CHAPTER CONTENTS

2

Patterns of breathing dysfunction in hyperventilation syndrome and breathing pattern disorders

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INTRODUCTION

This chapter covers both normal breathing patterns and what happens to individuals when breathing patterns become dysfunctional. The consequences of disordered breathing patterns are not only distressing to the patient but also expensive to our health care systems if they are not diagnosed and treated (once more serious pathologies have been ruled out).

Too often, patients present to emergency rooms or to general practitioners with frightening symptoms which mimic serious disease (Lum 1987), though blood tests, heart checks such as electrocardiographs (ECGs), and thorough physical examinations reveal nothing out of the ordinary. Breathing pattern abnormalities and their sequelae (see below) are commonly missed by doctors and health care professionals, or else dismissed as 'over anxiousness', and no treatment options are offered.

The incidence of hyperventilation syndrome (HVS) and breathing pattern disorders (BPDs) is as follows:

- Up to 10% of patients in general internal medicine practice are reported to have HVS/BPD as their primary diagnosis, although

equivalent data are not available for emergency department presentations.

- There is a female preponderance in HVS/BPD that ranges from 2:1 to 7:1. The peak age of incidence is 15–55 years, although other ages can be affected. Women may be more at risk because of hormonal influences: progesterone is a respiratory stimulant and in the luteal phase (post ovulation to the onset of menstruation) CO_2 on average drops 25%. Added stress would 'increase ventilation at a time when carbon dioxide levels are already low' (Damas-Mora et al 1980).
- One study reported a series of 45 patients with chest pain who had normal coronary arteries on angiography and who were ultimately diagnosed as HVS. Over a 3.5 year average follow-up, 67% had made subsequent emergency department visits for chest pain and 40% had been readmitted to rule out myocardial infarction. Consequently, not only do HVS/BPDs produce severe and genuine discomfort for patients, they also account for considerable medical expense in excluding more serious pathology (Newton 2000).
- Acute hyperventilation is only about 1% of all cases of hyperventilation, and is well outnumbered by chronic hyperventilation (Lum 1975).

NORMAL BREATHING

Normal resting breathing rates are between 10 and 14 breaths per minute, moving between 3 and 5 liters of air per minute through the airways of the chest. During the active inspiratory phase, air flows in through the nose, where it is warmed, filtered, and humidified before being drawn into the lungs by the downward movement of the diaphragm and the outward movement of the abdominal wall and lower intercostal muscles. The upper chest and accessory breathing muscles remain relaxed. The expiratory phase is effortless as the abdominal wall and lower intercostals relax downward and the diaphragm ascends back to its original domed position aided by the elastic recoil of the lung. A relaxed pause at the end of exhalation releases the diaphragm briefly from the negative and positive pressures exerted across it during breathing (see Ch. 4, p. 93). Under normal circumstances people are quite unaware of their breathing. Breathing rates and volumes increase or fluctuate in response to physical or emotional demands, but in normal subjects return to relaxed low-chest patterns after the stimuli cease.

DEFINITION OF HVS/BPDs

Hyperventilation is a pattern of overbreathing, where the depth and rate are in excess of the metabolic needs of the body at that time. Breathlessness usually occurs at rest or with only mild exercise. Physical, environmental, or psychological stimuli override the automatic activity of the respiratory centers, which are tuned to maintain arterial carbon dioxide ($PaCO_2$) levels within a narrow range. At that particular time, the body's CO_2 production is set at a certain level, and the exaggerated breathing depth and rate eliminates CO_2 at a faster pace resulting in a fall in $PaCO_2$, or arterial hypocapnia. This results in the arterial pH (acid/alkaline balance) rising into the alkaline region to induce respiratory alkalosis (Fig. 2.1).

ORGANIC CAUSES OF INCREASED BREATHING

It is important to exclude organic causes, where breathlessness is an appropriate respiratory response to a physical disease causing diminished arterial oxygen saturation (PaO_2) and elevated arterial carbon dioxide ($PaCO_2$) levels. In true breathlessness, tachypnea (rapid breathing) or hyperpnea (increase in respiratory rate proportional to increase in metabolism), the respiratory centers are responding *automatically* to rising CO_2 production due to exercise or organic disease, and deeper and faster breathing response is appropriate.

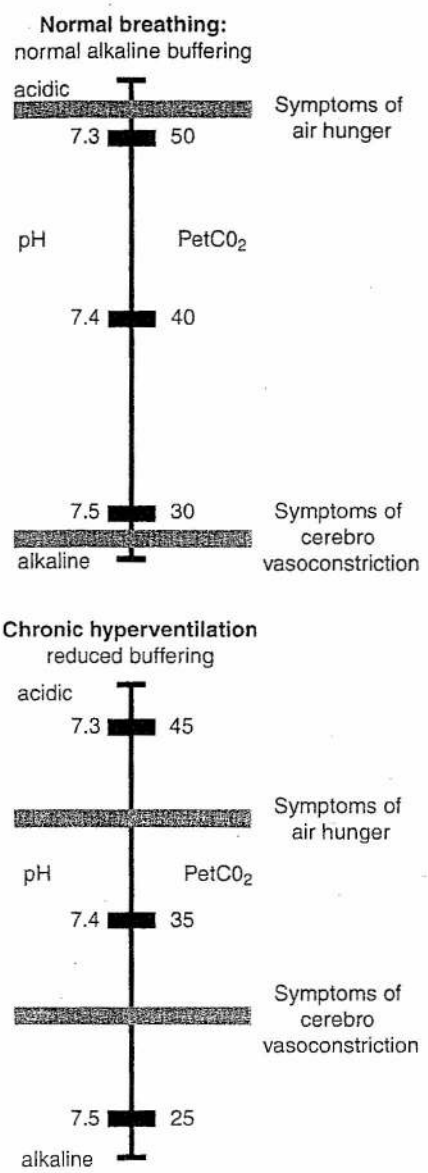

Figure 2.1 When hyperventilation continues for several hours or more, there is a drive to restore normal pH by excreting more alkaline buffer. This compensation permits CO_2 at a level of 35 mmHg to coexist with 7.4 pH. Such an individual becomes subject to reduced capacity to tolerate acidosis from any source, including breath-holding, and is also closer to the symptom line for reduced CO_2. (Reproduced from Gilbert 1999.)

⚠ **CAUTION:** It is dangerous to embark on a course of treatment for hyperventilation where the cause is an organic disease requiring *prompt* investigation and medical therapy. Table 2.1 lists the organic causes which should first be excluded, along with the main appropriate investigation(s) for them.

HISTORICAL BACKGROUND

The first description of hyperventilation in Western medical literature dates back to the USA, during the Civil War, when a surgeon published a paper entitled 'On irritable heart: a clinical study of a form of functional cardiac disorder and its consequences' (Da Costa 1871). The series of 300 soldiers studied suffered breathlessness, dizziness, palpitations, chest pain, headache, and disturbed sleep. The symptoms improved when the soldiers were removed from the front line, but their recovery was slow. Although Da Costa recognized the symptoms as functional in origin, he did not identify hyperventilation as the primary cause.

