CHAPTER CONTENTS

8

Self-regulation of breathing

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INFLUENCING THE BREATHING, AND TEACHING SELF-REGULATION

This chapter focuses on interventions with a psychological component, including emotional factors, the breaking of habits, details and implications of self-regulation, psychotherapeutic techniques, and aspects of dealing with panic. As long as the mind and the intent are engaged, learning to breathe differently is a psychological process. This is especially true when disorders of the breathing pattern are based in disordered thinking and feeling. Changing such patterns is a bigger order than bringing about steadier breathing, and is not always necessary. Proceeding as if the breathing is simply excessive and trying to make it slower and less deep may be all that is needed. Because of the bidirectional relationship of body and mind, strictly physical or behavioral changes generally also have an impact on the emotional state. Theoretically, one person can come for improvement in the breathing pattern and end up feeling more psychologically stable, while another comes in for psychotherapy and ends up having more stable breathing.

A person with respiratory symptoms must choose how to interpret those symptoms, and this often determines what kind of practitioner to consult. With a combination of symptoms such as shortness of breath, chest tightness, light-headedness, and anxiety, most people would go first to a general practitioner. But if the tests come out negative, some may next consult a nutritionist,

some an acupuncturist, some a chiropractor, some a psychotherapist, and some a physical therapist or osteopath. Thus the person is already displaying a biased belief about the source of the symptoms. Each of these disciplines (and others besides) has a valid approach to breathing problems, and each discipline will probably offer some relief through its particular approach to an identical set of symptoms – especially if the beliefs of the practitioner match the beliefs of the patient. This suggests that curative factors affect a multifactorial system which can be influenced from many directions.

Trying to guide or retrain someone's breathing usually involves teaching brief interventions which simulate natural relaxed breathing. With repeated practice and self-correction, the conscious intervention may become less conscious, perhaps habitual. Several books describe methods for retraining breathing along these lines, and they reflect careful observation and practice (Bradley 1998, Clifton-Smith 1999). This chapter offers a few general guidelines, with attention to the psychology of controlling the breath.

When the topic is 'learning to breathe better,' the teaching/learning situation as usually set up presents a quandary: the patient is informed of an erroneous breathing pattern and is offered help in learning to correct it. This exchange takes place during rational verbal interaction. But the breathing problem emerges from a system that is far from the rational verbal realm. Changing one's breathing is not the same as improving one's tennis serve or ski technique; breathing is a continuous process and fully automatic in the sense that it does not require conscious supervision. Also, since breathing is so essential to life, there are multiple controls and safeguards to ensure its operation. Teaching someone to interfere in this process is presumptuous. We can commandeer the breathing mechanism temporarily with full attention, but as soon as the mind wanders elsewhere, automatic mechanisms return.

Yet progress is quite possible. The interaction between voluntary and involuntary can be addressed with respect for the deep, protective systems which are trying to ensure adequate air exchange in spite of conflicting messages from various areas of the brain. The problems which create the need for breathing retraining may derive from emotional sources or from injuries, poor posture, or habits acquired through compensation for some other factor, as detailed in preceding chapters. Assuming there is no current structural or medical impediment to restoring normal breathing, the challenge is to allow the body to breathe on its own, in line with the metabolic needs of the moment. To change a chronic breathing pattern it is necessary to make the conscious intervention less conscious, more habitual.

If a chronic emotional state is contributing to the breathing problem, how could one change breathing in a long-term way? It would seem that both would have to be changed together. Yet if we consider that the mind is affected by the breathing as well as the reverse, the separation becomes artificial. The question posed emerges from assumptions about a mind–body dichotomy. We can talk about interaction, mutual influence, reciprocity, and synchrony until the distinction starts to seem like an artifact of language and ultimately pointless. Our English language preserves the mind/body distinction, but biological reality predates language.

Many breathing pattern problems can be seen as a too-tight coupling between the emotional life and breathing. This close interaction conflicts with regulation of breathing for strictly physical purposes, in the same way that eating can be influenced by psychological factors and begins to serve emotional needs. Voluntary regulation of the breath may also be opposed by emotional factors, sometimes serving as a brake on free abdominal expansion. The works of Wilhelm Reich and Alexander Lowen address this issue thoroughly (Gilbert 1999a).

In the case of hyperventilation, it is important to either interject a pause or create a slower exhale so that the volume of air flow per unit time is reduced. Patients often worry that they will be expected to monitor and control their breathing all the time. This is not practical or even possible. The degree of voluntary control over breathing is often below normal, and the trainer/therapist should not assume much native skill in the beginning.

When asked to pause at the end of an exhale, for example, many people simply cannot. They will partially comply, but will draw a slight amount of air in, either knowingly or unknowingly. When asked to exhale slowly, there may be little reversals, 'sneak breaths' on the way out. And if asked to not sigh so much (a very common problem in chronic hyperventilation), often the reverse happens instead: more sighing, as if thinking of the possibility stimulates more of it.

PAUSING THE BREATH

Breath-holding, voluntary apnea, is a simple place to begin. The breath can be stopped with the throat open so that air can still move easily in and out, or with the throat closed. Closing the throat is accomplished by pressing the back of the tongue against the soft palate so as to seal the air passage. This blocks all air movement, and can be achieved by preparing to pronounce a 'K' sound.

Stopping the breath is within everyone's repertoire. It is of course necessary when swimming underwater, and it is also part of the 'freezing' reflex. Vocalization requires interruption of the breathing cycle. We can also exhale forcefully, as when spitting or blowing out a flame. These are all manipulations of the breath for a specific purpose. But to meddle with the mechanism in the abstract is different in principle, and often feels threatening to those who most need it.

Interrupting the movement of air may bring out latent fears. Those very anxious about access to air at all times may ostensibly comply, but will still pull a little extra air in when asked to pause. This can be detected either with a strain gauge around the upper body or by close observation, or even a mirror held beneath the nostrils to collect the condensed moisture. The person may be unaware of this action. Breath-holding time is an indicator of a tendency to hyperventilate, if seen as excessive air hunger or as a fear of build-up of CO_2 – the so-called 'suffocation alarm' (see Klein 1993 for a discussion of this). Some patients cannot exceed 10 seconds of breath-holding at first; 30 seconds is reasonable; and 45 should be within reach. Using a pulse oximeter can display to the patient reassuring evidence that the O_2 saturation does not drop very much during breath-holding.

Interjecting a pause is the wedge, or 'foot in the door' of conscious control of breathing. It is useful to practice pausing with the throat both open and closed, to feel the difference. Once that is mastered, the next step would be a pause at the end of the exhale. This is a different act than a post-inhale pause and is usually harder and less familiar. Pausing without closing the throat is preferable, though there is no barrier to 'sneak breaths' this way. The object is to allow a complete exhale to 'happen', simply by releasing all breathing muscles and letting the movement subside, then resting just a moment before the next inhale.

In learning to pause the breath, it may seem just as logical to pause after the inhale as after the exhale. If the goal is to simply stop the loss of CO_2 in an urgent situation, then any method for accomplishing that is better than nothing. But there a few reasons why a post-exhale pause is better:

1. Pausing after the inhale, holding the lungs filled, creates tension and strain in the muscles of inhalation
2. Pausing after the inhale creates temporary hyperinflation, which works against relaxation and proper emptying of the lungs
3. Pausing after the exhale is more natural. The breathing system reduces volume by slowing the frequency, reducing the depth, and lengthening the post-exhalation pause. A post-inhale pause does not seem to occur naturally except when accompanying a state of suspense.

