CARDIAC FAILURE SECONDARY TO INEFFECTIVE BELLOWS ACTION OF THE CHEST CAGE

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Baldwin, Cournand, and Richards (1) have described the following classification of pulmonary insufficiency:

1. Ventilatory
   a. Restrictive, viz., pulmonary fibrosis, kyphoscoliosis.
   b. Obstructive, viz., pulmonary emphysema.
2. Alveolar-respiratory
   a. Distributive, viz., pulmonary emphysema.
   b. Diffusional, viz., pulmonary scleroderma.

The two cases to be presented demonstrate ventilatory insufficiency of the restrictive type due in the first instance to primary neuromuscular dysfunction and in the second to chronic pleural disease with resulting calcification and restriction of the underlying parenchyma and diaphragm. Of particular interest was the development of congestive heart failure in both of these patients. These cases represent examples of cardiac failure secondary to an ineffective bellows action of the chest cage without significant intrinsic pulmonary disease.

CASE REPORTS

Case 1

This 34 year old white male was well until 1945 when he noted the gradual onset of weakness of the right side of the face. A few months later he began to have bilateral jaw weakness, and difficulty in swallowing food with regurgitation of fluid through the nose. In 1947 there was onset of occasional diplopia. He was admitted to this hospital on three different occasions between January 1947 and November 1948 and on all these admissions showed signs and symptoms of bulbar involvement. These signs and symptoms persisted with little change except for development of dysarthria. In November 1949 he complained of difficulty in maintaining erect posture and he was readmitted for his fourth and final admission. At this time the patient stated he had noted dyspnea on exertion for the preceding one and a half years and during the month before admission, cyanosis of the lips had become apparent.

On admission the patient appeared chronically ill without respiratory distress but with cyanosis of the lips, nailbeds, and skin. There was a marked dysarthria. His chest cage moved very little on normal respiration but he had good expansion with effort and could clear most of his cyanosis with forced breathing. The heart was not remarkable on examination except for a markedly accentuated P2. The lungs were clear to percussion and auscultation. The abdominal examination was within normal limits. Neurological examination revealed the right pupil to be larger than the left, and both reacted sluggishly to light and accommodation. The external ocular movements were normal but he had diplopia in all fields. The disc margins were slightly blurred nasally and he had small hemorrhages about the discs. His gait was normal. There was weakness of the jaw, right facial, palatal, tongue, neck, and shoulder muscles with fasciculations noted in these groups. In addition there was atrophy of the masseters, platysmae, tongue, neck and shoulder muscles. There were a few fasciculations of the triceps muscles but no weakness or atrophy. The deep tendon reflexes of the arms were two to three plus bilaterally and there was greater finger stretch on the left than on the right. The abdominal reflexes were diminished bilaterally. There was no atrophy or weakness of the legs and the sensory examination was normal.

Laboratory examinations including complete blood count, urine analysis, blood chemistries, spinal fluid, serology, chest X-ray, skull plates, and electrocardiographs were all within normal limits.

The patient's neurological status remained unchanged while under observation and adequate nutrition was maintained with tube feeding. Two months after admission a medical consultation was requested because of increasing cyanosis. At this time marked cyanosis was observed without respiratory distress. Expansion of the chest cage during ordinary breathing was minimal but with forced inspiration he was capable of a three inch chest expansion. The lungs were clear and examination of the heart was unremarkable except for an accentuated second pulmonic sound. Abdominal examination was negative. There was no peripheral edema or venous engorgement. The red blood count was 6.75 million with a hemoglobin of 19.4 grams and a hematocrit of 64%. Fluoroscopic examination revealed the trachea to be in the midline and the motion of the bony framework was good. There was normal radiolucency of the lung fields. The range of motion of the right diaphragm was one

inch; that of the left diaphragm one-half inch. There was no diaphragmatic paralysis. The lung fields were clear. Examination of the heart revealed slight left ventricular rounding, moderate right ventricular enlargement, and marked enlargement of the pulmonary conus. The electrocardiograph demonstrated evidence of right ventricular hypertrophy. Venous pressure was 180 mm. of citrate and decholin circulation time 28 seconds. Because of these findings, the patient was digitalized. There was a five pound diuresis, and the venous pressure and circulation time returned to normal. Following this the patient was studied in the cardiopulmonary laboratory (Table I). Early in June 1950, despite vigorous therapy, he went into intractable congestive heart failure and died.

