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Treatment of the Hyperventilation Syndrome

Hans von Brauchitsch, MD*

A review of the literature on the hyperventilation syndrome reveals a variety of treatment approaches and a virtual absence of controlled studies of this common problem. Attempts to treat the condition by changing the respiratory pattern through mechanical means can be traced back to the first century A.D. The "paper bag method" of treatment has many psychological drawbacks and is often unsuccessful. Several avenues of drug treatment have been explored: acidifiers, minor tranquilizers, antidepressants, and stimulants. Reports of success with insight-oriented intensive psychotherapy are scarce. Some forms of group psychotherapy have been tried with success. The model of the "medical friendship" may be most appropriate, and abreaction as well as verbal ventilation may prevent respiratory hyperventilation at least temporarily. Research is badly needed, utilizing the physiological changes occurring in the hyperventilation syndrome.

The American Handbook of Psychiatry discusses the hyperventilation syndrome under the heading of "Commonly Neglected Psychosomatic Syndromes". Considering the fact that this disorder may be more prevalent than schizophrenia, that subjectively it is profoundly unpleasant, and that millions of dollars are spent yearly for emergency care and treatment, one wonders why most textbooks lend it a few fleeting sentences, at best.

It has been established that approximately 5% of all patients seen in gastroenterology, over 10% of those seen in internal medicine and up to one third of all patients in general practice suffer from hyperventilation. The syndrome is frequently encountered by the pediatrician and is of great concern to the obstetrician. Of all medical specialities, the psychiatrist's office appears to be the only one in which hyperventilators are seen rarely, if at all.

Since patients with hyperventilation syndromes are almost always first seen by non-psychiatric physicians, one questions why are they so rarely referred to psychiatrists? It may well be that the seemingly gross organic pathology of the syndrome leads the primary physician to rule out all possible concomitant organic factors. Or the opposite may be true: the sharp discrepancy between the alleged harmlessness of the condition and its dramatic symptomatology may

*Formerly, Department of Psychiatry, Henry Ford Hospital

Address requests for reprints to Dr. von Brauchitsch at the University of Oklahoma, Health Sciences Center, P.O. Box 26901, Oklahoma City, OK 73190.
produce a shrug-of-the-shoulder, “justanother-hyperventilator” attitude from the emergency room physician. The attitude of belittling the seriousness of the syndrome is the least justified. Hyperventilation attacks may not threaten physical survival, but the hyperventilation syndrome in its chronic form can become an exceedingly disabling and intractable condition. In my experience, very few hyperventilators are ever cured, and a large percentage become literally crippled to the point of social uselessness. For this reason, a review of the unfortunately meager therapeutic armamentarium available to the physician may help to stimulate efforts to find more reliable remedies.

**Nosology and Classification**

It is reasonable to question whether the hyperventilation syndrome is a disease entity or one of the ubiquitous physiological concomitants of anxiety. I have chosen to treat the condition for practical reasons as if it were a nosological entity. But I am willing to concede that, even in the context of this paper, the term could delineate a target symptom rather than a disease. The fact that the hyperventilation syndrome produces measurable organic changes of blood gases, pH of blood and urine, electrocardiogram, electroencephalogram, etc would place it theoretically into the category of psychosomatic diseases. One source of confusion regarding definition of the syndrome, however, is that it tends to be grouped with purely psychological disturbances, especially with the so-called “actual neuroses”. The list of conditions identical with or closely related to the hyperventilation syndrome appears endless.

