# Hyperventilation Syndrome: A Frequent Cause of Chest Pain

*Charles E. Wheatley, M.D., F.C.C.P.**

**Chest pain is frequently a prominent symptom of the hyperventilation syndrome (HVS) and must be distinguished from angina pectoris due to coronary atherosclerotic heart disease (CAHD). The association between hyperventilation and chest pain may be apparent if psychoneurotic traits or anxiety are present. Many patients with HVS are not overtly anxious or neurotic, but in the great majority, a careful history and physical examination will indicate whether chest pain is due to HVS or CAHD. The failure to make this clinical differential diagnosis, which often leads to unnecessary coronary angiography, should not be as frequent as generally experienced. Fifteen of 95 consecutive patients had chest pain and additional typical HVS symptoms. Reassurance and detailed explanation about the cause of the chest pain gave significant relief, so that all patients were less symptomatic 24 to 44 months later, and none had developed new signs or symptoms to suggest that symptomatic CAHD had been overlooked. The risk and expense of coronary angiography was avoided.**

The clinical diagnosis of HVS is suspected after a careful, subtle and penetrating history in which all symptoms are weighed separately and together. During history-taking, constant consideration that HVS may be the cause of chest pain is necessary. Meager physical and laboratory abnormalities confirm historic impressions. To contrast chest pain due to HVS vs CAHD, three patients are summarized as to history, physical abnormalities, laboratory and x-ray film abnormalities, ECG changes at rest and after exercise, treatment instructions, and results.

## Materials and Methods

The 95 patients in this study were evaluated over a one-year period, 1969-70. All patients received a complete history and physical examination with particular attention to emotional status. Screening laboratory values were obtained for pulmonary, liver and kidney function, serum lipids and carbohydrate tolerance. A chest x-ray film and electrocardiogram were obtained at the initial visit. Each patient with chest pain which required the differential diagnosis of HVS versus CAHD underwent a two-step exercise test with a double work load.[1]

Fifty-one (54 percent) of the entire group of patients had organic heart disease of congenital, rheumatic, atherosclerotic, hypertensive or myopathic etiology. Twelve patients were catheterized and nine underwent surgical correction in this community hospital.

Forty-two (44 percent) of the entire group did not have organic heart disease. Two patients (2 percent) had possible organic heart disease—one with a possible atrial septal defect could not be catheterized due to pregnancy, and the second may have had CAHD, but each patient's symptoms were most suggestive of HVS. Both of these patients are included among 27 patients with HVS, and the second is included among 15 with pain suggestive of CAHD (patient 3). Patients with chest pain not typical of CAHD or HVS and without additional symptoms suggestive of HVS had scalenus anticus syndrome and various musculoskeletal diseases including myositis, bursitis and cervical degenerative disc disease.

HVS was the dominant cause of symptoms in 27 patients of the entire group (28 percent), (Table 1). Fifteen (56 percent) of these 27 patients with HVS (16 percent of the total group) had chest pain suggestive of CAHD, but careful evaluation indicated their pain to be more likely due to HVS. These patients were assured that they did not have CAHD and were given a detailed explanation about the cause of HVS chest pain and how to prevent its occurrence. They were invited to return for further help if necessary. Their subsequent improved clinical course validated this judgment

**Table 1—*Characteristics of 27 Patients with Hyperventilation Syndrome***

| | | |
|---|---|---|
| Age | Mean 36.5 yrs. | Range 16 to 58 yrs. |
| Sex | Men 6 | Women 21 |
| Associated heart disease (5) | | |
| | Rheumatic | 4 |
| | Congenital (possible) | 1 |
| Associated pulmonary disease (3) | | |
| | Emphysema | 3 |
| No demonstrable heart or pulmonary disease (19) | | |
| | Functional murmurs | 4 |
| | Pain suggestive of coronary heart disease | 15 |
| | Age Mean 42 yrs | Range 21 to 59 yrs. |
| | Sex Men 6 | Women 9 |