Autopsy examination revealed the following pertinent findings. There was clubbing of the fingers and moderate subcutaneous edema. The lungs showed a moderate degree of congestion with occasional scattered areas of chronic pneumonitis in the lower lobes, probably on an aspirational basis. There was no gross or microscopic evidence of significant emphysema or fibrosis. The heart

was enlarged weighing 500 grams. The coronary arteries, myocardium, endocardium and valvular leaflets were normal. The right ventricular chamber was dilated and the right ventricular wall hypertrophied measuring 1 cm. in thickness. The pulmonic valve ring was dilated measuring 10 cm. in circumference. The right auricle was slightly dilated as was the tricuspid valve ring which measured 14.5 cm. in circumference. The left atrium and mitral valve were normal. The left ventricular wall was slightly hypertrophied to approximately 1.5 cm. in thickness. The aortic valve was normal in size and appearance. The pulmonary arteries showed mild atherosclerotic changes. The gastrointestinal tract, biliary system, genitourinary tract, spleen, pancreas, adrenals, thyroid, lymph nodes, and skeletal system were all normal. There was chronic passive congestion of the liver, spleen and kidneys. The liver weighed 1,750 grams and the spleen 220 grams. The central nervous system, including brain and spinal cord, showed no distinctive lesions on multiple sections with different staining techniques including Mahon's. The peripheral nerves were normal.

Despite the negative autopsy findings, it was the opin-

TABLE I

RESULTS OF PULMONARY FUNCTION STUDIES IN CASES 1 AND 2

Case 1

		Predicted	Observed	
1	Lung Volumes			
	Vital Capacity (lying) c.c.	4770	2285	
	Residual air, c.c.	1180	1005	
	Total capacity, c.c.	5950	3680	
	$\frac{R.A.}{T.C.}$ %	20	.27	
	Alv. N_2 %	<2.5	1.77	
II.	Maximum Breathing Capacity, L/min	119	38	
III.	Basal Ventilation/min L/M^2 B.S.A.	± 3.20 0.65	3.33	
IV.	Respiratory Gases in Arterial Blood			
		Rest	Rest	Exerc.
	CO_2 content vols. %	± 48.5 4.5	64.4	72.4
	O_2 saturation %	± 96 2	73.3	65.5
		Room air		
	pCO_2 mm. Hg.	40	84	
	pO_2 mm. Hg.	95	49	
	pH	7.4	7.2 (calc.)	
V.	Calculated Alveolar-Arterial Gradient on Room Air	0 to 12	Normal	

TABLE I (Continued)

CASE 2

I. Lung Volumes

	Predicted	Observed
Vital Capacity (lying) c.c.	3800	900*
Residual air, c.c.	1220	880
Total Capacity, c.c.	5020	1890*
$\frac{R.A.}{T.C.}$ %	24	42
Alv. N_2 %	less than 2.5	less than 2.5

II. Maximum Breathing Capacity L/min.   90   38*

*Results inaccurate because of poor cooperation.

III.

	Rest Predicted	Observed	Exerc. Predicted	Observed
Ventilation/min. L/M² B.S.A.	3.20 ± 0.65	6.45	8.70 ± 0.81	12.98
O_2 Intake in cc/M² B.S.A.	129 ± 13	161	480 ± 74	390
O_2 Removal in cc/L ventilation	46.8 ± 7.1	30.4	54.8 ± 6.3	35.8

IV. Respiratory Gases in Arterial Blood

	Rest Predicted	Observed	Recovery
CO_2 content vols. %	48.5 ± 4.5	58.3	54.7
O_2 saturation %	96 ± 2	92.3	84.6
pCO_2 mm. Hg.	40	69	
pO_2 mm. Hg.	95	66	
pH	7.4		

V.