A count by the therapist is useful at first, at one count per second or less: '1–2–3 (in) 4–5–6 – (out) 7 (pause).' Then add one or two more counts to the pause segment. Then instead of counting each number, say in the same rhythm: 'in ... out ... pause ...' Timing is not critical here; the depth will adjust to accommodate various ratios. It helps to focus attention on the sinking down of the chest, the deflating of the abdomen, the release of used air, and the quietness that ensues when all motion stops.

For individuals with anxiety about breathing, learning to do this means being desensitized.

Their uneasiness is being confronted by asking them to do what they usually avoid. Learning to tolerate a brief pause develops tolerance of CO_2 build-up, which may be important. Simply working with the breath in this way can provoke major fear if the person has had attacks of hyperventilation-linked panic. So it is important to go slowly, but also to convey that voluntary breathing control is worth learning because of the effects of relaxation. One must assume that an optimal breathing pattern is available underneath, if only the conscious mind can be induced to get out of the way.

SELF-MASTERY

When we ask a patient to breathe in a certain way we are encouraging self-mastery of a system which may have been a source of great fear and worry. Many patients are initially uneasy about following any directions to alter their breathing; such directions may initiate gasps or sighs, or else an apparent refusal to tamper with a biological process which they perceive as threatening. It would be the same in principle if a person were asked to drive a car for the first time, or better yet, to ride a horse, because a horse has a mind of its own and can move by itself. Trying to halt an episode of panicky hyperventilation may feel like trying to stop a runaway horse.

This alienation from a natural body function amounts to a withdrawal of responsibility, leaving the breathing process to be driven by emotional states. This alone constitutes a strong rationale for 'breathing exercises' – what is being exercised is voluntary control, with the intent of modifying or reversing influences from emotional centers.

Learning to interrupt out-of-control breathing is important, and so establishing some kind of entry point for conscious control is essential. This requires extending the conscious mind into a new, and sometimes frightening, realm. The breathing system may be the aspect of the body holding the most menace, by being associated with symptoms representing an uncontrollable aspect of the self. Breathing for some individuals is particularly linked to emotions; it may be a major route of emotional expression and represent something far more than simple air exchange. If so, requesting conscious regulation of this process is close to requesting conscious regulation of anger, grief, or feelings of abandonment – a much larger order. So it is not therapeutic to be frustrated with patients who balk at regulating the breath or have special difficulty with the task.

ACUTE HYPERVENTILATION

If a person is visibly hyperventilating, and it can be determined that the state is not due to organic factors but is generated and maintained by anxiety, this is not a medical emergency, only an emotional emergency. This is a condition frequently encountered in emergency medical departments, especially when chest discomfort is involved. A minor tranquilizer will terminate such episodes within a few minutes, but leaves the person with the conclusion that the solution to subsequent attacks lies in another pill.

The state of panicky hyperventilation can also be considered a state of heightened suggestibility. Brief hyperventilation is often used as the final 'push' to enter a hypnotic state, and many hypnotists routinely ask for deep breathing to facilitate entering a trance (Baykushev 1969). Cerebral hypoxia compromises orientation and perception of reality, and cognitive powers will usually be diminished. The emotional distress adds further to the need and willingness to be helped, and so anyone with a suggestion of authority or special knowledge is in a good position to do so.

Direct instruction works well here. For example: 'I can see you're very nervous and you're breathing pretty heavily. Would you like me to help you? You need to get control of your breathing, because it's a big part of the problem. Do you feel you're not getting enough air?' (*consider response*) 'You're getting plenty of air. You're breathing enough for three people! But the way you're breathing prevents your body from absorbing it. If you breathe more slowly, you'll start to absorb more oxygen. Let me help you. Watch my hand. Breathe with my hand.'

Then begin pacing the person's breathing with your hand, rising for inhale, falling for exhale, like a conductor, following the existing rhythm at first and then starting to lead it: in other words, *following, pacing, and leading.*

Following the person's breathing in the beginning imposes no requirement to change anything, but provides an external reflection of the breathing pattern. As the person's attention is fixated on the moving hand, the internally-directed drive is somewhat interrupted.

When there are signs of response to the hand slowing down slightly, change the magnitude of the movements, suggesting less volume as well as a slower rate. Questioning 'Do you feel a little better yet?' sets up the expectation that improvement is bound to happen. If the person is breathing through the mouth, suggest closing the mouth. If that is resisted, then suggest and demonstrate pursed-lips breathing to slow the exhale. Normally this procedure will bring about the desired results within 5 minutes, with perhaps an aftermath of the patient being shaken, with chest discomfort, but breathing and thinking normally again.

Fear of fear

A common idea when treating anxiety disorders is 'fear of fear.' With a simple phobia the object of fear is specific: dogs, deep water, heights, sharp objects. The alarm state rises up when close to the feared object or situation, and subsides upon withdrawal. It is rarely clear how much of the fear of the object itself (and the projected consequences) and how much is fear of the high-arousal state of fear itself.

In panic disorder, and usually in hyperventilation associated with anxiety, the fear becomes centered on the fear reaction itself. There may be avoidance of objects or situations which set off panic, but the essence of the panic is a fear of collapse, death, unconsciousness, insanity, or some similar catastrophe involving loss of control. In other words, it is fear of the fear state. Explaining this to a patient is sometimes helpful, and can initiate insight that can interrupt the cycle. But it usually takes more than explanation.

For this reason, tampering with a person's breathing, when breathing has been the object of fear, feels like unlocking the cage of a lion which once attacked. By the process of generalization (see Ch. 5), anything associated with the traumatic experience becomes a potential warning sign, and initiates avoidance.

'False equilibrium' and adaptation to imbalance

If the breathing pattern of hyperventilation is fairly fixed in the patient, it will feel familiar or even normal, regardless of the symptoms generated. This is partly due to the body's adaptation to the lower CO_2 levels, which includes reduction of bicarbonate buffer. The result is that achieving 'normal' breathing briefly will be opposed by the adaptation, which expects abnormal breathing to continue. Reduced breathing will raise CO_2 and may initiate a feeling of not getting enough air. If so, the patient may have become used to hyperinflation, so that reduced volume and a full, relaxed exhale do not feel right. If mouth breathing has become a habit, the extra resistance of nose breathing will feel odd at first.

This adaptation to an imbalanced situation can be explained to the patient, depending on the level of comprehension, with the prediction that the patient's physiology will adjust in time to an improved, lower level of breathing. Presenting printed norms showing where CO_2 and oxygen saturation should be, can be convincing enough to motivate acceptance and home practice.

ASSESSMENT

There are several ways to evaluate breathing pattern disorders, particularly hyperventilation, and none reigns supreme. Arterial blood gas analysis is the medical approach for evaluating hyperventilation, but can only detect the state at the moment of the test. Behavioral observations and symptom checklists may seem less precise, but measure things over a longer time period. Chapter 2 carries details on assessment from the physiotherapist's perspective.

The following tools have in common an attempt to bring some objectivity to subjective impressions. They are described along with suggestions primarily for their use in clinical situations.