It is possible to distinguish between acute and chronic hyperventilation syndrome. Transitions occur and the presence of one form does not preclude the emergence of the other. The acute hyperventilation syndrome is frequently associated with acute, overwhelming anxiety and therefore also called anxiety state, anxiety neurosis, hysterical attack, or hyperventilation tetany. The anxiety label is popular among psychiatrists, but not entirely correct, since not all hyperventilation attacks are accompanied by overt anxiety, and because not all anxiety states produce noticeable hyperventilation. It is certainly not related to hysteria in the form of neurosis, although it may occur more frequently in patients diagnosed as hysterical characters. The chronic hyperventilation syndrome tends to produce a multitude of physical symptoms often without pronounced emotional concomitants. Many physical complaints refer to the heart rather than to respiration and account for hyperventilation syndrome being labelled as Da Costa’s syndrome, neurocirculatory asthenia, effort syndrome, soldier’s heart, irritable heart, vasomotor instability, vasomotor neurosis, cardiac neurosis, etc.

In many cases, the chronic fatigue (chronic hyperventilation from vigorous physical exercise) forms a picture identical with that of neurasthenia, effort syndrome, or chronic exhaustion state. The condition may simulate the symptoms of functional hypoglycemia; the typical dry tongue caused by mouth-breathing has been described under the name of xerostomia. Finally, it may be associated with chronic aerophagia, producing a multitude of “functional” gastrointestinal symptoms. Common to all forms is the pathognomonic “sighing”, open-mouthed respiratory pattern which uses intercostal respiratory muscles.

**Treatment**

**A. Increase of Blood Dioxide Content**

Since the hyperventilation syndrome is characterized by carbon dioxide depletion and paradoxical stimulation of
Treatment of the Hyperventilation Syndrome

the medullary respiratory centers, it appears logical to treat the condition by manipulating the inspired gases. This approach has almost two thousand years of medical endorsement. Aretaeus of Capadocia recommended as early as 100 A.D. blowing "evil-smelling fumes" into the faces of those afflicted. A 17th century formulary gives the prescription for a "parfum" consisting of castorea, partridge feathers, paper and asa foetida to be smelled by patients suffering from "les suffocation de la matrice". Castorea is a substance extracted from the perineal glands of beavers and its effect may have been comparable to the sensation of being sprayed by a skunk — a powerful deterrent to hyperventilation, if not respiration of any kind. A textbook of medicine published in 1766 suggests "stinking and volatile spirits" or just singed feathers, burning leather or burnt horncombs be held under the nose of those suffering from "suffocatio hysterica". With the advent of the age of reason, the method was merely altered, but not abolished. That the ammonia in the smelling-salt bottles of the Victorian lady actually stimulated the medullary centers other than momentarily is hard to believe, but it is a permissible assumption that the act of smelling ammonia would slow down an accelerated respiratory rate. One British physician invented a spring belt to be worn around the chest in order to reduce the patient's breathing rate. Since the inventor of this gadget applied it indiscriminately to hyperventilators as well as to patients suffering from heart disease, it fell understandably soon into oblivion. One may speculate that the enormous popularity of the cigarette, especially in times riddled by the anxiety of strife and war, may not so much be due to "oral fixation" as to the fact that one cannot smoke and hyperventilate easily at the same time. (It is proverbial to "light up" after a bad scare). Since time immemorial, people have used singing and whistling ("whistling in the dark") to regulate their respiratory rates during times of anxiety.

Modern medicine has continued the tradition by recommending the use of the "paper bag method" (increasing arterial carbon dioxide tension by breathing from a bag and thus helping to correct the respiratory alkalosis which explains some of the acute symptoms). The method can be dramatically effective at times. In my own experience, however, the paper bag method is more a liability than an asset. It may work once or even twice; but since it does nothing to attenuate the anxiety underlying the hyperventilation attack, the patient soon loses faith or feels that he is being ridiculed. Its use may drive the patient to seek help in the emergency room of another hospital at the time of his next attack, leaving the physician with the erroneous belief that he had "cured" his patient.