	Alv. pO_2 mm. Hg.	70
	Art. pO_2 mm. Hg.	66
Room	Art. pCO_2 mm. Hg.	69
air	Alveolar-arterial gradient	4
breathing	Dead space ml.	193
	Dead space % tidal air	56
	ph calculated	7.27
	Alv. pO_2	118
High	Art. pO_2	105
oxygen	Alveolar-arterial gradient	13
breathing	Dead space ml.	178
(29.25%)	Dead space % tidal air	55
	ph calculated	7.27

ion of the neurological service that this patient's clinical picture could best be explained by ascribing the changes to amyotrophic lateral sclerosis.

Case 2

A 56 year old Puerto Rican male was admitted to the hospital because of increased dyspnea and orthopnea for three weeks, and a non-productive cough for a few days. In 1921 and 1925 the patient had "pleurisy." In 1936 he was told that he had "calcifications in his chest." In 1946 he was refused employment because of an abnormal chest X-ray. Three years before admission there was an episode of wheezing and undue shortness of breath which lasted a few days and then ceased spontaneously. During the two years prior to admission there was ankle edema. Because of the increase in dyspnea the patient sought hospitalization.

At the time of admission the patient's temperature was 103° F., pulse 116. He was orthopneic and cyanotic. Examination of the chest disclosed poor expansion bilaterally with more limitation on the right. There was dullness over the lower half of the right lung field and at the left base with diminished breath sounds and tactile fremitus over these areas. There were moist rales at the left base with occasional wheezing over the right upper lung field. The second pulmonic sound was accentuated. The liver edge was two finger breadths below the right costal margin, and tender. There was two plus pretibial edema.

The blood count on admission revealed a normal white blood count and differential. The red blood count was 6.0 million, hemoglobin 18.5 grams, and the hematocrit was 62%. An X-ray of the chest disclosed extensive calcification surrounding the lower two-thirds of both lung
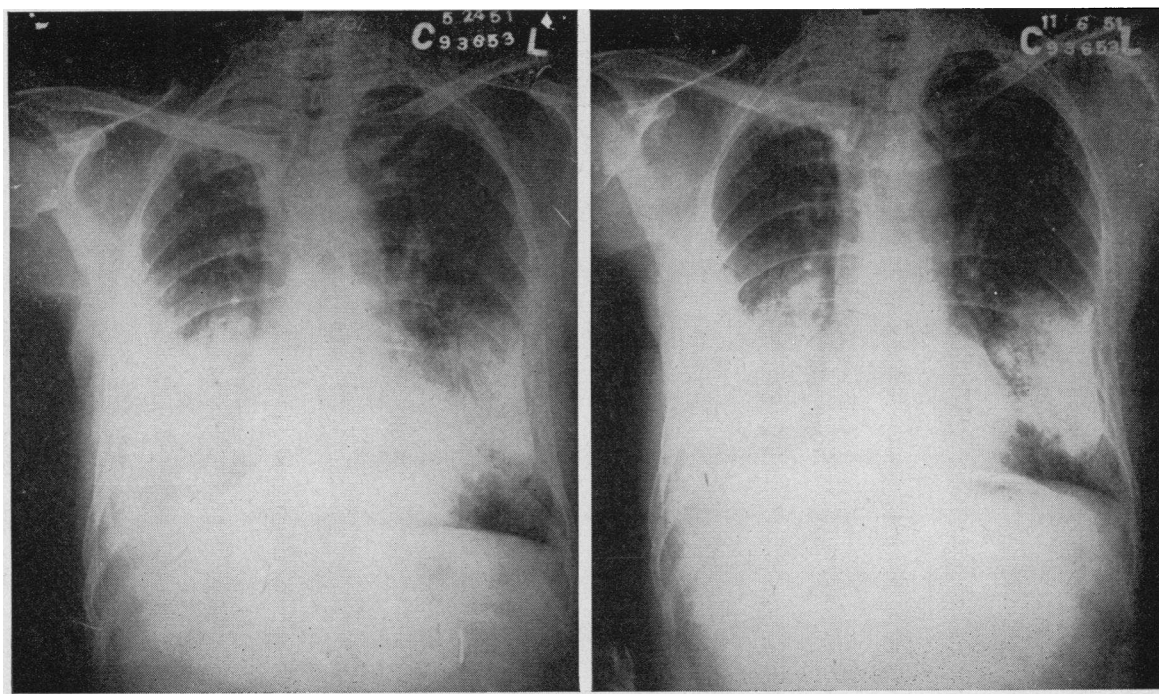
FIG. 1A. CASE 2 DEMONSTRATING CALCIFIED PLEURITIS AND CARDIAC ENLARGMENT BEFORE THERAPY
FIG. 1B. AFTER THERAPY DEMONSTRATING RETURN OF HEART SIZE TO NORMAL