Anxiety Sensitivity Index

The Anxiety Sensitivity Index (ASI) measures the amount of concern about various symptoms of anxiety. The actual items of the ASI (see Ch. 5, p. 120) are a fair representation of the special fear state typical of most individuals with panic disorder. A large subset of this group has hyperventilation as a factor, whether as a source of the panic or a complicating consequence of the panic (Hegel & Ferguson 1997).

The ASI responses fall into four major categories, according to a factor analysis (Cox et al 1995):

1. Fear of cardiorespiratory distress and gastrointestinal symptoms
2. Fear of cognitive/psychological symptoms
3. Fear of symptoms visible to others (social fear)
4. Fear of fainting and trembling.

These are quite diverse factors, but all can lead to full-blown panic if the patient has no way to counteract excesses in these fears. Prior sensitivities to any of these issues would probably be magnified during an episode of hyperventilation, when the natural consequences of hypocapnia shift the body and mind toward instability or malfunction regarding:

- Disturbed cardiac and gastro-intestinal activity
- Dyspnea or unusual air hunger
- Racing, erratic, anxiety-tinged thinking, including possible dissociation and feelings of unreality
- Faintness, light-headedness, muscle twitches and tremors, weakness.

The fear of being afflicted in public, losing control and feeling helpless or humiliated, adds an extra layer of social fear that easily adds to the sense of danger and urgency, which is usually fed back into the psychophysiological storm going on, to further destabilize the system. It is the therapist's job to block this feedback and try to halt the generation of symptoms to begin with.

Variations from person to person in how these factors are manifested may be determined by each person's physiology: for instance, if a particular person's neurological system is more susceptible to P_{CO_2} drop, causing light-headedness, then that may become the person's major panic symptom. Personal history may be a deciding factor; for instance, a prior heart attack, an incident of severe pneumonia, or history of vomiting would make the person more sensitive to those symptoms as warning signs that a dreaded experience is recurring. Selective apprehension about particular symptoms is a third factor: if a relative died of a heart attack or brain tumor, for example, then any disturbance in those systems will be more salient than usual.

Once this test is given and scored, however, one has only a number. Clinical effectiveness requires more than that. Use of the ASI encourages differentiation of a monolithic 'attack' into its components, which then can lead to inquiry about personal history, the experiences of close friends and relatives, and beliefs about the meaning of the symptoms. With this information in hand it is easier to begin correcting what seems to be a latent phobia, or an unopposed phobic potential – a susceptibility which becomes activated by evidence of the symptom appearing.

When presented with this sort of analysis, some patients will begin to understand that their reaction to, and interpretation of, the symptoms is an integral part of the episode. There has been some debate as to whether the hyperventilation-based panic attack is primary, giving rise to anxiety, or whether the anxiety is primary, set off by perhaps unconscious associations or emotional factors such as suppressed anger. After the onset of an attack, this debate becomes academic, because the two factors of interpretation and physiological disturbance become so intertwined that intervention becomes the main priority.

So when assessing a patient who has signs of acute or chronic hyperventilation, adding the ASI to the evaluation takes only a few minutes and provides additional data *about the person who has the problem*, as opposed to data about the problem itself (objective data such as respiration rate, CO_2 readings, reported symptoms, etc.). Knowing that

the person scores especially high, compared to the ASI norms, suggests a subjective factor at work which amplifies the apparent symptom picture. Discovering this, one might switch the therapeutic focus from reducing symptoms to examining what the person assumes about the meaning of the symptoms.

Nijmegen questionnaire

Most of hyperventilation's 'signs and symptoms' as listed in the Nijmegen questionnaire and similar scales are not specific for hyperventilation, and often occur during periods of anxiety regardless of whether CO_2 is depleted (Han et al 1998, Wientjes & Grossman 1994). Items of the scale are to be found in Chapter 7 along with discussion of its use. Some researchers have concluded that reporting of symptoms is influenced by psychological factors as well as by actual CO_2 levels. So there is much room for uncertainty in distinguishing between hyperventilation and an 'anxiety state.' The fact that a patient reports dyspnea, cardiac symptoms, or even peripheral tingling cannot be considered objective in the same way that an instrument reading is; the tendency to notice something and to report it is itself a powerful variable, and appears to be as much psychological as physiological.

The Nijmegen questionnaire has shown itself to correlate well with other objective signs of hyperventilation: 80% of high scores reported that their symptoms matched the sensations felt after a provocation test (van Dixhoorn and Duivenvoorden 1985). It is useful as a screening test and is certainly safer and simpler than the provocation test. Repeated administration of its 16 items can show improvement during a course of treatment, and provides a good foundation for discussion of symptoms.

Recently, Moscariello et al (1999) added to the validation of this questionnaire by provoking hyperventilation through maximal exercise on a cycle ergometer. Subjects were patients referred to a pulmonary center with 'suspected hyperventilation syndrome.' Failure of the $PaCO_2$ to return to baseline within 10 minutes after exercise stopped was considered slow recovery. A certain cut-off point was used on the Nijmegen questionnaire to designate the 'hyperventilating' group. In 88% of the subjects there was agreement in classification between the two tests, supporting the notion that either test can predict results of the other, and that they measure the same characteristics.

The variance in self-reporting because of psychological characteristics is not limited to respiratory complaints. It also applies to irritable bowel syndrome, for example; research has determined that the tendency to experience and report bowel pain, bloating, and abdominal cramping function correlates with psychological indicators of anxiety and depression as much as with objective laboratory findings. These variables also tend to correlate with a tendency to consult medical practitioners. An extensive sampling of individuals who had never complained to a doctor of irritable bowel symptoms revealed that such symptoms were relatively common, but did not bother them or at least did not motivate doctor visits. The psychological coping in these people was apparently more effective (Whitehead et al 1988).

When relating such conclusions to respiratory symptoms, it is important to consider the psychological and cultural context within which an individual is functioning. There may be something helpful which a practitioner can do with words and emotional communication that cannot be done by manipulation or breathing training alone. Even the structural and postural changes which give rise to some breathing problems may in some instances be physical expressions of chronic feeling states. If so, correcting them at the 'distal' point of expression may be of limited value and symptom-focused, analogous to an allopathic medicine approach.

Hyperventilation provocation test

This test is often used to rapidly determine whether a person's symptom complaints can be reproduced by hyperventilating, on demand. The regimen is usually rather extreme, such as one deep breath per second for 3 minutes. This is sufficient to create paresthesias and light-headedness

in nearly anyone. One minute of vigorous breathing may be sufficient (see Timmons 1994, p. 283, on this issue). The rationale is that a similar hypocapnic state develops more gradually from a less extreme form of hyperventilation, and reproducing it quickly by vigorous hyperventilation is a matter of time economy. The physician or therapist often has a paper bag ready to help, in theory, to terminate the state by rebreathing air with a higher concentration of CO_2 (but see Ch. 9 for a discussion of this).

⚠ **CAUTION:** Provocation of symptoms of hyperventilation carries some risk (see Ch. 2), and if there is any reason to suspect a compromised cardiovascular system, an increased risk of stroke, or possibility of seizure, asking someone to hyperventilate may not be wise because of the stress on the brain, heart, and blood vessels. Vigorous hyperventilation does stress the cardiovascular system, can initiate anginal pain in those who are susceptible, and could reduce cardiac blood supply enough to stop the heart. The mechanism of action is partly smooth-muscle vasoconstriction; coronary blood vessels are not immune to this effect, which operates also on the bronchi, esophagus and gastrointestinal tract, and cerebral blood vessels.

