Respiratory Alkalosis
with Especial Reference to Chest Pain

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Respiratory alkalosis may be defined as a state of altered physio-
chemical processes due to the excessive loss of carbon dioxide through
overbreathing. Though this condition is usually referred to as the hyper-
ventilation syndrome, respiratory alkalosis would be more accurate termi-

nology as hyperventilation may occur as a necessary physiologic require-
ment without producing an alkalotic state.

That the syndrome of respiratory alkalosis (hyperventilation syndrome)
is being recognized with increasing frequency is attested by the numerous
reports appearing in the literature. Detailed discussions of the biochemical
and clinical changes incident to overbreathing may be found in the reports
of Stead and Warren,1 Engle, Ferris and Logan2 as well as Ferris3 and his
group. It is not within the scope of this paper to discuss all of the facets
of the syndrome but rather to stress some of the clinical findings which
are believed to be important in the production of symptoms.

The major biochemical change occurring in this condition is the alka-
losis produced by the reduction of plasma carbonic acid through excessive
loss of carbon dioxide. Thus the alkalosis is relative rather than absolute.
Ferris3 and his co-workers demonstrated that a single deep breath could
reduce the carbon dioxide arterial concentration 5-7 volumes per cent and
the carbon dioxide tension as much as 16 mm. of mercury. Symptoms of
respiratory alkalosis therefore may develop rapidly and depending on the
rate and volume of breathing they vary markedly from time to time. Indeed
the symptoms of respiratory alkalosis are so varied and bizarre that a
clinical pattern is difficult to recognize. With experience and awareness
however, the condition becomes manifest.

As has been pointed out by Lewis4 and others, not all cases of respiratory
alkalosis are psychogenic in origin but it seems agreed that the great
majority of people who hyperventilate unnecessarily do so as an expression
of an emotional disturbance regardless of the presence or absence of
organic disease. Thus a cardiac patient may complain of shortness of
breath, pains in the chest and “poor circulation” as a result of his alkalosis
rather than his heart disease. The clinician familiar with the syndrome of
respiratory alkalosis will usually have little difficulty in detecting the true
nature of the complaints.

The earlier articles on this subject described the acute overt attack of
hyperventilation with severe symptoms, the patient being in an alarming
state bordering on, if not in, tetany. While this type of case is seen

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occasionally it is the more chronic case that is seen every day in the physician's office. Many of these patients have only two or three features of the syndrome at the time of examination but nearly all will have experienced the complete syndrome at some time in the past. It is not unusual to obtain a clearcut history denoting alkalotic symptoms for many years. Two almost constant complaints in chronic respiratory alkalosis are a sense of exhaustion and ease of fatigue. On questioning these patients two points serve to distinguish their exhaustion from that of organic disease. (1) The exhaustion is greatest in the morning and tends to improve progressively during the afternoon and evening. (2) The ease of fatigue as well as the exhaustion almost always improves after a period of moderate exercise. This improvement is due to the increased production and repletion of carbon dioxide following exercise.

A disturbed sense of balance (“the blind staggerers” of the Negro patient) is frequently felt by these patients. This sensation is intermittent and tends to occur in a particular environment. The acute episode is more likely to take place in close crowded spaces such as are seen in busses, theaters and elevators. Tachycardia, faintness, shortness of breath and a sense of impending disaster are often felt along with the disturbed balance.

Chest pain occurs in a large proportion of both the acute and chronic cases. This is usually, though not invariably, in the lower half of the left chest and may radiate to the neck, shoulder or to the finger tips. Often the pain will be in the scapular region and occasionally it is felt in the axilla or lower substernal regions. The pain may be intense, sharp, frequently burning in character; or a mild constant dull ache. It may occur as a fleeting twinge when lying on the side especially the left side, or bending over. When located substernally it is difficult to distinguish this distress from true angina. A useful point in differentiation is that the pain of respiratory alkalosis is often altered by holding a forced inspiration; occasionally this relieves the pain but more often it aggravates it. In almost every case moderate to severe tenderness is found on movement of the xiphoïd tip.

In most of the cases of respiratory alkalosis who complain of chest pain, some alteration of the diaphragm may be found. With fluoroscopy it is common to see a large accumulation of air in the stomach or splenic flexure, with considerable elevation of the left leaf of the diaphragm. This leaf lags on downward excursion either with or without the air bubble. The most extreme examples are seen in cases of paralysis of the left phrenic nerve and the symptoms of indigestion, chest pain and shortness of breath following such paralysis have long been recognized. While everyone swallows air to some extent, with an atonic left diaphragm, hypomotility of the stomach with mild pylorospasm delay the air in the stomach. Tenderness over the interspaces in the region of the precordium is an almost constant finding. Stewart Wolf reported distinct changes in the behavior of the diaphragm under conditions of stress. Decreased motility of the diaphragm has also been noted during hyperventilation.
The diaphragm is attached anteriorly to the xiphoid and fascia of the transverse abdominal muscles. Laterally it arises from the costal cartilages of the lower six ribs and the bony portions of the last three ribs. Posteriorly the attachments are to the three upper lumbar vertebrae.