Physiologists Haldane & Poulton (1908) associated numbness, tingling, and dizziness with overbreathing. A year later. Vernon (1909) added an additional symptom, muscular hypertonicity. These symptoms occurred with respiratory alkalosis when patients were hyperventilating.

Kerr and colleagues (1937) introduced the term 'hyperventilation syndrome' (HVS) and pointed out the diversity and variability of symptoms in many systems of the body. Before these publications, a number of cardiologists following up Da Costa's syndrome had debated whether the heart was involved and coined phrases to fit in with their own views. Thomas Lewis (1940) used the terms 'soldier's heart' and 'effort syndrome' in relation to British soldiers in and after the First World War, whereas US cardiologists were reluctant to label the symptoms as cardiac or related to effort. They preferred the term 'neurocirculatory asthenia'.

These arguments were largely settled when Soley & Shock (1938) found that all the manifestations of 'soldier's heart' and 'effort syndrome' could be induced by hyperventilation and consequent respiratory alkalosis. Since then, many names have been given to this complex set of symptoms – changing with the fads of the time.

Table 2.1 Organic causes of breathlessness

System	Disease	Investigations
Respiratory	Asthma	Peak expiratory flow (PEF) and other lung functions
	Chronic obstructive respiratory disease	X-ray, PEF, and other lung functions
	Interstitial lung disease	X-ray, biopsy, and lung functions
	Pneumonia	X-ray, sputum, and white blood count
	Pulmonary embolus	X-ray, arterial blood gases, V/Q scan
	Pneumothorax	X-ray
	Pleural effusion	X-ray and aspiration
Cardiovascular	Acute and chronic left heart failure	X-ray, ECG, cardiac enzymes, and echocardiogram
	Right heart failure	
	Tachyarrhythmias	X-ray, ECG, cardiac enzymes, and echocardiogram, plus Holter monitor
	Pulmonary hypertension	X-ray, ECG, cardiac enzymes, and echocardiogram, Holter monitor, plus catheter studies
Hemopoietic	Anemia	Blood count and bone marrow
Renal	Nephrotic syndrome	Urine and serum albumin, chest X-ray
	Acute and chronic renal failure	Arterial blood gases, serum creatinine. Ultrasound of kidneys and urinary tract
Endocrine	Diabetes with ketoacidosis	Blood glucose, arterial blood gases, urine ketones
	Pregnancy	Pregnancy test and ultrasound
	Progesterone therapy	Identify medication
Metabolic	Liver failure	Liver function tests and serum albumin
Drugs	Aspirin	Identify drug and overdose
	Caffeine	Identify drug and daily intake
	Amphetamine	Identify drug and daily intake
	Nicotine	Identify drug and daily intake

With thanks to David Scott MB ChB BMedSc FRCP(Lond) FRACP

'Designer jeans syndrome' (Perera 1988) was popular in the 1970s, and the current so-called Gulf and Balkan War syndromes include many of the same signs and symptoms. Broadly speaking, HVS/BPD was accepted as being of psychiatric origin in the USA and readily diagnosed, whereas in the UK physicians were reluctant to recognize it. A number of factors may have been operating. Most of the reports were in psychological and psychiatric literature, unnoticed by general practitioners and physicians. Influential UK cardiologist Paul Wood (1941) had reviewed Da Costa's syndrome and firmly placed it in the hands of the psychiatrists. Sadly there was little dialogue between the two specialties.

More recently, chest physician Claude Lum (1977), writing from the Addenbrooke and Papworth hospitals in Cambridge, England, with physiotherapists Diana Innocenti (1987) and Rosemary Cluff (1984), who developed assessment and treatment programs, has done much to enlighten the medical practitioners in the UK and reignite scientific interest and research into the condition. Since that time there has been a flowering of literature on the subject as more sophisticated and accessible research equipment has become available. Recommended reviews are listed in Box 2.1.

Despite such progress, there are still considerable numbers of cardiologists, general and specialist physicians, or general practitioners who are reluctant to diagnose or seek treatment for their patients with hyperventilation. Endless, increasingly sophisticated, tests are carried out. Or, alternatively, patients are referred to further specialists for symptoms related to other fields, or they are told 'nothing is wrong' with them. As hyperventilation has no reliable, repeatable, or easily performed diagnostic tests, investigations are protracted, the diagnosis is avoided, and the

Box 2.1 Recommended reviews of hyperventilation disorders

Brashear R E 1983 Hyperventilation syndrome. Lung 161: 257–273
Cowley D S, Roy-Byrne P R 1987 Hyperventilation and panic disorder. American Journal of Medicine 83: 929–937
Gardner W N 1996 The pathophysiology of hyperventilation disorders. Chest 109: 516–534
Grossman P 1983 Respiration, stress, and cardiovascular function. Psychophysiology 20(3): 284–300
Nixon P G F 1993 The grey area of effort syndrome and hyperventilation. Journal of the Royal College of Physicians of London 27(4): 377–383
Timmons B H, Ley R (eds) 1994 Behavioural and psychological approaches to breathing disorders. Plenum, New York

patient's file is often relegated to the 'too hard' basket. This puts patients at great risk of invalidism or of being labeled as malingerers. Medical historians have suggested, for example, that the chronic invalidism of Florence Nightingale and Charles Darwin in the 19th century was more likely chronic hyperventilation, rather than heart disease resulting from infections picked up in the Crimea and the Andes respectively, as was previously believed (Timmons & Ley 1994).

SYMPTOMS AND SIGNS OF HYPERVENTILATION

Table 2.2 lists the diverse symptoms and signs of hyperventilation. None is absolutely diagnostic. Consequently, clinicians rely on a suggestive group of symptoms. Each patient has a characteristic set of symptoms which can be amplified during an acute episode or when hyperventilation is exaggerated. The intermittent nature and variable intensity of the symptoms adds to the difficulty of diagnosis. In addition, many patients fail to mention some of their symptoms, either because they think they are unrelated or because they are ashamed to discuss them. Examples are hallucinations, phobias, sexual problems, fear of impending death or madness, and nightmares. Careful interrogation about the relationship of breathlessness to exercise usually reveals a variation in severity from day to day.

ACUTE HYPERVENTILATION

The diagnosis of an acute episode, either witnessed by the clinician or recalled by the patient, is relatively easy. The patient appears distressed, the pattern of respiration involves deep and rapid breaths using the accessory muscles visible in the neck and the upper chest. Wheezing may be heard as a result of bronchospasm triggered by hypocapnia. Oxygen saturations (measured by pulse oximetry, see p. 178) are within normal ranges (95–98%), and are commonly up to full saturations of 100%. A stressful precipitating event is usually reported.

Neurological signs

Hypocapnia reduces blood flow to the brain (2% decrease in flow per 1 mmHg reduction in arterial CO_2), causing frightening central nervous system symptoms. Poor concentration and memory lapses may result, with tunnel vision and onset in those susceptible of migraine-type headaches or tinnitus. Sympathetic dominance brings on tremors, sweating, clammy hands. Palpitations, and autonomic instability of blood vessels causing labile blood pressures (Magarian 1982). Bilateral perioral and upper extremity paresthesiae and numbness may be reported. Unilateral tingling is most often confined to the left side. Dizziness, weakness, visual disturbances, tremor, and confusion – sometimes fainting or even seizures – are typical symptoms. Spinal reflexes become exaggerated through increased neuronal activity caused by loss of CO_2 ions from the neurons. Tetany and cramping may occur in severe bouts (Fried & Grimaldi 1993).