The nervous system is also affected by low CO_2 and the resulting alkalosis, and nerve conduction within the heart can be disrupted (Nixon 1989, 1993a, b). A large Japanese study compared patients with angina to control subjects. After hyperventilating for 6 minutes, 62% of the angina subjects developed clear signs of ischemic electrocardiographic changes indicating coronary spasm, and none of the control group did. This study implied that hyperventilation is a specific and reliable test for diagnosing coronary artery spasm (Nakao et al 1997) and is quite potent for disrupting the heart function in susceptible individuals.

In the practice of neurology, using hyperventilation to provoke latent defects in a physiological system has long been employed to detect the abnormal spikes and discharges which indicate seizure susceptibility. The cerebral circulation is stressed by hyperventilation during EEG monitoring; the rapid development of hypoxia increases the chance of seizure activity. So if the person has a history of seizure activity, it should be clear why provoking hyperventilation for testing purposes is not recommended.

Most people hyperventilate now and then in the course of normal activities. This probably does not equal what is requested during a hyperventilation 'challenge' during which a capnometer may be used to confirm that sufficient hypocapnia occurs – usually 20 mmHg or below. Complying with such a procedure removes any natural restraints the person may have, which may be protecting against seizure or anginal pain.

Therefore, before considering the hyperventilation provocation test it is prudent, as a minimal precaution, to check for susceptibility to seizures, lung disease, which compromises breathing, and heart trouble or chest pains which could be angina. 'If in doubt, leave it out' – or substitute the hyperventilation provocation *suggestion* test. To do this, simply describe a proposed test in which the patient would be asked to breathe fast and deep for a short time to see what happens. If the response is apprehension, refusal, or obvious discomfort (often accompanied by hyperventilation!) this suggests that the patient probably knows already that symptoms might be provoked, in which case demonstration of this fact would be superfluous, as well as insensitive.

There are positive and negative aspects to provoking hyperventilation as a diagnostic aid. When familiar symptoms are made to appear and then recede by a simple breathing manipulation, some patients will be surprised and intrigued, and this will begin a process of considering the symptoms as potentially controllable, rather than implacable and mysterious, appearing without warning or reason. But since natural breathing increases are rarely as drastic as during a provocation test, the transfer of insight to real life can be difficult. The maneuver of 'proving' to the patient something about the source of the symptoms may take away some of the fear, but not necessarily, because reducing the magnitude of breathing during an episode of hyperventilation still takes training and practice.

In spite of the risk, the provocation test is routinely performed by researchers in the field, some-

times with emergency resuscitation equipment available and sometimes not. The advantage of having a patient deliberately hyperventilate is that it can be reversed by controlled breathing, and this can prove to be a powerful demonstration.

There is a greater complication, however, inherent in the HVPT as usually practiced: it implies that the symptoms are entirely due to CO_2 depletion. Several factors work against this conclusion, related to the power of context and the difference between the testing situation and real life: the occurrence of strong emotion in natural situations, the lack of safety and support, and the lack of clear triggers for the sensations. Provocation of hyperventilation in the office or hospital setting does not reproduce the usual context in which the symptoms occur. Therefore, symptoms provoked in this way may not match what is experienced naturally, leading to false conclusions.

Modified provocation

A two-step educational maneuver can be very effective in recruiting the patient's interest and cooperation, especially if the hyperventilation involves panic attacks. (This is not suitable for cases where even mild hyperventilation could be dangerous.)

The first step would be explaining in detail the physiological route by which excessive breathing can create such symptoms. The second step would be requesting a very brief bout of hyperventilation to demonstrate that the symptoms can be created and eliminated by restoring normal breathing.

This 'provocation of the symptoms' can be quite gentle, consisting of perhaps five deep open-mouthed breaths in 15 seconds, emphasizing complete exhalations to maximize CO_2 clearance. Reassurance should be provided beforehand that any sensations which appear can be quickly reversed by terminating the hyperventilation. A capnometer if available will furnish some objective data, but the patient's sensations are valuable data also.

From this brief provocation, the beginning or 'edge' of the panic symptoms commonly appears quickly in many patients: slight light-headedness or faintness, peripheral or lip tingling, chest tightness, or general uneasiness. Since these changes were predicted and their reversal promised, panic is less likely to occur.

The reversal consists of returning to slow, normal breathing, mouth closed, pausing briefly at the end of each exhale if possible. Usually within 2–3 minutes the patient feels normal again. This symptom reversal, together with explanation and discussion, offers clear evidence to the patient that the symptoms (and the fear) are potentially controllable.

Delayed recovery

One marker of a tendency to hyperventilate is delayed recovery of $PaCO_2$ following a hyperventilation provocation test. This was originally studied in detail by Hardonk & Beumer (1979). These researchers standardized the hyperventilation phase to one deep breath per second for 3 minutes, after which the subjects were asked to stop and resume normal breathing. The researchers used this procedure to compare a control group with patients already diagnosed by other means as having hyperventilation syndrome. There was a large variation in the time elapsed before baseline level was achieved, sometimes as long as 15 minutes. After studying differences between the two groups, they determined that the best-discriminating criterion was a ratio of 1.5 or larger between percent CO_2 at rest (before the test) and percent CO_2 at 3 minutes. For example, if the baseline reading was 40 torr and the post-hyperventilation level 3 minutes into the recovery was 25, this would yield a ratio of 1.6 and therefore would qualify as hyperventilation. A reading of 32 would yield a ratio of 1.25 and would fall below the diagnostic criterion (Fig. 8.1).

The reason for this phenomenon is unknown; Folgering (1988) describes it as 'irradiation' of cortical activity to the brainstem in resemblance to a 'flywheel' effect. In other words, an after-discharge persists even though the individual intended to resume normal breathing. Another factor related to this is the finding that hyperventilators tend to judge their breathing normal again, following a provocation test, even though

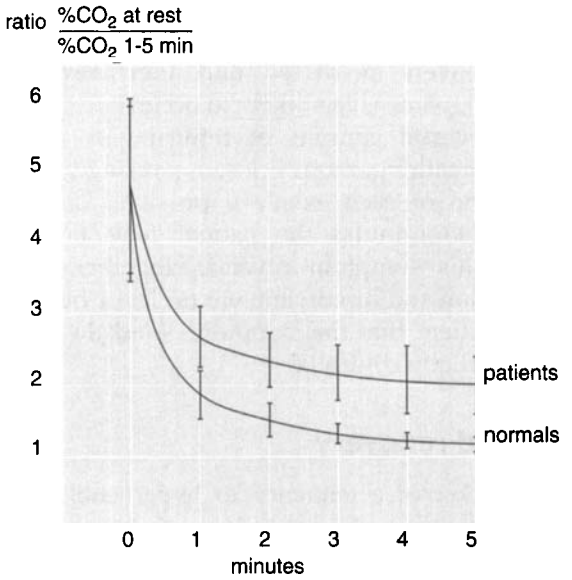

Figure 8.1 The relationship between the ratio $\%CO_2$ at rest and $\%CO_2$ after 1 through 5 minutes recovery from 3 minutes of hyperventilation. Vertical lines are standard deviations. Normals returned more quickly to their resting values than patients (chronic hyperventilators). A ratio of 1 signifies complete recovery. (From Hardouk & Beumer 1979.)

it has still not recovered (King et al 1990). This may mean that their set point for 'normal' was altered because of habituation to a chronic hypocapnic state.