Thus the location, radiation and type of pain along with the tenderness over the rib and xiphoid attachments as well as the frequent findings of the elevation and lag on fluoroscopy all seem to point to the diaphragm as the chief source of pain in respiratory alkalosis. (These findings are commonly seen in the early pneumoperitoneum patient whose diaphragm is beginning to rise.) It may be assumed that the pain originates in the tendinous insertions through prolonged stretching and atony of the diaphragm. A similar type of pain and soreness is produced in the tendon attachments of the skull in tension headaches.

Though not all patients with respiratory alkalosis exhibit pain in the chest it is possible that some patients may hyperventilate as a direct result of the upward dislocation and stretching of the left diaphragm through distention of the gastric fundus or the splenic flexure of the colon. In many of these cases the upward displacement of the diaphragm is enough to compress the left lung to a degree sufficient to alter the normal respiratory reflexes and produce overbreathing. Stretching and atony of the diaphragm may account for the sensations of air hunger so often felt by these patients.

The diaphragm rises in the reclining position through the pressure of the abdominal wall and viscera; it is not unusual to find the tympanitic note of a large gastric air bubble well above the fourth anterior rib in this position. With the increased stretching of the diaphragm incident to its rise in the reclining position, undoubtedly overbreathing occurs during sleep, and so the symptoms tend to be more prominent in the morning. Often hyperventilation is the presenting symptom in cases of diaphragmatic hernia, the symptoms being relieved when the hernial sac is empty.

Treatment

Though much has been written about this syndrome very little has been said about treatment. Most authors have stressed paper bag rebreathing, reassurance of the patient by reproduction of symptoms and psychotherapy. Calcium gluconate has been condemned as having no rational place in treatment. Very rarely one will see mention of acidulation with ammonium chloride or ammonium nitrate. The fact is no trustworthy plan of relief has been found and the physician must largely improvise his own regime. A simple non-technical explanation of the condition after a thorough, unhurried history and physical examination is a necessity. Routine laboratory work and, if the patient is heart-conscious, an electrocardiogram present negative evidence to strengthen your diagnosis. To the skeptical patient reproduction of symptoms through forced overbreathing is at times convincing but all too frequently acute symptoms occur which are unlike the complaints and serve to undermine rather than increase confidence. The demonstration of a large gastric air bubble on a chest
x-ray or at the time of fluoroscopy is very convincing evidence. The fact that a positive diagnosis rather than "just nerves" has been made affords the anxious patient a great deal of comfort and relief. Though no defect in calcium metabolism has been attributed to respiratory alkalosis, parenteral calcium gluconate in my experience often gives dramatic relief for several days. Calcium salts are probably effective solely through their ability to produce both smooth and striated muscle relaxation thereby reducing the spastic elements which are so prominent in the production of symptoms.

Ammonium chloride given to tolerance (usually about 5 grams a day) affords relief to some. Mild sedation is of considerable help even in the exhausted cases. Avoidance of sodium bicarbonate, fruit juices and other alkalinizing agents is strongly recommended but other dietary restrictions seem unnecessary. Holding a deep breath to the breaking point is many times effective in breaking the symptom cycle particularly in crowded places. Dizziness, numbness and tingling are effectively controlled by re-breathing in a paper bag to dyspnea or by inhaling the fumes of a small block of dry ice (solidified carbon dioxide).

Perhaps the one most effective item in treatment is the institution of regular outdoor exercise in the form of long walks. This is difficult treatment to enforce because of the ease of fatigue and chronic exhaustion. It is explained that exercise releases tension, replenishes the carbon dioxide and strengthens the diaphragm. With these strong arguments most of these sufferers will at least take a few trial walks. It is gratifying to both the patient and physician to see what a few days of long walks will accomplish in relief of symptoms.