Metabolic disturbances

Two tests of nerve hyperexcitability produced by hypocapnia-induced hypocalcemia are

Table 2.2 Symptoms and signs of hyperventilation

System	Symptom/sign	Probable cause
Neurology	Headache	Cerebrovascular constriction
	Numbness and tingling extremities, more often the left hand and perioral	Neuronal excitability from alkalosis or hypocalcemia, or flux in ionized calcium
	Positive Trousseau's and Chvostek's signs	Neuronal excitability from alkalosis or hypocalcemia or flux in ionized calcium
	Giddiness and dizziness	Vasoconstriction of the vertebral arteries and reduced O_2 availability
	Ataxia and tremor	Vasoconstriction of the vertebral artery and reduced O_2 availability
	Blurred and tunnel vision	Vasoconstriction of the carotid arteries and reduced O_2 availability
	Anxiety and panic	
	Phobias	
	Irritability	
	Depersonalization	
	Detachment from reality	
	Impaired concentration, thinking, performance, and affect	
	Poor stamina	
	Disturbance of sleep, nightmares	
	Hallucinations	
Cardiovascular	Chest pains and angina	Reduced coronary blood flow
	Palpitations and arrhythmias	Changed excitability of SA and AV nodes and cardiac muscle
	Tachycardia	Compensatory for reduced cardiac output and reduced blood pressure
	Lightheadedness and syncope	Reduced cardiac output and blood pressure from peripheral vasodilatation
	ECG changes with ST depression or elevation and prolonged QT interval and sometimes T wave inversion	Reduced coronary blood flow
	Associated conditions: — Mitral valve collapse — Prinzmetal's angina	Reduced coronary blood flow
Respiratory	Breathlessness and inability to take a deep breath, often nocturnal	
	Sighing and yawning	
	Upper-chest breathing and use of accessory muscles in the neck	
	Chest wall tenderness	Muscle fatigue
	Two hand test, one on upper sternum and the other on upper abdomen – top one moves more	
	Voluntary hyperventilation and end-tidal Pco_2 measurement	
	Dry, non-productive cough and mannerism of clearing the throat	
	Wheezing	Bronchospasm from dry air and activation of eicosanoid and muscarinic receptors
Muscular	Aching and stiffness due to hypertonicity	The motor nerve hyper-excitability
	Limb weakness	Muscle fatigue
	Cramps, carpopedal spasm and tetany	Hyper-excitability of motor nerves
Gastrointestinal	Lower chest and epigastric discomfort	
	Esophageal reflux and heartburn	
	Upper abdominal distension	Air swallowing and stomach distension
	Dry mouth	Mouth breathing
	Mannerism of air swallowing and belching	
Skin	Sweating	Cutaneous vasoconstriction

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Trousseau's sign and Chvostek's sign. The Trousseau test consists of occluding the brachial artery into the arm by pumping the blood pressure cuff above the systolic pressure for 2.5 minutes. A positive sign is where paresthesia is felt severely within the period and the wrist and fingers arch in carpopedal spasm – termed 'main d'accoucheur' or obstetrician's hand. The Chvostek sign is when tapping the facial nerve at the point where it emerges through the parotid salivary gland elicits a contraction of the facial muscle which twitches the side of the mouth. This is also a test for magnesium deficiency (Werbach 2000).

Acute hypophosphatemia also contributes to weakness and tingling.

Cardiac signs

Chest pain is another alarming symptom challenging the clinician to exclude heart disease. Epinephrine-induced ECG changes can occur in hyperventilation uncomplicated by coronary heart disease. One study suggests that up to 90% of non-cardiac chest pain is thought to be induced by HVS/BPD (De Guire et al 1992).

In older patients, established coronary artery disease can be exacerbated by vasoconstriction arising from hypocapnia, putting these patients at risk of coronary occlusion and myocardial damage. Alternatively, hyperventilation can trigger spasming of normal caliber coronary arteries. This type of variant angina (Prinzmetal's angina) occurs without provocation, usually at rest. This phenomenon is prevented by calcium channel blockers, which reduce calcium ion migration from the cells. To date, no specific studies of breathing retraining and outcome measurements for this type of angina have been done.

Syndrome X refers to those patients with a history of angina and a positive exercise test (chest pain within 6 minutes or less), yet who have normal angiography. Thought to be a functional abnormality of coronary microcirculation, it is much more common in women than in men (Kumar & Clark 1998).

Gastrointestinal signs

Rapid and/or mouth breathing instigates aerophagia from air gulping, causing bloating, burping, and extreme epigastric discomfort. Irritable bowel syndrome (IBS) is listed as a common symptom of chronic overbreathing. Fear and anxiety may induce abdominal cramps and diarrhea (Lum 1987). The median swallowing rate in healthy, non-dyspeptic controls is 3 or 4 per 15 minutes. In the absence of food, up to 5 ml of air accompanies saliva into the gastrointestinal tract with each swallow (Calloway & Fonagy 1985).

Some clinicians think aerophagia may exacerbate hiatus hernia (part of the stomach passes up through a weakened esophageal valve

Case study 2.1 Acute hyperventilation

A 39-year-old man with profound deafness from past middle ear infections came into the emergency department with a severe headache, abdominal pain, and an inability to stand and walk. Numbness and pins and needles also affected his legs. He looked and felt distressed. Five doctors assessed him in the course of the 12 hours he spent in the department. The history obtained was fragmentary because a person skilled in sign language was not sought. The medical registrar called in the physician, who ordered a CT scan of the patient's head because of the headache, paresthesiae, and paraparesis. The CT scan was normal. They noted an increased respiratory rate but dismissed this finding, as both chest X-ray and ECG were normal and there were no abnormal cardiac signs. The surgical registrar and surgeon considered the abdominal pain warranted an ultrasound of the patient's abdomen and were relieved that this was normal. As the day wore on the symptoms improved, and the patient and the fifth doctor were reassured by the normal investigations. It was therefore decided to send him to the ward pending an investigation of a spinal cord lesion. Overnight, a sympathetic nurse found out that the man had just lost his 48-year-old partner. Her grown-up children had removed a lot of the patient's furniture from his house, believing it to be their mother's, and one of her sons had written off the patient's (uninsured) car in a crash. This set of misfortunes – bereavement, loss and depression – set up the acute hyperventilation, less apparent on admission, when the consequent symptoms were dominant. Eventually a signer was brought in and the symptoms and hyperventilation explained. The patient responded to this explanation and to counseling.

in the diaphragm into the chest cavity), or may even be the cause in susceptible people. Case study 2.1 describes a patient with abdominal pain following acute hyperventilation.

CHRONIC HYPERVENTILATION

The diagnosis of chronic and intermittent hyperventilation is more difficult, as the patient will often only present when having an acute episode on top of chronic hyperventilation.