*Clinical Instructor in Internal Medicine, University of Michigan Medical Center, Ann Arbor.
Manuscript received March 6, 1974; revision accepted January 24
*Reprint requests: Dr. Wheatley, 44875 Thornapple Lane, Northville, Michigan 48167*

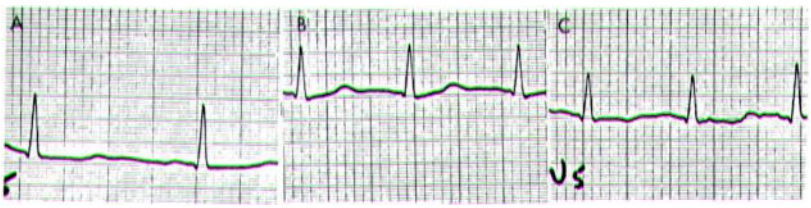


FIGURE 1. Patient 2. Lead $V_5$ recorded at rest (A), immediately after exercise (B), and 5 minutes after exercise (C).

and treatment. Three of these patients are presented in detail.

In our study, women had HVS more frequently (Table 1). However, when men have HVS, it is more likely that their symptoms will include chest pain which must be distinguished from angina pectoris due to CAHD.

## CASE REPORTS

### CASE 1

A 46-year-old white man first had aching substernal chest pain which radiated into the right axilla as he lifted 60 lb oil cases. Perioral and extremity paresthesia, deep and rapid respiration, and wet palms disappeared spontaneously with the chest pain. Similar episodes since 1967 most often began as he sold retail auto parts. Dissatisfied customers made him feel "like he was going to hell," but he continued to work breathing rapidly and experiencing chest pain. A lunch hour rest often prevented afternoon chest pain. A good night's rest prevented some morning attacks, but pain appeared later in the afternoon when he was more fatigued. He shoveled sand and snow for several hours without chest pain, but became short of breath after a few minutes. Symptoms disappeared during vacation in Florida, but returned at work. Emphysema and tracheobronchitis were diagnosed after he coughed and wheezed while cutting grass. Chest pain and fatigue appeared during normal pulmonary function tests.

Abnormalities included rough inspiratory breath sounds and serum triglyceride level of 462 mg percent. There were no ECG changes at rest or after exercise. Treatment consisted of reassurance and instruction on how to prevent hyperventilation, the institution of a regular exercise program, stopping smoking and changing occupations, as well as a 1500 calorie, low-fat diet. Chest pain disappeared after he changed employment and he was asymptomatic 36 months later.

### CASE 2

A 36-year-old white woman first had chest pain, "like an elephant sitting on my chest," that started beneath the left breast and radiated to the left shoulder, as she pushed cars through the snow. The pain inconsistently began as she sat in the bathtub or swam in the lake. When she climbed 88 steps from the beach to their cottage, chest pain forced her to stop a few seconds. Dizziness began without chest pain or breathlessness. Since her husband had lost both hands, she often assumed the more strenuous role in prolonged sexual intercourse which frequently caused chest pain that never stopped the act. Nitroglycerine did not prevent or relieve the pain, but increased it and added a sensation of suffocation. Pressure on the left chest often caused acute pain and residual tenderness.

Her home life was happier after a hysterectomy. She "coped" with her hypercritical mother by "keeping my mouth shut." Work in an emergency room exposed her to HVS patients. Physical findings noted were twitching eyelids and tenderness of the chest wall inferior to the left breast. Search for a trigger point that referred pain to this area was negative. ECG at rest showed nonspecific repolarization changes (Fig 1-A). Immediately after exercise, T-waves became more upright and junctional RST-segment depression appeared. Similar earlier tracings were interpreted to show CAHD. Breathlessness during and after exercise was followed by "typical" chest pain eight minutes after exercise. Nitroglycerine increased the chest pain, but did not change the ECG. Perioral and extremity paresthesia and a "passing-out" sensation subsided with the chest pain. Assurance that HVS caused the chest pain prompted tearful hostility toward a previous cardiologist. Instruction was given on how to prevent hyperventilation. She was much improved after 24 months. No additional treatment necessary.