One simple method to regulate the patient's breathing rate is by encouraging him or her to talk. Provided that the acute hyperventilation attack has not progressed too far (in which case the patient's sensorium may be clouded to the point where rational conversation is no longer possible), it is usually helpful to ask the patient to give a thorough and clear description of symptoms. Relatives ought to be excluded because they tend to insist on telling their side of the story. The patient is encouraged to talk loudly and clearly (it may be permissible to develop a transient partial deafness during the interview). The combination of reassurance and enforced respiratory slowdown is often sufficient to bring relief within five to ten minutes.

B. Drugs

Evaluation of the efficacy of drugs in the hyperventilation syndrome is difficult. First, hyperventilation attacks are
too unpredictable and the reassuring effect of medical attention is too pronounced to distinguish between a genuine drug and a placebo-effect. Second, since many hyperventilators have been exposed to a multitude of drugs, prescribed unsuccessfully by their physicians, they tend to be skeptical about any kind of drug. Subsequently, their drug-taking habits are unpredictable and there is a pronounced tendency to modify or discontinue prescriptions at will. Finally, because of the marked anxiety pervading the condition, and the need for medical support, the patient may reject the very medication which would ultimately be effective, because he subconsciously fears that improvement will cut him off from his justification to contact his doctor. These are the patients who develop the strangest and most outlandish "side effects" to drugs which are known to be totally harmless.

Prior to the advent of effective and safe tranquilizers, the medical literature recommended the use of drugs which would affect the blood pH directly. Ammonium chloride, 1 gm three to four times daily, was said to be the medication of choice, although to my knowledge there are no controlled or follow-up studies reported in the literature. Equally unsupported appears the claim that acid-ash diets, carbonated water, phenoxyethyamine, ergotamine, yohimbine, hydrastine, quinine, pilocarpine, histamine or acetylcholine will bring relief.

With the development of modern tranquilizers, it is possible to affect the anxiety component of the hyperventilation syndrome. Surprisingly enough, although much effort has been dedicated to the evaluation of the various drugs' usefulness in anxiety states, very little has been said about their use in the hyperventilation syndrome. Many hyperventilators have been exposed over the years to the entire pharmacological armamentarium of office practice. In my practice, I cannot remember a single patient who had not had previous drug treatment. This drug exposure implies limited usefulness but ineffectiveness may not be so much a function of their chemistry, as of the prescribed dosage. Many physicians tend to use the minimum recommended dosage of any psychotropic drug only, notwithstanding the fact that the massive anxiety, encountered especially in the acute hyperventilation syndrome, cannot be expected to yield to such homeopathic administrations as five or ten milligrams of chlordiazepoxide. In view of the paroxysmal nature of the acute hyperventilation syndrome, I have found tranquilizers useful on a demand schedule in moderate to high dosage. If the fullblown syndrome is already present (for instance, at the time of admission to the emergency room), 100 mgm of chlordiazepoxide may be administered intramuscularly. Patient and family should be told that drowsiness or sleep will ensue. After establishing a good working relationship with a patient, I prescribe 25 mgm chlordiazepoxide capsules (or an equivalent dosage of any minor tranquilizer), two capsules to be taken as soon as an impending attack is felt. Again, the patient is instructed to rest and to abstain temporarily from driving a car or any potentially hazardous occupation. Sometimes the dosage has to be repeated until the attack is under full control. Admittedly this regimen is less effective once the disease has entered a chronic stage.

Controlled clinical trials have been conducted of antidepressant drugs with favorable results, although clinically manifest depression is quite rare in hyperventilation syndrome. No pharmacological rationale has been offered, nor are there long-term or follow-up
Treatment of the Hyperventilation Syndrome

studies in the literature. I tend to agree cautiously that, in chronic hyperventilation syndrome, antidepressant drugs appear more beneficial than tranquilizers. A trial with antidepressants or antidepressant/tranquilizer combinations appears justified especially if a strong pain component (chest wall pain) is present. In patients with the chief complaint of lightheadedness, depersonalization sensations, xerostomia, and excessive thirst, such drugs may produce adverse rather than beneficial results. From the antidepressants it is a short step to the use of stimulants. Again, my own experience is not unfavorable, especially if the patient complains about chronic fatigue and exhaustion. Due precautions against the abuse of these drugs have to be taken.