Often the patient will dwell on one symptom in a particular system and will be referred to a specialist, for example a cardiologist, gastroenterologist, neurologist, psychiatrist, or respiratory physician. Each will diagnose and investigate within the particular speciality, delaying the diagnosis for months, or even years. Some patients have such a thick folder of notes, including all their previous tests, that this in and of itself is considered diagnostic by some experienced physicians – the 'fat folder syndrome' (Lum 1975).

A careful history and systemic inquiry, checking all other symptoms in the other body systems, usually highlights a suspicious pattern, particularly to the experienced clinician who can think beyond his own special interest. There are often some symptoms which do not fit with the referred complaint and provisional diagnosis.

Careful inquiries as to the precipitating causes of attacks helps both with the diagnosis and with focusing on choice of treatment. There are often attacks where there is no preceding stressful event. It is thought that in those with chronic hyperventilation the respiratory centre is reset to tolerate a lower than normal partial pressure of arterial carbon dioxide ($PaCO_2$) in the blood (Nixon 1993). In such patients a single sigh or one deep breath will reduce the $PaCO_2$ enough to bring on symptoms.

Examination must exclude organic diseases of the brain and nervous system, diseases of the heart (particularly angina and heart failure), respiratory disease, and gastrointestinal conditions, especially if there are suspicious symptoms in these systems.

SPECIAL AND LABORATORY TESTS

The following tests are usually carried out by the patient's doctor, or in a laborotary setting as requested by the doctor:

- There are preliminary tests to exclude respiratory and cardiac disease: peak expiratory flow (PEF) rate, chest X-ray, and electrocardiogram (ECG). Where chest pain is a presenting symptom an exercise ECG is done, and during the hyperventilation provocation test (HVPT) (in which the patient is asked to voluntarily overbreathe to bring on symptoms) the ECG should be monitored.
- Arterial blood gas determination is an invasive and painful test (arterial puncture) appropriate in the emergency room where the diagnosis of acute hyperventilation is required. With patients in whom chronic hyperventilation is suspected, the end-tidal carbon dioxide ($PETCO_2$) can be measured. $PETCO_2$ is equivalent to $PaCO_2$ in subjects unstressed and with normal lungs. It can be measured non-invasively from a continuous sampling through nasal prongs, with the mouth occluded, or for those with nasal obstruction the tube can be sited in an oral airway to monitor CO_2 deficits.

  The patient can be put through a 4-minute quiet breathing rest period, followed by exercise and recovery, or one may do an HVPT test in the recovery period. Most patients with chronic hyperventilation will have a $PETCO_2$ at or below 30 mmHg and a markedly delayed recovery from hypocapnia after overbreathing (Chambers et al. 1988).
- Some clinicians place a 'think test' (Nixon-Freeman 1988) 3–4 minutes into the recovery period. The patient is asked to recall a painful emotional experience where symptoms developed. If the $PETCO_2$ drops 10 mmHg, the test supports hyperventilation. In a non-laboratory setting a modified version of this test may also be used to provoke symptoms, as breathing pattern change during disclosure of emotionally charged events. Subjective symptoms are recorded instead of CO_2 levels (see Ch. 8).

Table 2.3 Respiratory function tests and exercise tests (from Kumar & Clark 1998 with permission)

Test	Use	Advantages	Disadvantages
PEFR	Monitoring changes in airflow limitation in asthma	Portable Can be used at the bedside	Effort-dependent Poor measure of airflow limitation
FEV, FVC, FEV_1/FVC	Assessment of airflow limitation The best single test	Reproducible Relatively effort-independent	Bulky equipment but smaller portable machines available
Flow–volume curves	Assessment of flow at lower lung volumes Detection of large airway obstruction both intra- and extra-thoracic (e.g. tracheal stenosis, tumor)	Recognition of patterns of flow–volume curves for different diseases	Sophisticated equipment needed
Airways resistance	Assessment of airflow limitation	Sensitive	Technique difficult to perform
Lung volumes	Differentiation between restrictive and obstructive lung disease	Essential adjunct to FEV_1	Sophisticated equipment needed
Gas transfer	Assessment and monitoring of extent of interstitial lung disease and emphysema	Non-invasive (compared with lung biopsy or radiation from repeated chest X-rays and CT)	Sophisticated equipment needed
Blood gases	Assessment of respiratory failure	Can detect early lung disease when measured during exercise	Invasive
Pulse oximetry	Postoperative, sleep studies, and respiratory failure	Continuous monitoring Non-invasive	Measures saturation only
Exercise tests (6 min walk)	Practical assessment for disability and effects of therapy	No equipment required	Time consuming Learning effect At least two walks required
Cardiorespiratory assessment	Early detection of lung/heart disease Fitness assessment	Essential in differentiating breathlessness due to lung or heart disease	Expensive and complicated equipment required

In some patients with hyperventilation the $PaCO_2$ and the $PETCO_2$ may be in the normal range. In those who are asymptomatic at the time of testing, this finding could be accepted. However, a normal level while experiencing symptoms negates hypocapnia as the cause of symptoms. It prompts a search for an alternative explanation.

- A peak expiratory flow (PEF) measurement (compared with age, sex, and height tables) provides a simply done, quick exclusion of significant respiratory restriction in the clinic room. Further lung function tests would be scheduled if signs of respiratory obstruction or restriction or cardiac disease was suspected. These might include the tests listed in Table 2.3. Examples of the first common initial tests are described in Figure 2.1.
- Resting ECG and chest X-rays are usually routinely done in patients presenting to emergency medical centers with chest pain.
- The breath-holding time test is done in the clinic and does not require additional measurements or equipment. The time a hyperventilating patient can breath-hold is usually greatly reduced, often not beyond 10–12 seconds – 30 seconds has been used as the approximate dividing line between hyperventilators and normals by some clinicians. It is worth noting that breathless patients *without* hyperventilation may have equal difficulty in breath-holding (Gardner 1996).
- Voluntary overbreathing is a useful test to reproduce symptoms – the hyperventilation provocation test (HVPT) – in a laboratory setting. If there is simultaneous measurement of end-tidal $PETCO_2$ during the test and recovery, the

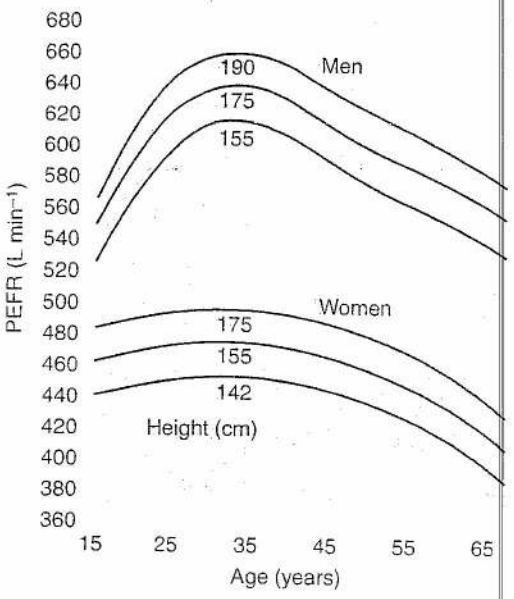

Figure 2.2 Graph of normal readings (from Kumar & Clark 1998 with permission).