### CASE 3

A 42-year-old white man had sudden, sharp and severe anterior chest pain as he walked downhill to work in an auto factory. The pain disappeared after two minutes of rest. Thereafter, substernal aching, "like an orange sitting in my chest," began with emotion and exertion. When the pain became severe and radiated into his hands, he became weak and nauseated, but never dyspneic or diaphoretic. Two weeks later he walked rapidly up the same hill without chest pain. Chest pain began if he walked rapidly during his daily 5 miles at work. He secretly left the factory before shift change at 4:30 AM to steal sleep before starting his second garbage-collection job; chest pain appeared most often at these times. At the check-out clock he felt "weird and my arms felt funny." Severe chest pain began, but examination and ECG were normal. He did 18 consecutive chin-ups without pain. Chest pain began after he lifted two heavy garbage barrels, but if he persisted without rest, it disappeared after eight barrels. The only physical abnormality found was arcus lipidus. An exercise ECG in another hospital in January, 1970, was interpreted as abnormal due to left posterior hemiblock immediately after exercise (Fig 2). Normal conduction re-

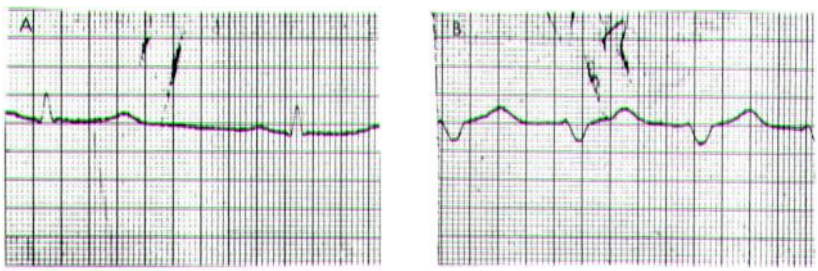


FIGURE 2. Patient 3. Lead 1 recorded at rest (A) and after exercise (B), January 1970 (25 mm paper, 50 mm paper speed).

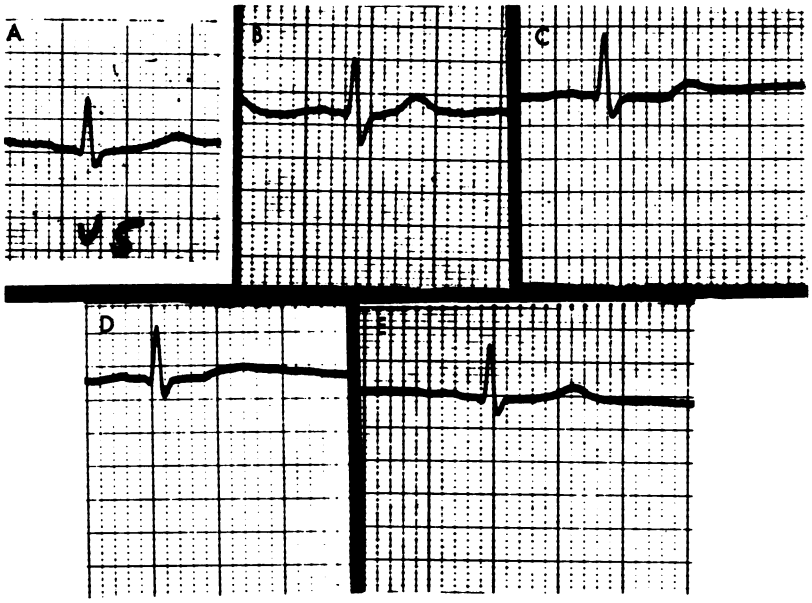


FIGURE 3. Patient 3. $V_5$ at rest (A), immediate (B), 2 min (C), 10 min (D), and 15 min (E) after exercise, May 12, 1970.