Relevant in this context is the question which drugs not to use in the hyperventilation syndrome. The chronic hyperventilation syndrome, especially if chest wall pains are present, may be mistaken for angina pectoris and treated with nitroglycerine or amyl nitrite. Both drugs have been proven to aggravate abnormal EEG findings related to hyperventilation. If the psychological roots of the disease are recognized, some physician may erroneously conclude that the patient is “hysterical” and revert to the use of placebos. This does the patient injustice because it is recognized by many psychiatrists that placebos should be used under no circumstances.

Finally, since chronic hyperventilators suffer from such a large number of ills and ailments, the role of over-the-counter drugs should also be assessed. I have seen hyperventilating patients who consumed staggering doses of acetylsalicylic acid day in and day out. Salicylates, if used in sufficiently high dosages, can produce hyperventilation alkalosis by themselves, thereby predisposing the hyperventilator to some of the most troublesome symptoms of the disease.

C. Psychological approaches

Some authors recommend a very simple procedure to alleviate the patient’s apprehension. They instruct the patient to forcibly hyperventilate in the presence of the physician. The re-emergence of the symptoms of the hyperventilation syndrome then not only secures the correct diagnosis, but serves as a major therapeutic tool. It clarifies the underlying mechanism of the condition for the patient, thus alleviating his anticipatory anxiety and strengthening his confidence in the physician. This technique can be highly effective but should be used cautiously. First, not all hyperventilators are able to reproduce an attack in the physician’s office. There is no real motivation for prolonged hyperventilation because the very presence of the physician is reassuring to the point where no true anxiety build-up can occur. The more chronic the condition, the less likely is the test to succeed. The net result may be a mere embarrassment to the patient and the physician alike, or an angry reaction on the side of the latter because the patient “did not cooperate”.

Even if some or all of the acute symptoms can be reproduced, the relief felt by the patient may turn out to be short lived. The influence of intellectual understanding on the underlying emotions is minute, and the first recurrence of an uncontrollable hyperventilation attack outside of the doctor’s office may shatter all hopes, as well as the faith in the physician’s skills. Hyperventilation is a chronic habit disturbance, and bad habits cannot be overcome by simply pointing out to the patient that they exist.

Psychotherapy is almost universally recommended, but since not all investigators were psychiatrists, the meaning of the word is frequently vague. It ranges
from simple reassurance, preferably by a non-psychiatric physician\textsuperscript{5,17} over "psychocatharsis and re-education",\textsuperscript{4} "attitude therapy"\textsuperscript{19} to intensive, uncovering, psychoanalytically oriented psychotherapy.\textsuperscript{1,18} The outcome is usually considered favorable, an optimism which is as surprising as it is unsupported by clinical data.

To clarify the issue, it will be necessary to digress briefly into the history of the development of psychotherapeutic concepts. In classic psychoanalytic nosology, the hyperventilation syndrome is viewed as an organ neurosis.\textsuperscript{19} The contemporary psychiatric term is psychosomatic disease. Even if viewed as a modification of an anxiety state (or anxiety neurosis), in the older nomenclature the condition would be categorized as an actual neurosis. (The "actual neuroses" are anxiety states, neurasthenia, and hypochondriasis). Unfortunately, and obviously caused by a historical misunderstanding of the Freudian use of the word neurosis, contemporary nomenclature lists the actual neurosis together with the transference neuroses, (hysteria, phobia, obsessive-compulsive and depressive neurosis) with which they have little in common. Psychoanalytic psychotherapy is the treatment of choice in the transference neurosis, but almost all psychoanalysts, including Freud and Fenichel,\textsuperscript{19} state that it is ineffectual in the actual neuroses and the organ neuroses.