This 'delayed recovery' test distinguishes individuals who complain of chronic symptoms typical of hyperventilation, and so if they actually are hyperventilating, they probably do so a lot of the time. However, this test might not pick out the individual who hyperventilates occasionally in response to a particular emotional state (this is the category defined by Conway et al (1988) and also referred to by Gardner (1996).) Individuals who occasionally lapse into this state are less likely to have the prolonged recovery time from hyperventilation. This may mean that they are less dominated by a feeling state which overstimulates breathing.

'Think test'

Peter Nixon, a London cardiologist, pointed out the inadequacies of the simple HVPT, observing that episodes of hyperventilation are often triggered by moments of strong emotion. His technique requires more active involvement by the practitioner, but is more likely to recreate the conditions under which the symptoms have appeared.

His procedure (detailed in Nixon & Freeman 1988) starts with a HVPT duplicating the standard protocol of 60 breaths per minute for 3 minutes, with the $PaCO_2$ required to fall below 19 mmHg. (His 57 subjects all had cardiovascular symptoms of various sorts.)

During the 3-minute recovery period, PCO_2 was monitored for return to baseline according to the Hardonk & Beumer criterion. Then subjects were asked to 'close their eyes and recreate in their minds a time and place where they had experienced their typical symptoms: they were invited to think back and remember all the feelings and sensations which were present at the time' (p. 277). Also, topics that had seemed emotionally significant during the initial history taking were brought up again, and the patients were asked to recall those feelings and sensations. The criterion for this phase was a fall of more than 10 mmHg maintained for 1 minute or more.

Using this more personal experience-based testing, the researchers found many positive responses in subjects who would not have been labeled 'hyperventilators' according to the delayed recovery test. 60% actually met the second criterion, and only seven would have been identified by the previous provocation test. (One subject, incidentally, had a grand mal seizure during this phase; he was not a known epileptic.)

This result fits with the observations cited in Chapter 5, of Conway and colleagues, including that hyperventilation often occurs occasionally in certain people in response to strong emotion, but that they do not hyperventilate routinely. This group also is less likely to display the delayed recovery from hyperventilation; that trait is more likely in those who could be termed chronic hyperventilators, and perhaps are doing it out of habit or else some fairly steady emotional state.

It should not be necessary to put patients through the first provocation procedure in order

to test them with the second. Nixon & Freeman speculate that the prior hyperventilation sensitized the subjects, reducing cerebral blood flow in comparison with flow to lower brain centers such as the amygdala, which are more involved in emotionality. However, clinical work is not research; the goals are different. Discussing situations and events which were associated with an 'attack' is likely to disrupt the breathing pattern somewhat, and with either capnometry monitoring or close observation, this disruption can be focused upon as an opportunity for self-knowledge and self-regulation.

BREATHING TRAINING

Biofeedback

The following descriptions use end-tidal CO_2 feedback, using a capnometer, to illustrate some strategies for teaching breathing control. The principles, though, are general, and some can be adapted to other kinds of biofeedback.

Monitoring and display of end-tidal CO_2 can be valuable for reversing the behavior of hyperventilating, whether it is an occasional event or a chronic condition. The basic instrument samples exhaled air by either a mask, nasal cannula, or a thin tube at the nostril. Most commonly, the instrument's display panel shows the breath-by-breath measurement as digits, either mmHg (torr) or as percentage of the exhaled air. There is often a CO_2 waveform display also. End-tidal CO_2 correlates well with arterial CO_2 (in the absence of lung disease or heavy exercise) and so serves to indicate the degree of blood saturation with CO_2.

The monitoring set-up can be used for clinician information only, for spot displays to the patient, or for continuous biofeedback. Interpreting the data correctly requires a bit of explaining, but once it is clear in the patient's mind what is desirable in the CO_2 trace, a good learning situation is established.

There are several things that can be done clinically with a capnometer. It can:

- Check on adequacy of breathing (presence of hyperventilation) at any moment
- Observe the slope of recovery after a provocation test
- Demonstrate the effects of emotional recall
- Show the effect of various manipulations: slow breathing, rapid breathing, breath-holding, sighing, and brief exercise
- Provide feedback on attempts to relax and slow the breathing
- Assess the variability ('steadiness') of the breathing pattern during various conditions.

1. Checking for presence of hyperventilation

Provided the capnometer is switched on and warmed up, a single reading of end-tidal CO_2 ($PetCO_2$) can be obtained in a matter of seconds, barely more than the time it takes to exhale into the sampling line. This reading does not necessarily imply anything beyond that moment. The normal range is 35–45 mmHg; by convention, 30 mmHg is a rough dividing point for signifying hyperventilation below that point. $PaCO_2$ fluctuates continuously and is more labile in some people than others. A single deep breath can push the next breath's CO_2 content temporarily below 30 torr, while suspending breathing for 30 seconds may result in the next breath reading close to 50.

Blood gas measurements in the medical setting are usually done via arterial puncture. This is a less trivial procedure than venipuncture, so since repeat measurements are not routine, the single value of $PaCO_2$ is taken to represent a steady state. Like end-tidal measurements, this represents one moment only, but more information is available with a blood gas measurement. The values of oxygen saturation plus the partial pressure of oxygen and bicarbonate help to clarify the meaning of the $PaCO_2$ reading.

With capnometry, a series of end-tidal readings is easily obtained and begins to outline the pattern of breathing. Since both the depth of inhalation and the rate of breathing combine to determine total air flow, it is difficult to decide by observation alone whether an individual is actually hyperventilating. The florid cases are obvious: heaving chest and rapid breathing. But with a

more subtle deviation from normal, sampling the exhalations for a minute or two adds objective information which would permit the conclusion that the person is truly hyperventilating. It may be a habitual style, it may be because of what was just discussed, or it may be expressing anxiety about the measurement process. But the information is a beginning. If certain symptoms are being reported (light-headedness, chest discomfort, confusion, etc.) and the $PetCO_2$ is low as well, then making this explanation can be very helpful.

2. Rate of recovery of CO_2 after hyperventilation provocation

The procedure of provoking hyperventilation in order to observe speed and completeness of recovery requires a capnometer, since the test was developed around end-tidal CO_2 measurements. Following Hardonk & Beumer's standard procedure of 3 minutes of deep breathing, one per second, still allows variability in respiratory variables, but the timing of the breaths and requiring a certain degree of drop in measured CO_2 at least provide some standardization. Some researchers use 20 torr as a criterion for adequate hyperventilation. In clinical use one looks for 'indications' and 'apparent tendencies' rather than arbitrary pass–fail cut-off points, and determining hyperventilation on the basis of one test is unwise in any case.

With those reservations, using this procedure to observe the rate of recovery can be very useful both for the clinician and for the patient. The addition of O_2 saturation monitoring via pulse oximeter adds even more information. The rate of recovery after the patient is asked to 'resume normal breathing' is assumed to depend primarily on minute volume rather than some constitutional physiological variable.

