined in the same syringe with the morphine.
4. If the patient is still in distress, the venous return to the heart should be reduced by withdrawal of 600 to 800 cc. of blood. However, the inconvenience of this procedure and the liklihood of coexisting anemia make bloodless phlebotomy more practical. This procedure, first advocated by Chrysippus of Cnidos about 380 B.C., is the application of tourniquets to all four limbs. If blood pressure cuffs are used, inflation to a point just greater than diastolic pressure is usually effective; if these are not available, constriction adequate to occlude venous circulation is necessary. This will pool as much as 1700 cc. of blood in the venous bed of the extremities. Treatment should be continued until symptoms are relieved. Caution should be exercised in removing the tourniquets lest the sudden return of large volumes of blood to the circulation cause more ventricular strain.
5. If the patient has not yet been digitalized this should be done as soon as the acute attack is controlled. The oral route of administration is satisfactory.
6. The patient should be kept on a maintenance dosage of digitalis following an attack. Moderate restriction of sodium from the diet accompanied by adequate amounts of water by mouth and the occasional use of mercurial diuretics are helpful in preventing further attacks.

Discussion

The mechanism of acute left ventricular failure must account for the characteristics implied in the term "paroxysmal nocturnal dyspnea", which is often used to describe it. The abrupt onset is probably its most distinguishing characteristic, yet the most difficult to explain. Unpleasant dreams, poorly tolerated position during sleep,[14] prolonged coughing,[15] deep breathing, sudden skeletal movement, increased blood volume,[16] pulmonary engorgement,[17] and early congestive heart failure[3]

have all been suggested as precipitating agents. However, the stereotyped nature of this phenomenon and the frequent absence of one or many of these stimuli offer meager support for any one of them as the sole excitant.

The nocturnal occurrence of attacks is less difficult to understand. Weiss and Robb[3] were able to find evidence of pulmonary engorgement and early fluid retention in all of their patients with this syndrome. That the recumbent position places the pulmonary circulation at a further disadvantage is evident from the observations of Hamilton and Morgan,[18] who showed that the lungs then serve as a reservoir for the blood of the extremities.

Not only is the lung capacity less while the patient is in the recumbent position (Hurtado and Fray[19]), but the vital capacity is decreased by 26.7 per cent, as demonstrated by Christie and Beams[10] in their study of the effect of position on the vital capacity of patients with heart disease.

Any or all of these factors could effect the abnormal physiologic processes responsible for the signs and symptoms of acute left ventricular failure, provided a trigger-mechanism capable of creating a sudden decrease in left ventricular output and resulting increase in pulmonary engorgement were present. That this actually can be true was demonstrated by Ernstene and Lawrence,[20] who observed a typical attack in a person with a large thrombus occluding the mitral valve. The explosive nature of the attack strongly suggests that most of them might have their inception within the wall of the abnormal left ventricle. It is well known that showers of ventricular extrasystoles and paroxysmal auricular tachycardia are not unusual among patients with left ventricular disease. These recurrent arrhythmias can temporarily decrease left ventricular output. With the conditions that have been shown to exist in the pulmonary circuit when a person with heart disease sleeps, any slight change in the output of the left ventricle could well upset the precarious relationship between right and left ventricular output so that pulmonary edema ensues.

As with other suggestions, this concept must remain theoretical until electrocardiographic evidence of such arrhythmias immediately preceding acute attacks can be obtained. However, it is noteworthy (table) that of the 20 patients observed, 4 had recurrent paroxysmal auricular tachycardia while ambulatory and 10 had frequent showers of extrasystoles. These observations were made in retrospect, and the others could have had irregularities not significant enough to record prior to the attack. It is also of interest that the adequate use of digitalis after acute left ventricular failure is helpful in preventing future attacks, as well as in controlling cardiac irregularities.

Summary

1. There is abundant evidence that pulmonary engorgement accompanies the recumbent position and sleep in patients with left ventricular heart disease.

2. Because 14 of the 20 patients reported here had paroxysmal cardiac irregularities while ambulatory, the suggestion is offered that if this occured during sleep, left ventricular output might be sufficiently decreased to precipitate attacks of paroxysmal nocturnal dyspnea.

3. The treatment found most useful in our hands includes:

(a) immediate assumption of sitting or upright position,
(b) intravenous morphine and aminophylline,
(c) bloodless phlebotomy,
(d) digitalization and the use of diuretics as prophylaxis against future episodes.

Table

Summary of Treatment and Course of 20 Hypertensive Subjects Who Had Acute Left Ventricular Failure

Age	Diagnosis	No. of Attacks	Treatment	Duration of life after first attack		Cardiac Irregularity
				living	dead	
56	Essential Hypertension	3	Sitting position Morphine Tourniquets		12 mo.	Extrasystole
36	Essential Hypertension	2	Sitting position Morphine Tourniquets		20 mo.	Extrasystole
42	Malignant Hypertension	3	Standing position Morphine		3 mo.	Paroxysmal auricular tachycardia
48	Malignant Hypertension	2	Standing position Morphine		15 mo.	Paroxysmal auricular tachycardia
44	Malignant Hypertension	1	Standing position Morphine		16 mo.	Extrasystole
29	Essential Hypertension	2	Sitting position Morphine		20 mo.	Extrasystole
7	Malignant Hypertension	1	Sitting position Morphine Tourniquets		4 mo.	Paroxysmal auricular tachycardia

Summary of Treatment and Course of 20 Hypertensive Subjects Who Had Acute Left Ventricular Failure—Continued

Age	Diagnosis	No. of Attacks	Treatment	Duration of life after first attack		Cardiac Irregularity
				living	dead	
54	Malignant Hypertension	6	Sitting position Morphine Tourniquets		17 mo.	Extrasystole
49	Malignant Hypertension	2	Standing position Morphine	36 mo.		Paroxysmal auricular tachycardia
46	Malignant Hypertension	1	Standing position Morphine		8 mo.	Extrasystole
38	Malignant Hypertension	1	Standing position Morphine		13 mo.	
42	Essential Hypertension	2	Sitting position Morphine Phlebotomy		12 mo.	
45	Malignant Hypertension	1	Sitting position Morphine Tourniquets		16 mo.	
51	Malignant Hypertension	1	Sitting position Morphine Tourniquets		1 mo.	
53	Malignant Hypertension	1	Sitting position Morphine Tourniquets		1 mo.	Extrasystole
35	Malignant Hypertension	2	Standing position Morphine		2 mo.	Extrasystole
39	Essential Hypertension	3	Sitting position Morphine Tourniquets		24 mo.	Extrasystole
22	Essential Hypertension	2	Standing position Morphine		16 mo.	Extrasystole
45	Essential Hypertension	1	Standing position Morphine	18 mo.		
46	Essential Hypertension	2	Sitting position Morphine Tourniquets		20 mo.	

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