fields and an increase in the transverse diameter of the heart (Figure 1A). An electrocardiogram was consistent with right ventricular hypertrophy. The venous pressure on admission was 280 mm. of citrate.

The patient was treated with penicillin and bed rest for the first five days with no change in the clinical course.

On the fifth hospital day the patient was digitalized, placed on a low salt diet, and oxygen was administered intermittently by mask. Later that day he became semi-stuporous. He was placed in an oxygen tent and given mercurial diuretics. Shortly thereafter he became comatose although the cyanosis diminished. The stupor per-

TABLE II

	pO_2 mm. Hg.	O_2 Capac. vols. %	O_2 Sat. %	pCO_2 mm. Hg.	CO_2 cont. vols. %	ph
6/1/51 Room air immediately after oxygen tent with narcosis	26	22.2	39.2	96	59.3	7.14
6/4/51 Breathing room air	55	20.1	87.6	57	62.4	7.40
6/6/51 Respirator with nasal oxygen	98	19.7	98.9	68	61.1	7.30
6/12/51 Respirator with nasal oxygen	147*	20.2	100	51	59.4	7.42

*Inaccurate, above range of applicability of bubble technic.

sisted until the patient was removed from the oxygen tent for routine care the following day. Arterial blood studies suggested that carbon dioxide narcosis was responsible for the coma (Table II). In order to combat the CO_2 narcosis, artificial respiration with the Pneumatic Balance Resuscitator was initiated. Within a short time the patient was more alert and there was no cyanosis. A phlebotomy was also performed. Artificial respiration was discontinued later that day, and the patient maintained satisfactory clinical progress for the next 48 hours. The blood gas studies reflected this improvement. Although the patient had improved considerably, oxygen therapy was again administered because of the anoxia disclosed by the arterial blood studies. Artificial respiration was reinstituted by means of a Drinker-Collins respirator, and oxygen was administered by nasal catheter. This was continued for one week along with penicillin, bronchodilators and continued digitalis therapy, phlebotomy, low salt diet, and occasional mercurial diuretics. At the end of this period the patient was relatively asymptomatic and the signs of congestive heart failure had disappeared. Follow-up X-rays disclosed that the heart size had returned to normal (Figure 1B). Fluoroscopy revealed the pleural calcifications, no motion of the rib cage, and restriction of diaphragmatic motion. Complete pulmonary function studies were performed one month after recovery from the acute episode (Table I). Three months after discharge the patient was seen in the Follow-up Clinic. He was asymptomatic and capable of climbing two flights of stairs. Digitalis therapy and a low salt diet were continued.

METHODS

The measurement of residual air volume was done using an open circuit method in which the nitrogen of the lungs is washed out by continuous inhalation of pure oxygen and collected over a period of seven minutes. An index of intrapulmonary mixing was obtained by sampling of the alveolar air at the end of this period (1).

The measurements of lung volumes and maximum breathing capacity were obtained using the spirographic technique (1).

Arterial blood samples were obtained from the brachial artery using an indwelling Cournand-type needle. The blood was collected by the technique described by Riley, Proemmel and Franke (2), using heparin to prevent clotting and a small globule of mercury in the syringe to facilitate mixing. The oxygen content and capacity and the carbon dioxide content were determined on duplicate samples using the Van Slyke-Neill apparatus (3). All samples checked within 0.2 volume %.