Patients may be unaware of brief periods of apnea, most often at the end of an exhale. Such interruptions of breathing rhythm are normal to some extent, since breathing is driven by CO_2 level, and if CO_2 stays low the pattern may alternate between continued hyperventilation and loss of respiratory drive. This area has not been much studied, but O_2 saturation can drop because of cessation of breathing. This is what happens when some swimmers prepare for an underwater dive by intentionally hyperventilating; by depleting their blood CO_2 they are removing the marker that serves as a signal for 'air hunger.' Thus the swimmer can remain underwater a little longer before the urge to breathe becomes overpowering. It feels as if there is more oxygen available, but in truth the warning mechanism has been disabled, making blackout a more imminent danger.

Demonstrating to patients how long it takes to return to normal, baseline CO_2 levels with a capnometer can underscore the point that their breathing has persistent physiological consequences. If oximetry monitoring shows a drop in O_2 saturation in the process of recovering normal breathing, that is useful information also. Breath-holding is relatively easier after hyperventilation (as in the underwater swimming example), but while it may return CO_2 levels to normal more quickly, it is also depriving the body of continuous oxygen supply. The instability and oscillation between the two extremes may be characteristic of those who chronically hyperventilate.

In any case, patients should be led to understand that an experience of strong negative emotion can be prolonged partly through breathing not returning to normal; this makes them more susceptible to recurrence of the disturbing feeling. Thus an argument, for example, might go on longer than necessary because the state of outrage or insult is reverberating with excessive breathing. A state of fright might persist also, defined partly by mental phenomena and partly by continued low CO_2.

The above illustrates the principle of psychophysiological reverberation between the two aspects of a person, a curious interaction between mind and body. Such resonance provides an opportunity to intervene from two different directions: change the thinking–feeling state and the breathing will change. Change the breathing and the thinking–feeling state will change.

3. Demonstrating the effects of emotional recall

The work of Conway (see Ch. 5) as well as Dudley (1969) and others has demonstrated the

extreme 'thinness' of the interface between emotions and breathing in certain people. If the respiratory system is acting out each emotional state that runs through the mind, and is forever preparing for some new imaginary threat, then physiological stability will be significantly compromised. Using the capnometer (or any other kind of breathing monitor) helps to magnify and sharpen patients' awareness of their breathing responses to various emotional states (Fig. 8.2). This information is of course useful to the therapist also, not only to confirm that the breathing reacts strongly to induced emotion, but also to show more precisely which topics and emotions affect the breathing. But until this information is understood by the patient it is not of much practical use. The capnometer can be used like any other psychophysiological monitoring instrument, in the manner of a polygraph examination, except that the goal is to detect strong emotional responses without the motive of detecting deception (see Case study 8.1).

Most often connections such as the ones in the case study are not so clear or retrievable, but it is usually enough to ask the patient to describe a recent instance of hyperventilating while being monitored with the capnometer. Normally this request elicits a detailed image from which the patient retrieves details, but in the process the whole response begins to start up again. The capnometer will show a drop in CO_2, and often the breathing change is obvious: mouth and chest breathing at an irregular rate. The point is made, then, that thinking about the symptoms can actually recreate the symptoms. Distraction from this experience of recall (or simply giving instructions to breathe normally) will restore normal breathing and thus a rise in CO_2. Seeing such evidence of mental manipulation usually impresses the patient with how self-generated the symptoms actually are. With an adequate sense of self-power, or efficacy, this fact can be translated into more active control.

4. Show the effect of various breathing manipulations

Most of the inappropriate breathing changes which create problems happen naturally without conscious intent, unless one ascribes intent to less ostensibly conscious parts of the person. An individual does not usually decide to suspend breathing in order to think more clearly in an emergency; breathing just stops. In this sense breathing is fully automatic in the way that stomach contractions are. Yet the abundant nerve connections from the cortex to the breathing centers permit us to direct our breathing very thoroughly. The most basic neural control centers in the pons and medulla receive inputs from chemoreceptors for fine-tuning of breathing variables, but they also receive input from the limbic

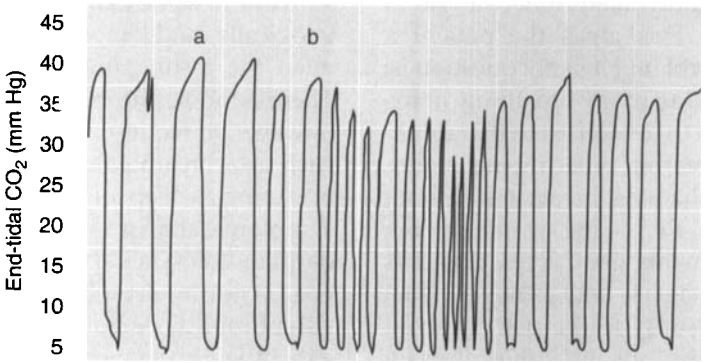

Figure 8.2 Capnogram showing drop in 36-year-old male patient's end tidal CO_2 while relaxing (a) and (b) starting to think (with frustration) about a missed job promotion. Trace duration was 90 seconds. Each wave represents a breath: peaks are end-points of exhalations.

Case study 8.1

A 32-year-old woman with panic attacks and hyperventilation was being monitored by a capnometer while discussing the origin of a recent panic attack. She was asked to recount, with eyes closed, the various aspects of the context where the attack occurred, remembering and imagining 'being there.' She had intended to visit a friend's house, but on the way began to feel chest tightness, dyspnea, and light-headedness. She began to worry about being able to control the car and as she continued driving toward the friend's house, this worry escalated into panic over the possibility of crashing. She stopped the car, tried to calm her breathing and rapid heartbeat, and finally felt able to drive back home.

Recalling and describing this whole episode dropped her CO_2 about 20%, especially as she described pulling the car over and becoming convinced that she could not drive safely. She could not state what set off the incident, but assumed it came 'out of the blue' or else was connected somehow with driving. She denied any uneasiness about the woman she was visiting, or the neighborhood, or the car itself. She speculated that her panicky breathing might be connected with the chest tightness the last time she had panicked.

To pursue the question of what triggered her attack, I asked her to visualize each stage of the trip and briefly mention the associations she had to these images. The capnometer was recording, and intervals of silence permitted adequate samples of her breathing.

As she spoke more about driving toward the woman's house, her breathing became faster and the CO_2 level dropped from 39 to 28–30 in about a minute. As I mentioned this to her, she confirmed that she was now feeling some chest tightness and rapid heartbeat again. Suddenly she remembered that her friend sometimes took care of a relative's dog, chaining it in the back yard. This had major psychological meaning for her because she had once walked into someone else's back yard and was attacked by a dog. This connection became clear to her for the first time; it was as if her 'warning system' was operating beneath her awareness. The mere possibility of the friend keeping a dog that day had set off anticipatory hyperventilation, and her attention went to the resulting symptoms instead of to the real cause.

Understanding this was a revelation to her and led to greater understanding of the concept of 'triggers' activating her alarms. Without this understanding, teaching her to control her breathing in order to approach her friend's house might not have been successful, because it would be opposed by the 'hidden alarm' being activated.

structures (emotional input) from the reticular formation when the person is awake (general arousal, alertness), and from the cerebral cortex via corticospinal neurons for full voluntary control. It is this last pathway that can be practiced, or 'exercised' in order to strengthen its dominance over breathing patterns.