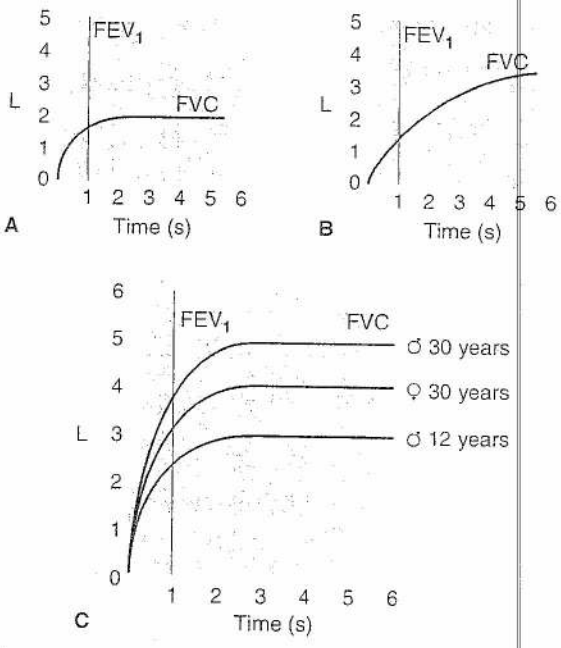

Figure 2.3 Graphs showing **A** restrictive pattern FEV_1 and FVC reduced **B** airflow limitation (FEV_1 only reduced) and **C** normal patterns for age and sex (from Kumar & Clark 1998 with permission).

slow return of this measurement from hypocapnia can be diagnostic. If chest pain is a presenting symptom, ECG monitoring is desirable.

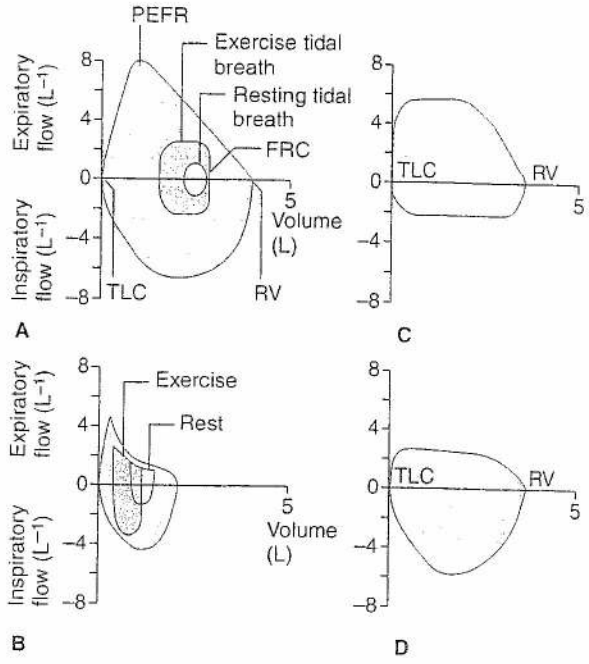

Figure 2.4 A and B. Maximal flow volume loops, showing the relationship between maximal flow rates of expiration and inspiration (a) in a normal subject (b) in a patient with severe airflow limitation. **C and D.** Flow volume loops of patients with large airway (tracheal) obstruction showing plateauing of maximal expiratory flow high in the lung volume (from Kumar & Clark 1998 with permission).

⚠ **CAUTION:** As this test can provoke cardiac arrhythmias, with the possibility of a coronary event in those with perhaps undiagnosed coronary artery disease, it is best used *only* where there is medical back-up on site.

The test is best done before explanations of symptoms, to prevent suggestion and bias. Patients need to be warned only of a dry mouth. The patient is asked to concentrate on how they feel during the 1–2 minute period when they are overbreathing at the rate of 30–40 per minute. The rate is set by the examiner's hand movements. The operator must stress the importance of the test and the need to continue for as long as the patient can. An arterial blood gas determination at the end of the test can be of use to establish the depth of hypocapnia. Some clinicians rely on as little as 12 deep breaths which the patient can recover from easily, and subjective symptoms produced are recorded.

Case study 2.2 An example of chronic HVS

A 38-year-old woman was referred for an exercise ECG because of chest pain at rest and on exercise, though its relation to exercise was variable. The test was normal, but a detailed history was suspicious. She was a happily married woman with two children who were doing well. A senior bank officer, she had been promoted to a position for which she had had no past experience, nor was she given any orientation. She was meticulous in her work and it frustrated her that immediate mastery of the new job eluded her. This promotion and the move into a new house had coincided with the onset of the chest pain. The pain was left sub-mammary, coming on mainly during rest, but sometimes with light exercise. It was associated with breathlessness and some tingling of the left arm and around her mouth. She had previously enjoyed walking and keeping fit by going to the gym 2 days a week, but she had to give up these pastimes because of the chest pain and breathlessness. She had an anxious air. Though her respiratory rate was normal, the two-hand test revealed an intercostal breathing pattern. Significant respiratory and cardiac disease was excluded by a normal PEF, chest X-ray, and normal resting and exercise ECGs. An HVPT was positive in producing chest pain and paresthesiae, and the ECG during the pain was normal.

She was relieved by the normal tests and quickly saw the logic of the multiple stressors generating symptoms from hyperventilation. Over 6 weeks her attacks diminished and eventually stopped as she reduced her working hours and had breathing retraining with an experienced respiratory physiotherapist.

- In both the breath holding and voluntary over breathing tests, the skills of the clinician are important for maintaining the trust and cooperation of patients.

DIAGNOSIS

Clinical diagnosis of primary HVS/BPD would be made on the findings of specific or relevant tests done, and after exclusion of organic disease. Case study 2.2 gives an example of a patient with chronic HVS.

When considering the myriad of balanced biochemical reactions which make up normal metabolism, and their dependence on the careful maintenance of an optimal pH, it is not surprising that respiratory alkalosis resulting from hyperventilation causes such widespread disturbances and symptoms (Fig. 2.5; see also Ch. 3). Out of the mass of data some major threads emerge – changes in vasomotor tone, mainly reducing cerebral, coronary, and cutaneous blood flow, diminished oxygen availability to tissues, and increased neuronal excitability of the peripheral nervous system:

- Buffering mechanisms protect the pH by renal excretion of bicarbonate (HCO_3). If this compensation is prolonged chronic loss of buffer base reserves further stimulates respiration to avoid metabolic acidosis.
- Oxygen uptake is impaired by a leftshift of the oxyhaemaglobin dissociation curve (Bohr effect) leading to sensations of breathlessness (Fig. 2.6). As $PaCO_2$ is depleted, there is a linear reduction in cerebral blood flow (see above). The respiratory centre becomes reset to a lower CO_2 threshold. A chronic exhausting circle is established (Nixon 1993).
- Proton magnetic resonance spectroscopy reveals increased levels of brain lactate in patients with hyperventilation. These abnormalities would explain all the profound neurological symptoms except tingling, numbness, muscle hypertonicity, and carpopedal spasm.
- Neuronal hyperexcitability is related again to falling CO_2 levels. These result in elevated sensory and motor nerve potentials and bursts of spontaneous discharges, giving rise to paresthesiae of the hands, trunk, and around the mouth as well as hypertonicity, cramps, and carpopedal spasm of the muscles. The mechanism is uncertain. There is a dramatic lowering of serum phosphate which may influence calcium flux; calcium ions stabilize the nerve membrane potential. The same symptoms occur with hypocalcemia, but there is no evidence of reduced serum calcium levels in respiratory alkalosis (Magarian 1982).
- Hyperexcitability of cardiac muscle fibers and electrical conducting systems may cause atrial cardiac arrhythmias.
- As $PaCO_2$ falls there is a reduced coronary blood flow with myocardial hypoxia due to vasoconstriction, and sometimes spasming of coronary arteries. This may be one cause of

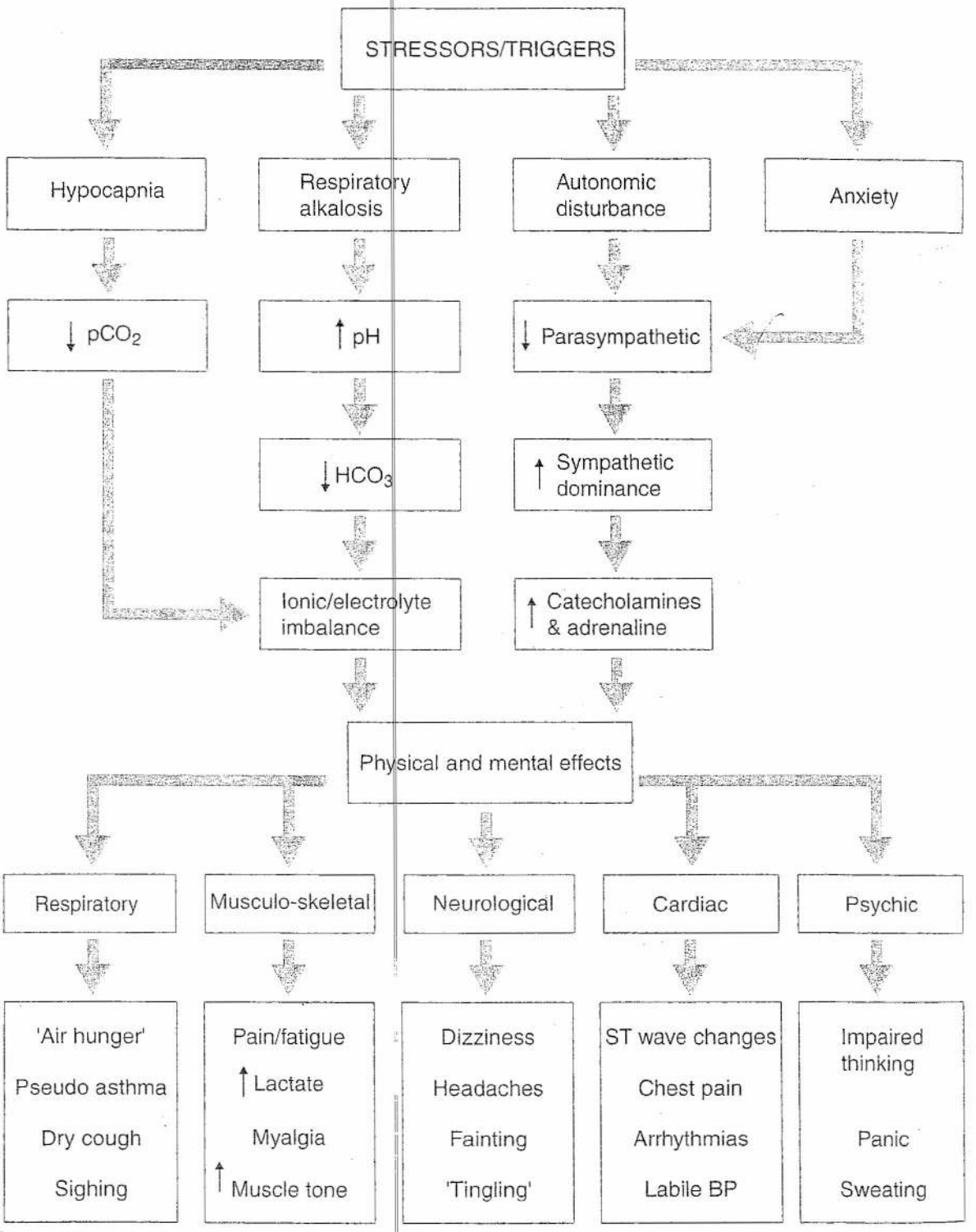

Figure 2.5 Pathophysiological explanations of the symptoms. (Adapted from Bradley 1994.)

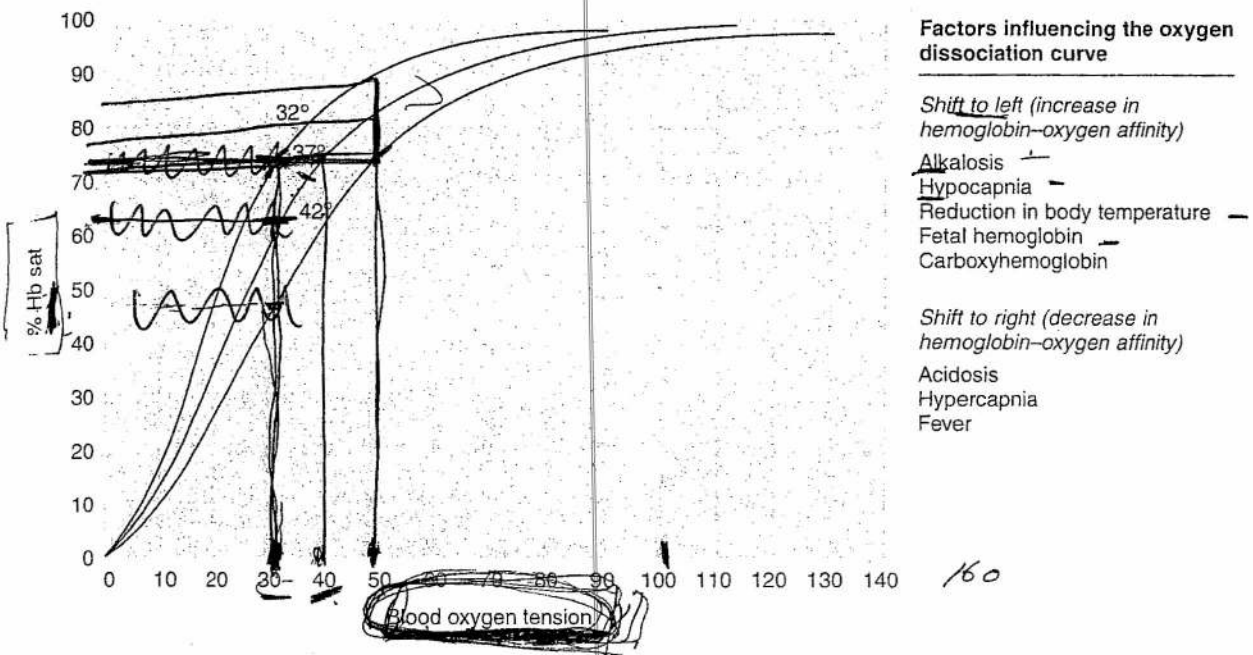

Figure 2.6 Oxygen dissociation curve of blood at a pH of 7.4 showing variations at three temperatures. For a given oxygen tension, the lower the blood temperature the more the hemoglobin holds onto its oxygen, maintaining higher saturation. (Reproduced from Wilkins et al 2000.)

anginal chest pain, which is of particular relevance to those with pre-existing coronary artery disease, with the risk of dislodging plaque and precipitating occlusion/infarction/death.