The arterial blood tensions of oxygen and of carbon dioxide were determined directly in mm. of mercury, using the direct method of Riley (2). Duplicate measurements were made using two Roughton-Scholander syringes, and all samples checked within 2 mm. of mercury. Expired air samples were collected in duplicate simultaneously with the blood specimens. Oxygen intake and carbon dioxide output were calculated from the percentage

of oxygen and carbon dioxide found in these expired air samples, as determined in a Scholander gas analyzer (4). The duplicate measurements checked within 0.04%.

Arterial blood samples and expired air were collected under basal conditions with the patients breathing room air, high, and low oxygen concentrations, each for twenty minute periods when indicated. Finally oxygen saturation was determined after one minute of standard exercise and the expired gas during the exercise was collected in a Douglas bag and analyzed. During the five minute recovery period the expired air was collected and analyzed. The expired air, except during exercise, was collected in the Tissot apparatus and the spirogram attached permitted calculation of the respiratory rate and tidal air under the various conditions. The alveolar-arterial gradients were calculated from the data obtained according to the method of Lilienthal, Riley, Proemmel and Franke (5).

Values for pH were calculated from line charts of the Henderson-Hasselbalch equation (6).

COMMENTS

Case 1 disclosed a markedly reduced vital capacity, total capacity and maximum breathing capacity. The residual air and alveolar nitrogen after breathing pure oxygen for seven minutes were normal. The ratio of residual air to total capacity was slightly but not significantly increased and reflected the decrease in total capacity. Thus the lung volume studies revealed principally a reduction in vital capacity which was due primarily to the marked limitation of diaphragmatic excursion. The normal residual air and alveolar nitrogen indicated the absence of any significant degree of emphysema. During the performance of the maximum breathing capacity an element of fatigue was apparent in that the patient was unable to maintain the depth or speed of ventilation uniformly for the 12 second interval. This was probably due to muscle anoxia and weakness associated with chronic disease. In addition, a satisfactory depth of respiration could not be established presumably as a result of impaired diaphragmatic excursion. Finally an adequate speed of ventilation could not be established because of muscular weakness and poor coordination. The total basal ventilation was normal but the effective alveolar ventilation was grossly inadequate as reflected by the high arterial pCO_2. Despite the presence of strong stimuli for hyperventilation (high arterial pCO_2 and low arterial pH and pO_2) the resting ventilation was normal. This would suggest decreased sensitivity

of the respiratory center to chemical stimuli. In addition, the polycythemia may have been an aggravating factor (7). The inadequate alveolar ventilation resulted from the fact that too large a proportion of the total ventilation was wasted in ventilating the dead space as a result of the shallow breathing. It is of interest to note that this patient's resting ventilation was less than 10% of his maximum breathing capacity which would account for the absence of dyspnea at rest in the presence of severe anoxemia (1). The arterial CO_2 content was elevated but not commensurate with the pCO_2 so that the pH was decreased. The arterial pO_2 and oxygen saturation were markedly reduced and the latter decreased even further after standard exercise. These changes were due solely to inadequate alveolar ventilation. The calculated alveolar-arterial gradient on room air, assuming an RQ of 0.8 to 0.9, was normal. Because of the patient's muscle weakness he was unable to keep a mouthpiece in place for prolonged periods of time, and the RQ of 0.8 to 0.9 was therefore assumed.

Case 2 demonstrated marked decrease in all lung volumes. The observed figures for the vital capacity, total capacity and maximum breathing capacity probably were inaccurate since the patient did not appear to cooperate fully during the performance of these tests. The ratio of residual air to total capacity was high due to the fact that the total capacity reduction was out of proportion to that of the residual air. The maximum breathing capacity was reduced. These changes were primarily due to the chronic constrictive calcific pleuritis which interfered with the bellows action of the chest cage. The high ratio of residual air to total capacity was not a reflection of emphysema since the alveolar nitrogen was less than 2.5%. The tracings showed no graphic evidence of a prolonged expiratory phase. There was no evidence for impairment of gas exchange. Ventilation studies demonstrated that the patient was hyperventilating at rest and during exercise, presumably as a response to the high arterial pCO_2 and anoxia. This patient's calcific constrictive pleuritis markedly reduced his effective alveolar ventilation. Thus, his respirations were rapid but shallow and probably did little more than wash out the dead space. This caused a high arterial pCO_2 and CO_2 content and anoxia. There was no indication of significant abnormalities of gas exchange since

the gradient studies were within normal limits (5, 8).