If the end-tidal CO_2 is initially low (<35 torr), slow breathing may be requested directly, and the capnometer will most likely reflect a rise in CO_2 toward normal. This gives the patient a sense of potential control, but a likely question is: 'Will I have to be thinking of my breathing all the time?' The next step is to demonstrate that thinking of other things, creating other mental states, is sufficient to normalize the breathing without focusing on it directly. Removing or minimizing the feedback helps in this goal. Eyes closed is better, to promote imagery and remove visual distraction. Speaking softly to the patient about the seashore, mountains, or some other pleasant and relaxing experience will usually induce a calm state which reduces breathing volume temporarily, with a corresponding rise in CO_2. The implication of this for the patient should be clear. Assigning home practice, perhaps with a prepared audio tape in the therapist's voice, or listening to peaceful music, can provide structure for the necessary rehabilitation practice.

Rapid breathing

The full hyperventilation provocation test is stressful to some people more than others, physiologically and emotionally. Of course, that is what the testing process is trying to discover. There is no strong reason in the clinical setting, however, to request a drastic change in breathing such as a breath every second for 3 minutes, or breathing as deep and fast as possible until major symptoms appear. The test is difficult to standardize unless a capnometer is available. If the goal is not to test a body of subjects under consistent conditions but to work with one person in a therapeutic manner, then the individual patient 'becomes the experiment.' A more moderate and exploratory procedure is more appropriate in such cases.

A request to breathe slightly faster and/or deeper is usually adequate, and more acceptable to the patient. While monitoring by capnometer, the breathing is mildly accelerated to see what will happen, both psychologically and objectively. The capnometer should confirm a lowering of $PaCO_2$ and the patient may or may not feel the beginning of symptoms. At that point it is fine to back off by slowing the breathing, and observe whether the $PaCO_2$ rises and whether there is a diminution of any symptoms.

Apart from demonstrating a possible relationship between breathing volume and the twin variables of sensations and $PaCO_2$, this gentle acceleration provides practice in subtle sensing and adjusting of the breathing. It is easy to breathe fast and deep, and it is easy to hold the breath, but these are ends of a continuum rather than a dichotomy. Infinite points of adjustment are more useful for matching metabolic need, but the patient may have to practice to discover this. The 'discovery' may occur at unconscious levels, in the same manner as motor skills learning, and since the goal is to return the breathing system to automatic and minimize conscious or emotional interference, the normalizing of breathing may really be returning breathing regulation to more automatic functions.

Breath-holding

Many researchers have observed the reduced tolerance for breath-holding in hyperventilators and commented on it as indicating a higher sensitivity to CO_2 build-up. This may be because of a lower level of bicarbonate buffer, which might normally oppose the higher acidity though not the actual CO_2 rise. It is easy enough to test this capacity by requesting a breath-hold without hyperventilating beforehand. One would think that if hyperventilating before underwater swimming, for example, increases the breath-holding time by depleting CO_2 (see Ch. 2) then a chronic hyperventilator would have a head start here, but it seems to work in the opposite way.

Breath-holding times of 10–15 seconds are common when individuals fitting the hyperventilation profile are tested, which is below the normal, usually estimated as 30 seconds. This cannot be too meaningful a test, however, without controlling the style of the delivering the instructions. Presumably the build-up of the urge to breathe will be opposed by the will to please the experimenter, to appear normal, to achieve a high score in something, or some other motivation which may increase the tolerance of an uneasy sensation.

Apart from using breath-holding time as a possible diagnostic aid, there are two other uses:

1. When using a capnometer, breath-holding provides a good demonstration of how PCO_2 and breathing interact. Holding after a moderate inhale for as long as possible, the patient will see CO_2 trace reading zero, and when the breath is finally released, the trace will rise higher than it was before. A person who had a hard time rising above 35 torr might see 40 or 45 for a few breaths, as the accumulated CO_2 is released. This can be pointed out as a fast way to alleviate symptoms of hyperventilation and possibly terminate a panic attack, although not the best way; interrupting the natural breathing cycle induces a rebound air hunger for the next few breaths. It can be offered as a quick alternative to the 'paper bag' remedy. The point can be made that if breath-holding raises $PaCO_2$ quickly by restricting air exchange, the slow, limited breathing will raise $PaCO_2$ also, though more slowly.
2. Using breath-holding as training to tolerate air hunger is valuable and easily practiced. This amounts to suggesting voluntary apnea for brief periods. Resisting the urge to breathe, postponing the next breath, may be in the same category as resisting an urge to smoke, drink, or eat. The idea of going on an 'air diet' (Figure 8.3) clarifies the goal. It should be explained that the rising urge to breathe is caused by CO_2 build-up and not a drop in oxygen; the latter does not contribute to breathing drive until the oxygen content falls much lower. This is why hyperventilating before swimming underwater prolongs tolerance for submersion. At first, most people will hold the breath after inhaling, with throat stoppage (back of tongue against soft palate) but one should work toward a pause after an exhale, with no throat stoppage. This simulates the

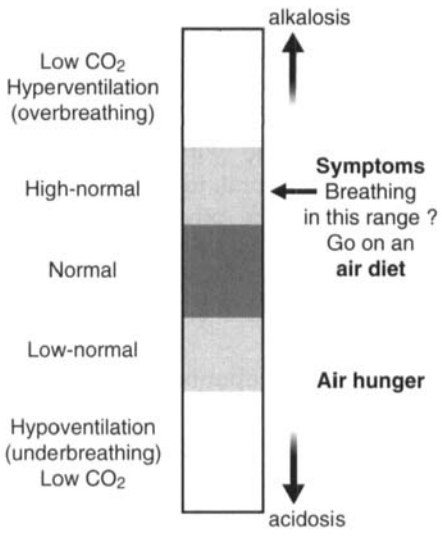

Figure 8.3 Chronic hyperventilation in principle resembles overeating: perhaps to avoid feeling out of air, some people overbreathe, depleting their systems of carbon dioxide and provoking symptoms associated with alkalosis. (Reproduced with kind permission from Gilbert 1998.)

natural breathing pattern of one count in, one count out, and one count rest. (See Buteyko notes in Ch. 9.)

Exercise

To demonstrate that more CO_2 is generated when muscles are working, suggesting light exercise on-the-spot is useful. Not every patient can be talked into deep knee bends with a capnometer sampling tube taped to their upper lip, but when they try it, it is memorable. A few squats will push the CO_2 up 10–15%. The rise is not immediate, but within 30 seconds should be apparent. This is not an exercise or endurance test, but is meant to educate the patient as to alternative ways to raise CO_2 during a hyperventilation episode. Most people during panic feel a strong urge to move around, possibly to escape where they are, and this urge for activity is strong. So cooperating with this urge generates the extra CO_2 that the body 'expects' and the discrepancy between breathing volume and breathing demand should lessen. It also provides the impression of a remedy, which is psychologically important, placebo-style.

Many other clinical techniques are available in Fried (1993). From his long experience with capnometry and with hyperventilation, he describes the use of music, imagery, biofeedback, EEG monitoring, and other approaches combined with a capnometer to normalize breathing pattern problems.