Chest pain in hyperventilation may also stem from:

- Sharp pains felt on inspiration from *pressure on the diaphragm* from aerophagia ('air gulping' from mouth breathing) (Evans & Lum 1981).
- A typical dull and diffuse pain due to *intercostal muscle fatigue and spasm* (Evans & Lum 1981; see also Ch. 6).
- Heavy retrosternal pain, sometimes radiating to the neck and arms, which mimics angina, lasts longer, and does not abate at rest nor become worse with continued activity as with classic angina; nor does it respond to nitrolingual sprays (anti-anginal medications) (Magarian 1982).
- *Esophageal reflux* is another source of central chest discomfort outside the respiratory system.

Case study 2.3 gives an example of panic breathing.

Hyperventilation may initiate bronchoconstriction in non-asthmatic subjects. As hyperventilators are often mouth breathers, air entering the bronchi is dry and increases the airway fluid osmolarity, rendering it more sticky and tenacious. This stimulates nicotinic and muscarinic receptors, releasing prostanoids and leukotrienes, which cause bronchospasm and mucosal damage.

During hyperventilation there is an additional reduction in peripheral vascular resistance, with a drop in mean arterial blood pressure. This can result in fainting or extreme light-headedness. However, compensatory increases in heart rate and cardiac output supervene with blood pressure (BP) rising above baseline, causing elevated or fluctuating BP levels.

Case study 2.3 Example of panic breathing (adapted from Gilbert (1998)).

A middle-aged woman presented with panic attacks specific to driving, severe enough to limit her to about a 3-mile radius from her home. Her first panic attack had occurred 2 weeks previously as she was driving in light traffic on a city street. Her symptoms had a large component of hyperventilation, with consequent dizziness, anxiety, and chest tightness. She feared that she might lose control of the car if she went too far from her home, and believed that her overbreathing was a consequence of the anxiety rather than contributing to it. Medical examination had not provided any explanations and she had been given a minor tranquilizer, but medication frightened her and so she refused it.

Since the panic had started only 2 weeks before, she was questioned closely about the context of the first attack, in the expectation that her memory would still be fresh. Her general account of how the problem started shifted gradually from 'It came out of nowhere' to 'I was just driving to the hospital to visit my husband. I was a bit frustrated with him that day. I was furious with him, but couldn't admit it. I guess.' This history-gathering over two sessions was alternated with instruction on how to regulate her breathing (mouth closed, abdominal inhale, slow exhale, pause and relax) and suggestions to gradually expand her driving range while practicing this procedure.

The whole story finally emerged. Her husband had been hospitalized for investigation of a mild cardiac incident and was actually enjoying his hospital stay, but did not have the good judgment to keep this to himself. While his wife struggled to carry out the family chores, care for the children, work part-time, and dutifully visit her husband every day, he was telling her how pretty the nurses were and how he could watch all the TV he wanted. She brought him special treats and diversions from home, but apparently felt in competition with the nurses. Her rage grew and swelled against the containment of her prohibition against expressing anger at a sick man. This, it was surmised, had stimulated the ragged, frustrated breathing so typical of anger in conflict with niceness. As she related her feelings about her husband, her breathing style changed to thoracic, open-mouthed, and hyperinflating, and she felt at times some of the familiar dizziness and disorientation.

She finally accepted the probability that, by her own testimony, her first panic attack had definitely not come out of nowhere. The panic had solved her immediate conflict by curtailing her hospital visits until her husband was sent home (the hospital was about a mile beyond the edge of her safe perimeter). But her overbreathing had probably become conditioned to the experience of driving, and she still felt unsafe behind the wheel. During weekly sessions she discussed how symptoms of panic could emerge from suppressed rage affecting her breathing, and spoke more freely about her marriage and about how she managed feelings of anger in general. She continued to practice the breathing control. Within a month the panic attacks subsided and her driving ability returned to normal.

BREATHING PATTERN DISORDERS SECONDARY TO OTHER HEALTH PROBLEMS

Observation of changes to patients during primary hyperventilation and disordered breathing patterns also needs to extend to disorders in which hyperventilation prevails as a coexisting complication. The aim is to provide a broad overview of common conditions and alert clinicians to secondary breathing pattern dysfunctions in patients which may complicate the original disorder and/or add unwarranted stressors.

Obstructive disorders

Starting with the most common obstructive lung disorders (asthma, chronic obstructive airways disease (COAD) and emphysema), airways obstruction may be due to:

- Reversible factors (as in asthma), e.g. inflammation, bronchospasm, or mucus plugging
- Irreversible factors (as in emphysema and chronic bronchitis) e.g. damaged alveoli leading to loss of elastic recoil of adjacent lung tissue, or scarred airway walls (Hough 1996).

Careful viewing of Figure 2.7 shows that there is a great deal of overlapping between these disorders, with mixing and matching of signs and symptoms. Clear definitions are sometimes elusive.

Restrictive disorders

Restrictive lung disorders, with reductions in both lung volumes and lung compliance, include:

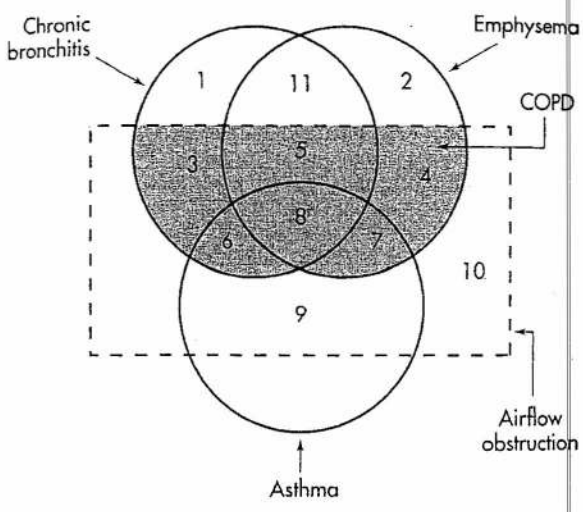

Figure 2.7 Schema of chronic obstructive pulmonary disease (COPD). This nonproportional Venn diagram shows subsets of patients with chronic bronchitis, emphysema, and asthma. The subsets composing COPD are shaded. Subset areas are not proportional to actual relative subset sizes. Asthma is by definition associated with reversible airflow obstruction, although in variant asthma special maneuvers may be necessary to make the obstruction evident. Patients with asthma whose airflow obstruction is completely reversible (*subset 9*) are not considered to have COPD. Because in many cases it is virtually impossible to differentiate patients with asthma whose airflow obstruction does not remit completely from persons with chronic bronchitis and emphysema who have partially reversible airflow obstruction with airway hyperreactivity, patients with unremitting asthma are classified as having COPD (*subsets 6, 7, and 8*). Chronic bronchitis and emphysema with airflow obstruction usually occur together (*subset 5*), and some patients may have asthma associated with these two disorders (*subset 8*). Individuals with asthma who are exposed to chronic irritation, as from cigarette smoke, may develop a chronic, productive cough, a feature of chronic bronchitis (*subset 6*). Such patients are often referred to as having *asthmatic bronchitis* or the *asthmatic form of COPD*. Persons with chronic bronchitis and/or emphysema without airflow obstruction (*subsets 1, 2, and 11*) are not classified as having COPD. Patients with airway obstruction caused by diseases with known etiology or specific pathology, such as cystic fibrosis or obliterative bronchiolitis (*subset 10*), are not included in this definition. (From Scanlan et al 1999.)

- Acute inflammations such as pleurisy or pneumonia
- Chronic disorders, often under the collective term, interstitial lung disease (ILD) which covers over 200 variants. Examples of these are fibrosing alveolitis, sarcoidosis, asbestosis, bird fancier's lung, pneumoconiosis ('coal miners lung')

Breathing patterns

Breathing patterns often provide clues as to the type of condition involved:

- Rapid shallow upper-chest breathing suggests loss of lung volume seen in restrictive diseases, where the work of breathing is increased to maintain ventilation
- Prolonged exhalation time as witnessed in someone having an asthma attack indicates acute intrathoracic obstruction
- Prolonged exhalation (perhaps 'pursed-lip' in severe cases) caused by chronic intrathoracic obstruction in patients with COAD
- Prolonged inspiratory time occurs in acute upper airway obstruction as in croup or globus (throat spasm) (Wilkins et al 2000).

In all the above, accessory muscle use would be clearly visible, and mouth breathing would probably be the chosen route to move air in and out of the lungs. Assisting patients to reduce the work of breathing and diffuse anxiety, with the use of rest positions and relaxation techniques, may be of benefit (see Ch. 8).

Case study 2.4 describes the experience of a patient with a breathing disorder mistakenly diagnosed as chronic hyperventilation.

Patients who are recovering from acute chest infections or asthma attacks require 'debriefing' to ensure correct breathing patterns are restored. This is particularly important in those with asthma, as hyperventilation is a very common secondary problem which may trigger attacks. Lowered CO_2 levels from chronic hyperventilating encourage catecholamine and histamine release into the blood, which in turn stimulates mast cells in the lung parenchyma, promoting bronchoconstriction and hyperinflation. Inhaling cold air via the mouth has also been shown to trigger bronchoconstriction (Gardner 1996).

Those working in pulmonary rehabilitation programmes with COAD patients need to be aware of breathing retraining measures to help maximize respiratory function and encourage relaxed breathing. Mild to moderately affected patients benefit more than those with more severe disease with diaphragm flattening and

Case study 2.4 An example of a non-HVS/BPD diagnosis

A 44-year-old woman was referred to physiotherapy by her family doctor for assessment and treatment of chronic hyperventilation following a very stressful period at work, and the decision to leave her marriage. Her main symptoms were chest pain and exertional shortness of breath, and overt upper thoracic breathing. Her doctor had carried out an ECG which was normal, as were the results of a routine blood test.

The woman ran her own successful business, exercised regularly at a gym, and looked extremely fit and well. She had a history of smoking (from the age of 14) but had stopped 6 years ago (aged 38). She had no children, and her periods were regular with no history of low blood iron levels or PMS (premenstrual syndrome). She complained of increasing shortness of breath during exercise and noticed this particularly at work climbing stairs. She was clearly worried by this, and was generally anxious and tearful.

The patient had no history of wheeze or productive cough. Her Nijmegen score (see p. 176) was negative at 20/64, the highest scores across the listed symptoms were breathing related. Her upper thoracic trigger points were exquisitely tender and she was a habitual mouth breather. Her PEF was slightly lower at 370 liters a minute (l/min) than the normal predicted for her age and height (420 l/min).

The patient's resting O_2 saturations were 93% and she desaturated down to 90% on a preliminary exercise test walking upstairs. She was immediately re-referred on to a respiratory specialist physician for further lung function tests and investigations. Spirometry revealed a moderate loss of lung volume and special blood tests revealed she had an α_1-antitrypsin deficiency (sometimes called *genetic emphysema*. This deficiency prevents the protective effect of the protein α_1-antitrypsin against break-down of lung elastin which supports the alveolar walls of the lung. Her hyperventilation apart from her social stressors signaled a serious underlying obstructive lung disease. During a follow up telephone conversation the woman wondered why her doctor had not sent her straight to a specialist in the first place and considered that it might be sex stereotyping, pigeonholing her as a highly anxious and neurotic woman. The irony of this case was that her soon-to-be-ex husband, who had also been experiencing chest pain, had been sent straight to a cardiologist by his doctor, and was diagnosed as having hyperventilation syndrome.

reduced respiratory competence. But breathing assessment and retraining where necessary make a useful adjunct to pharmacological therapies and in some cases help patients safely to reduce anxiety levels and medications.

PRE- AND POST-SURGICAL BREATHING PROBLEMS

Patients waiting to undergo an operation may hyperventilate in response to fear and pain, which may persist after surgery. Those who have been in chronic pain for long periods (waiting for hip replacement, for instance) may be especially vulnerable to HVS/BPDs.

Coronary bypass patients facing open-chest surgery are often briefed beforehand on the importance of deep breathing exercises to expand and clear the lungs of mucus secretions in order to prevent chest infection. Some at-risk patients (perhaps with coexisting COAD from smoking) are given incentive inspirometers – hand-held devices to breathe through which make the work of inhalation harder, encouraging air entry down to the lung bases.

Part of the debriefing before the patient leave hospital (and as part of any coronary rehabili ation programs) should be the restoration c normal, relaxed nose/abdominal breathing Closure of coronary bypass grafts may be consequence of vasoconstrictive or vasospasti influences of chronic hyperventilation (Nixo 1989).

CONCLUSION

HVS/BPDs are common problems affecting th health of 10% of the normal population (Newto 2000). Doctors and other health care professior als need to be aware of the effects of depletion c the body's buffering systems in response t chronic hyperventilation. They can be alerted b clearly visible abnormal changes in breathin patterns and postural changes, and this shoul prompt a search for symptoms and signs. (Th subsequent application of diagnostic tests an the establishment of diagnoses and treatment are discussed in later chapters.) Checking respi

atory rates and breathing patterns should be an essential part of all health care investigations, with treatment options offered to those with this omnipresent debilitating disorder.

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