Case Reports

Case 1: A 65 year old widow was seen March 1940 with a severe acute anterior myocardial infarction. Following a stormy illness during which she had pulmonary edema and congestive failure she returned to a normal though somewhat restricted life. Her blood pressure was stabilized at 150/110 and she had no return of failure at any time. She had few complaints until January 1952 at which time she developed a constant dull aching over the precordium not related to exertion. The pain was worse at night and was partially relieved by sleeping on several pillows. Nocturnal leg cramps, smothering sensations, numbness and tingling in the extremities and tachycardia occurred almost every night. In the mornings she felt thoroughly exhausted and was worn out after cooking breakfast. By 4 P.M. she felt perfectly well. Examination revealed no new findings other than xiphoid tenderness and an elevated, atonic left diaphragm, with marked lagging of the downward excursion on fluoroscopy. Heart size and electrocardiogram were unchanged. Gastro-intestinal series revealed a large hiatus hernia which reduced promptly in the erect posture. She was given ammonium chloride, 1 gram three times daily and told to elevate the head of her bed with eight inch blocks. Rebreathing in a paper bag on arising was advised if she felt faint. On this simple plan she rapidly improved and is at present asymptomatic, except for an occasional mild nocturnal pain. This patient presented a clear cut example of respiratory alkalosis directly due to a disturbance in diaphragmatic function.

Case 2: A 35 year old female office worker was found to have minimal exudative tuberculosis in the left upper lung field in June of 1940. She spent 11 months in a sanitarium on a modified rest regime. During this time a left phrenic crush was performed resulting in a rise of the left diaphragm to the fourth anterior rib level where it has remained since. She was discharged as arrested and gradually returned during the course of a year to a normal life. Since 1945 chest x-ray films have been consistently unchanged and numerous sputum studies have been reported negative. Her general health has been excellent. However, she has complained of a dull aching
pain over the precordium referred to the left seapular area, a sense of breathlessness and marked exhaustion with ease of fatigue, particularly in the morning hours. Numbness and tingling in the extremities, tachycardia and drawing sensations were almost daily symptoms. All of her complaints were dated from the time of the left phrenic crush. Except for obvious hyperventilation of the rapid shallow type, xiphoid tenderness, and evidence of a huge gastric air bubble, physical examination was negative. On fluoroscopy the left dome of the diaphragm was at the level of the fourth anterior rib and was immobile. A grapefruit sized accumulation of air was present in the gastric fundus. Barium swallow revealed mild pylorospasm with hypomotility of the stomach.

Ammonium chloride and phenobarbital-belladonna mixture gave only slight relief. Intramuscular calcium gluconate was temporarily effective. She was instructed to take a two mile walk on her way to work every morning. After a few days improvement was marked, particularly as to fatigue and dizziness.

This patient was offered a plastic repair of the diaphragm but preferred to remain on a medical regime.

Unquestionably improvement occurred as a result of carbon dioxide repletion following exercise.

Case 2: A 28 year old veteran was seen in February 1947 with a two year history of of frequent attacks consisting of nervousness, fatigue, dizziness, palpitation, polyuria and stabbing over the precordium. The pain radiated to the left elbow and cut off his breath. All symptoms increased while riding to work on the bus. By nightfall he usually felt improved. A peptic ulcer was treated successfully while in the army in 1944. Medical and psychiatric treatment were given in a Veterans Administration Hospital with no improvement.

Examination revealed a tense anxious male with frequent sighing respirations. The xiphoid tip was exquisitely tender and tenderness was present over the precordial interpace. Evidences of a large gastric air bubble were apparent. A gastro-intestinal series showed a duodenal deformity apparently due to an old healed ulcer. Moderate pylorospasm and hypomotility of the stomach were also noted. The left diaphragm was elevated and moved sluggishly.

Phenobarbital-belladonna mixture relieved the bloating somewhat but the other symptoms remained. Coated tablets of ammonium chloride, 3 grams each day were prescribed and long walks were advised. Within a week all symptoms and findings had disappeared and he "felt wonderful." Aside from minor relapses he has had little trouble since and "walks off" his alkalosis without medication.

This patient was discouraged by his failure to improve on previous medical and psychiatric care. Perhaps the strong reassurance given was a factor but improvement was sustained only after regular exercises.

**SUMMARY AND CONCLUSION**

Chronic respiratory alkalosis is a common syndrome with varied and bizarre symptoms.

Physical findings as well as fluoroscopic studies indicate abnormal diaphragmatic function as a cause of pain and hyperventilation.

Reassurance, acidulation, mild sedation and, especially, regular exercises form the main treatment.

**RESUMEN**

La lacalosis respiratoria crónica es un síndrome común con síntomas variados y bizarros.

Los hallazgos físicos así como los estudios fluoroscópicos indican una función diafragmática anormal como causa de dolor y de hiperventilación. Son el trámado principal la recuperación de la confianza, la acidificación, sedación moderada y especialmente los ejercicios con regularidad.

**RESUME**

L'alcalose des affections pulmonaires chroniques est un syndrome banal dont les signes sont variés et curieux.

L'examen physique et l'étude à l'écran radioscopique montrent que la
fonction diaphragmatique est anormale et provoque douleurs et hyperventilation.
Le traitement essentiel consiste en reprise de la confiance, acidification, traitement sédatif doux et particulièrement exercices réguliers.

REFERENCES