The explanation for the lack of success of insight (interpretative) psychotherapy in the hyperventilation syndrome is obvious. As in many psychosomatic diseases, the ultimate root of the condition is a physiological emergency reaction. In the face of danger the organism is biologically prepared for fight or flight.\textsuperscript{7} This is an entirely different mechanism than the one seen in the transference neuroses, which are symbolic expressions of repressed instinctual conflicts. In the majority of cases, hyperventilation is a physiological reflex and therefore not accessible to psychoanalytic interpretation or insight. The actual cause of the anxiety is, according to my observations, not so much repressed by the hyperventilator, but rather suppressed or plain ignored. A great many of these patients are either too isolated to be able to relate their fears to a sympathetic listener, or have a peculiar sense of shame preventing them from "complaining", which they regard as a sign of weakness. If encouraged, they are able to recall traumatic experiences leading directly to their acknowledged fear of sudden death. In a large number of cases this event was either the sudden death of a member of the family or the patient's own involvement in an accident. It is quite surprising to observe the rapid decrease of hyperventilation attacks once the patient is given the opportunity to talk about his worries. The drawback of this therapeutic approach is the fact that it is interminable: As soon as the patient ceases to ventilate his fears, he is bound to hyperventilate again.

Theoretically, the need for ventilation and catharsis and the disturbances of interpersonal communication skills could make the hyperventilator an excellent candidate for group psychotherapy. In my experience, that approach works only in a few selected cases. Unless very well prepared, the patient will quickly revert into customary silence, feeling that he cannot afford to "bother" other group members with his apprehensions. One important exception to this rule are inspirational groups like "Recovery Inc." who are often highly effective in keeping the patient functioning.

Thus, the psychotherapeutic approach to the hyperventilation syndrome resembles closely the psychotherapy of most psychosomatic diseases. The main accent is on the establishment of a
Treatment of the Hyperventilation Syndrome

“medical friendship”, involving the therapist as a person who is capable of showing sympathy and understanding, who will not hesitate to help the patient with everyday decisions and the resolution of environmental problems, if warranted. Since fear of death is one of the mainsprings of the condition, the physician will do well to emphasize the medical role. Dependency on the physician is bound to develop but must be regarded as a reasonable price to prevent invalidity. As in many other psychosomatic conditions, therapy can offer cure only in very few cases, some degrees of improvement in many, and prevention of further deterioration in patients who already have entered into the chronic stage of the disease.

Discussion

If the approach used in this paper is impressionistic and possibly even polypragmatic in an era of strict scientific demands, one has to realize that this less-than-scientific treatment of the issue was not by choice, but by necessity.

Dealing with a problem with scientific objectivity requires operating in a defined conceptual framework. Criteria for diagnosis must be standardized and the natural history of the disease — its course without treatment — must have been established. To assess the effectiveness of therapy there must be some yardstick to measure the degree and severity of the illness. None of these are possible in the hyperventilation syndrome. Here, conditions for research are still pre-scientific. Any attempt at investigating the efficacy of treatment is at such an early and unsophisticated stage that results may sound like a treatise on the remedy of swamp fever written in the early nineteenth century.

This is a surprising state of affairs. The physiological findings in the hyperventilation syndrome have been well explored. At least the physical aspects of the condition should be easily accessible to exact, scientific measurement. There seems to be no valid excuse for diagnosing a condition characterized by simple and clear metabolic and physiological abnormalities by the same intuitive and impressionistic criteria as “neurasthenia” and “hypochondriasis.”

If this observation is true for diagnostic methods, it should be equally valid for therapy. If the metabolic aberrations responsible for the condition can be measured, then, one may assume, they can also be reversed. A medical science that feels competent to modify the genetic code ought to be capable of dealing with the biochemical sequelae of transient hypocapnia. One may anticipate that correction of the underlying somatic factors may do very little to influence the emotional maladjustment reaction. But, that is an indispensable first step to rendering the patient accessible to other means of treatment.

References