turned after three minutes of rest. An ECG recorded May 12, 1970, (Fig 3), showed upright T-waves in the chest leads in the resting and immediate post-exercise tracings. T-waves were terminally inverted in the 2, 4, and 6 minute tracings and upright in the 10 and 15 minute tracings. After exercise he became hyperpneic and pale. A "mild ache" at the xiphoid process disappeared spontaneously in two minutes. The test was interpreted as borderline. Reassurance was given that we had not found absolute evidence of CAHD along with instruction as to how to prevent hyperventilation. Forty-four months later, chest pain was greatly reduced in frequency and severity without medication. He avoided chest pain by "relaxing" and impressed younger workers with 30 chin-ups without chest pain.

## DISCUSSION

Three distinct types of HVS chest pain can be identified. The differences in location, radiation, quality, intensity, fluctuation and periodicity, and duration allow their identification. The first type is sharp, fleeting and periodic and most often originates in the hypochondrium or anterior left chest and radiates into the neck, left scapula and along the inferior rib margins (patient 2). Its intensity is increased by deep breathing, twisting and bending. Pressure on the diaphragm by the stomach distended by swallowed air[2] and transient arrhythmias[3] are postulated mechanisms. Spasm of the homolateral diaphragm leaf is another acceptable etiology.

A persistent, localized, aching discomfort, usually under the left breast, may last for hours or days and not vary in intensity with activity or motion of the chest wall. Typically the chest wall is tender at the site of pain (patient 2). Wood[4] proved that this inframammary pain is local when he achieved relief from pain and tenderness by injecting novocain into the intercostal muscles at the point of tenderness. Referred muscle pain and deep tenderness from a remote trigger area can cause similar chest pain, but infiltration of the trigger area with local anesthetic gives relief from pain and tenderness in the referred site.[5] The presence of additional HVS symptoms helps differentiate the two causes. A precipitating event can often be elicited for referred pain, *eg* trauma, strain, exposure to wind. Careful search for trigger points in our HVS patients was negative. This second type of HVS chest pain is probably caused by localized spasm of the intercostal muscles induced by hypocapnia. Intercostal muscle spasm, more widely distributed, spreads an aching, constrictive pain over the entire hemithorax. Viar and Grote[6] reported a HVS patient with diffuse, unilateral, intercostal muscle spasm which was demonstrated by retraction of unilateral intercostal spaces on x-ray examination. Relief was obtained with intravenous calcium gluconate.

A diffuse, dull-aching, heavy-pressure sensation over the entire precordium or substernum which does not vary with respiration or activity may last for minutes or days (patients 1, 3). This third type of HVS chest pain is probably caused by tonic contraction, strain and fatigue of respiratory muscles,[4,7] and is most frequently confused with angina pectoris caused by CAHD. Friedman[7] correlated this pressure sensation with a peculiar breathing pattern in which the upper chest musculature maintains respiration while the lower chest muscles and diaphragm remain relatively immobile.

Angina pectoris due to CAHD appears most often during exercise and disappears promptly with rest. In contrast, HVS chest pain usually begins after exercise and increases in intensity as unnecessary overbreathing is prolonged (patient 2). HVS chest pain rarely causes cessation of activity, particularly if the activity gives pleasure and it is important to

question the patient about what activities he must stop when pain begins (patients 2, 3). HVS chest pain often begins at bedtime, or pain and breathlessness may awaken him after several hours of sleep. Months of intermittent pain and unproductive examinations by numerous indecisive physicians increase anxiety and pain (patients 1, 3).