The patient was admitted with an acute pulmonary infection. This was enough to accentuate the anoxia and thereby increase the pulmonary hypertension and precipitate cardiac failure. He was treated with antibiotics, bronchodilators, digitalis, phlebotomy, and oxygen. The use of continuous oxygen therapy in patients with chronic anoxia has been known to produce CO_2 narcosis (9–13) and this patient was a vivid example of this phenomenon (Table II). The Pneumatic Balance Resuscitator probably represented a life saving procedure at this time. The use of the respirator permitted continuous use of oxygen therapy without the threat of the return of CO_2 narcosis. This will be discussed in another paper (14).

DISCUSSION

We have presented two patients whose primary difficulty was a defective bellows action of the chest cage. In the first case this was due to disease of the neuromuscular system. In the second case the patient had extensive calcific pleuritis of undetermined etiology which interfered with the mobility of the thoracic wall. In the latter instance there undoubtedly existed some degree of pulmonary
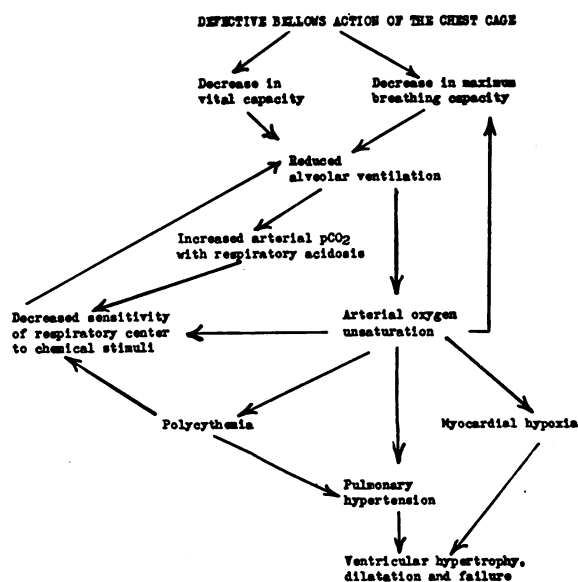

FIG. 2. SEQUENCE OF EVENTS WHICH MAY HAVE OCCURRED IN TWO CASES WITH AN INEFFECTIVE BELLOWS ACTION OF THE CHEST CAGE

disease, probably on a tuberculous basis. In view of the entire clinical picture and pulmonary function studies, we are inclined to believe that parenchymal disease contributed little if any to the abnormalities observed.

In both these patients (Figure 2) the defective bellows action resulted in a reduction in vital capacity, maximum breathing capacity, and ineffective alveolar ventilation manifested by a high arterial pCO_2 and arterial oxygen unsaturation. The anoxia was probably a stimulus for the production of the polycythemia (15). The hypercapnia, anoxia, and polycythemia all represented factors favoring medullary center damage which these patients demonstrated. There is evidence to indicate that both polycythemia (7) and anoxia (16, 17) may lead to pulmonary hypertension and eventually cardiac failure which these patients also manifested. In both these patients the normal changes in circulatory dynamics associated with the mechanics of respiration were undoubtedly altered (18). No objective studies were made to evaluate these factors.

Finally, it is of interest to correlate the situation in primary polycythemia with the sequence of events in these two cases. In the former instance there is polycythemia which we believe may lead to hypoxemia (7). In the latter we have ineffective bellows action of the chest cage leading to hypoxemia and secondary polycythemia. Evidently, once polycythemia and hypoxemia have occurred, the subsequent tendency to develop congestive heart failure probably depends upon similar mechanisms, *viz.*, myocardial hypoxia and pulmonary hypertension.

SUMMARY

Two cases of ventilatory insufficiency of the restrictive type resulting in congestive heart failure have been presented. There was no evidence of significant intrinsic pulmonary disease. The sequence of events leading to heart failure are discussed.

ACKNOWLEDGMENTS

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