Characteristic secondary symptoms are clues which support the diagnosis of HVS chest pain. Frequently these secondary symptoms are elicited after a history and physical examination directed toward cardiovascular disease has failed to suggest that the chest pain is due to CAHD. A history of breathlessness without exertion, inability to obtain a satisfying breath, and frequent yawning or sighing is obtained easily. Obvious deep breathing may not be present at the initial examination, but many patients will take frequent deep, sighing breaths. The patient may be unaware that he is breathing more rapidly and deeply than his level of activity should demand, or awareness of deep breathing and unusual fatigue may appear after exercise. A chronic state of hyperventilation may be induced which will require only a few additional deep breaths to reach the level of pain production.[6,8,9] During hyperventilation crises, rapid, shallow or deep breathing is prominent. Exercise-induced bronchospasm and wheezing due to hypocapnia are features of HVS (patient 1).[10]

Giddiness or feeling off-balance, postural faintness due to momentary hypotension, headache, pallor, diaphoresis, blurred vision, and palpitation with bradycardia or tachycardia may be present. "Pins and needles" perioral and extremity paresthesia is characteristic of HVS. Perioral distribution distinguishes HVS paresthesia from that caused by intrathoracic disease and neuropathy at lower cervical and upper dorsal levels. Dysphagia due to a "lump in the throat," and extremity heaviness, weakness and cramps are additional muscle spasm symptoms. If the left arm is the only symptomatic extremity, confusion with CAHD is more likely.[9]

Sympathetic probing of patient reaction to marital, home, social and work situations will usually yield an emotional background for hyperventilation (patients 1, 2, 3). HVS patients frequently work secretly at second jobs to support real or imagined family needs with little time for sleep or relaxation (patient 3). Review of his life at the onset of chest pain may yield emotion-laden material and allow him to understand the relationship between emotion and chest pain (patient 1).

ECG abnormalities caused by HVS may be incorrectly attributed to CAHD. Hyperventilation and changes in posture at rest and during exercise cause arrhythmias, RST-segment depression and T-wave inversion.[11-16] Biberman[17] found that hyperventilation caused T-wave inversion in 73 percent of randomly selected healthy subjects. Solitary T-wave changes must not be considered definite ECG evidence of heart disease.[18] Pre-exercise tracings in supine, sitting and upright positions with and without rapid and slow hyperventilation must be recorded before one may conclude that ECG changes which appear with exercise are possibly due to CAHD (patients 2, 3).[16]

RST-segment and T-wave changes induced by hyperventilation have been attributed to various causes, but the exact cause is still uncertain.[17] Yu et al[13] showed that hyperventilation T-wave changes, which are similar to those caused by intravenous epinephrine, can be reversed by propantheline and phentolamine. Since propranolol prevents these changes,[19] beta-adrenergic stimulation may be the cause. Biberman's group[17] found that beta-adrenergic stimulation with isoproterenol caused transient T-wave inversion similar to that caused by hyperventilation in 10 of 11 subjects without heart disease. They postulate that the common mechanism underlying these changes is an asynchronous shortening of ventricular repolarization during sympathetic stimulation caused by hyperventilation. Anxiety followed by hyperventilation and sympathetic catecholamine discharge with resultant shortening of ventricular repolarization becomes an acceptable hypothesis for the etiology of hyperventilation T-wave changes.

Patients with ECG changes before and after exercise may not have similar changes in subsequent tracings (patient 3). A 20-year follow-up study of 173 neurasthenia patients clearly showed that reversible ECG changes are not associated with decreased longevity.[20] A patient with chest pain must receive a careful history, emotional status evaluation and physical examination before resting and exercise ECG changes can be considered suggestive of CAHD.

## References


1 Master AM: The two-step exercise electrocardiogram; A test for coronary insufficiency. Ann Intern Med 32:842-863, 1950

2 Rice RL: Symptom patterns of the hyperventilation syndrome. Am J Med 8:691-700, 1950

3 Friedman M: Functional Cardiovascular Disease. Baltimore, Williams and Wilkins, 1947, p 25

4 Wood P: DaCosta's syndrome (or effort syndrome). Br Med J 1:767-772, 1941

5 Travell J: Referred pain from skeletal muscle. The pectoralis major syndrome of breast pain and soreness and the sternomastoid syndrome of headache and dizziness. New York State J Med 55:331-340, 1955

6 Viar WN, Grote CA: Chest pain produced by hyperven-

tilation with spasm of intercostal muscles. Relief by calcium. J Med Assoc Ala 21:149-153, 1951
7 Friedman M: Studies concerning the etiology and pathogenesis of neurocirculatory asthenia. Am Heart J 30:557-566, 1945
8 Saltzman HA, Heyman A, Sieker HO: Correlation of clinical and physiological manifestations of sustained hyperventilation. N Engl J Med 268:1431-1436, 1963
9 Tavel ME: Hyperventilation syndrome with unilateral somatic symptoms. JAMA 187:301-303, 1964
10 Ferguson A, Addington W, Gaensler EA: Dyspnea and bronchospasm from inappropriate post-exercise hyperventilation. Ann Intern Med 71:1063-1072, 1969
11 Kemp GL, Ellstead MH: The significance of hyperventilative and orthostatic T-wave changes on the electrocardiogram. Arch Intern Med 121:518-523, 1968
12 Kannel WB, Dawber TR, Cohen ME: The electrocardiogram in neurocirculatory asthenia (anxiety neurosis, or neuroasthenia). A study of 203 neurocirculatory asthenia patients and 757 healthy controls in the Framingham study. Ann Intern Med 49:1351-1360, 1958
13 Yu PN, Bernard JB, Stanfield CA, et al: Hyperventilation syndrome. Arch Int Med 103:902-913, 1959
14 Semler HJ: Cardiac disorders during acute emotional stress. Circ Supp 32:II-192, 1965
15 Wildenthal K, Fuller DS, Shapiro W: Paroxysmal atrial arrhythmias induced by hyperventilation. Am J Cardiol 21:436-441, 1968
16 McHenry P, Cogan O: False positive ECG response to exercise secondary to hyperventilation. Cineangiographic correlation. Am Heart J 79:683-687, 1970
17 Biberman L, Sarma RN, Surawicz B: T-wave abnormalities during hyperventilation and isoproterenol infusion. Am Heart J 81, No. 2:166-174, 1971
18 Prinzmetal M, Goldman A, Massumi RA, et al: Clinical implications of errors in electrocardiographic interpretation. Heart disease of electrocardiographic origin. JAMA 161:138-143, 1956
19 Furberg C, Tengblad CF: Adrenergic beta-receptor blockade and the effect of hyperventilation on the electrocardiogram. Scand J Clin Lab Invest 18:467-472, 1966
20 Wheeler EO, White PD, Reed EW, et al: Neurocirculatory asthenia (anxiety neurosis, effort syndrome, neuroasthenia): A twenty year follow-up study of one hundred and seventy-three patients. JAMA 142:878-888, 1950

# ANNOUNCEMENTS

## 50th Anniversary Congress, Pan American Medical Association

The 50th and Golden Anniversary Congress of the Pan American Medical Association will be held in Hollywood, Florida, October 24-29. Dr. Seymour M. Farber, San Francisco, is President of the Section on Pulmonary Diseases and Dr. Miguel Jimenez, Mexico City, is Latin American Chairman. For information, contact Dr. Joseph J. Eller, Director General, Pan American Medical Association, 745 Fifth Avenue, New York, New York 10022.

## Seventh International Conference on Sarcoidosis and Other Granulomatous Disorders

The International Committee on Sarcoidosis will present the Seventh International Conference on Sarcoidosis and Other Granulomatous Disorders October 6-10 at the Delmonico Hotel, New York City. For information, contact the conference chairman, Dr. Louis E. Siltzbach, Mount Sinai School of Medicine, 100th Street at Fifth Avenue, New York, New York 10029.