Other biofeedback

Most practitioners will not have access to a capnometer, but may wish to use some method of displaying breathing information to the patient to aid in learning to control breathing. What follows is a summary of useful ways to do this:

Strain gauges

Sensors consist of expandable tubes, belts, or bellows placed either around the abdomen (1-channel) or around the abdomen and chest (2-channel). The feedback display may consist of a chart recorder, a biofeedback screen, or other display. With two channels, the relative movements of each breathing region may be displayed for comparison. The patient can see instantly the effect of chest or abdominal expansion. The feedback is vivid and comprehensible. It is helpful for practicing abdominal breathing, for detecting paradoxical breathing, for enhancing awareness of breathing, and for helping transfer this awareness to inner sensing.

The drawbacks: attaching the sensors can be intrusive, since it requires fastening the expansion gauges around the thorax and abdomen (over the clothing). The gauges can slip down and change the measurements. Also, the gauges are hard to calibrate to objective volume quantities, and they are best used as a relative measure of volume and direction of movement (in–out) for comparison within the subject.

Surface electromyography (EMG)

Adhesive electrodes can be placed over many muscles involved in breathing. The display may be by a meter or LED light bar gauge, or on a stand-alone battery-operated instrument, via

chart recorder, or via computerized processing and analysis. This last is a part of most modern biofeedback setups, and provides a display and recording of the raw signal, the processed, rectified signal (single line), a bar graph display for different locations simultaneously, various graphic transformations such as circles, random color arrays, and games in which the body functions as the game controller. Thresholds may be set for goals of relaxing. Sound can be provided for biofeedback, and toggled on or off depending on threshold settings. The most common general strategy is to inhibit use of accessory breathing muscles during inhalation.

Muscle sites

1. Trapezius Either one or both sides may be monitored at once. Electrodes are placed over the upper trapezius, with the ground elsewhere. The display shows a rising and falling trace proportional to the magnitude of muscle contraction. The greater the movement, the more the trapezius is participating in the inhalation. When the signal is minimized, upward shoulder movement is inhibited and air is directed lower down in the body. The patient can attempt to breathe exclusively with the lower abdominal muscles and inhibit trapezius movement altogether. Though probably not natural, attempting to achieve this builds control and the ability to inhibit shoulder breathing.

2. Scalenes Located midway between trapezius and sternocleidomastoid (SCM) muscles on the side of the neck, these muscles attach variously to the clavicle and 1 and 2 ribs to elevate them, thus increasing the volume of the upper chest. They are less easily isolated and monitored than the trapezius, but it is worthwhile learning to inhibit their contractions for normal breathing. This delegates more of the work of inhalation to the lower costal and diaphragmatic muscles (Figs. 8.4 and 8.5).

3. Sternocleidomastoid This muscle mainly turns the head to the side, but it also aids in lifting the clavicles and expanding the chest. If the scalenes are active in quiet breathing, then the SCM will probably be also. It can be hard to isolate with the electrodes since it moves laterally under the skin, so the patient's head must be held still and facing straight ahead (see Ch. 6 for more discussion of these muscles).

Temperature feedback

A biofeedback thermometer with fast response and small thermistor can be used to monitor and display the difference in temperature between inhaled and exhaled air. The tip of the wire thermistor is taped onto the upper lip in the airstream outside the nostril without touching the skin. Depending on ambient temperature the difference between inhaled and exhaled air is usually between 2 and 10°F, sufficient to mark the breathing cycle. Though insensitive to location of breathing (chest vs. abdomen) it does verify nasal as opposed to mouth breathing. This display is very sensitive to speed of exhale during exhale, and is useful for reinforcing slower exhalation. Audio feedback can enhance the experience.

All of these biofeedback modes can be helpful as temporary learning aids. People with 'bad breathing' habits often seem relatively unaware of proprioceptive sensations; this may be a subtle perceptual or sensory deficit or a habituation to 'error signals' that arise from habitual chest breathing, mouth breathing, or excessive speed. The biofeedback information helps to direct conscious attention to the relevant sensations. The goal is to sensitize the patient to how suboptimal breathing feels compared with optimal breathing. Other methods such as hand-on-abdomen, the practitioner's hands on shoulders, breathing into a candle flame, lying prone on the floor (for feedback from whatever area is expanding most), and mirror feedback can all help to extend conscious control to the breathing realm.

Sighing

Frequent sighing is frequently mentioned by researchers and clinicians alike as a sign of the chronic hyperventilator. It contributes to breathing instability and has been interpreted variously as responses to surges of emotion, an expression

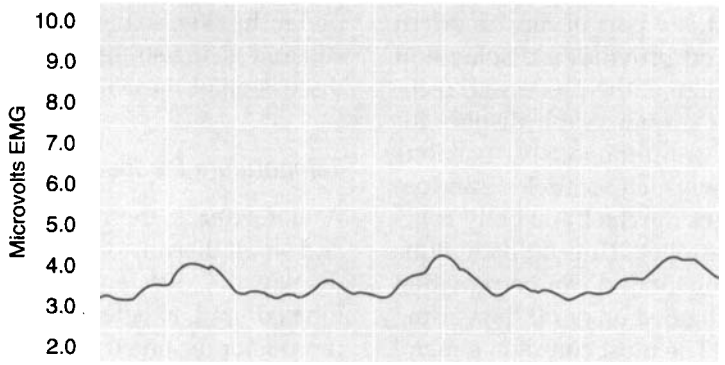

Figure 8.4 EMG tracing of scalene muscle (side of neck) during normal breathing (three breaths) with abdominal expansion and shoulders relaxed.

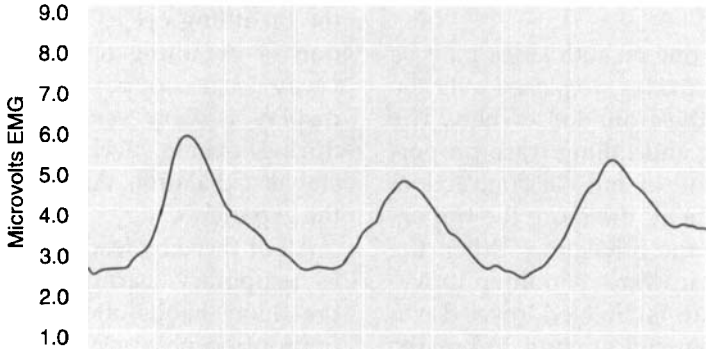

Figure 8.5 EMG tracing of scalene muscle (three breaths) with abdominal movement restricted; accessory muscles recruited.

of dyspnea, hypersensitivity to rising CO_2 levels, and an attempt to loosen tight breathing muscles. A recent study (Wilhelm et al 2001) looked at this variable in more detail by monitoring breathing volumes, timing, and end-tidal CO_2 in panic disorder patients and others. Compared to controls, sighing was more frequent in those with panic disorder, sighs were deeper, and return to baseline was slower. The authors concluded that the lower baseline CO_2 present in those with panic disorder was not explainable by minute volume in general, but by frequency and depth of sighing, plus slower recovery. The sighs did not occur following a rise in CO_2 and they did not appear to be attempts to correct for pauses in breathing.

This suggests that training steadiness of breathing and suppression of sighing is important for reducing hyperventilation. The explanations of individuals vary as to why they sigh so often, and they may be rationalizations for a behavior that occurs spontaneously for unknown reasons. Teaching tolerance of whatever feeling precedes sighing is sometimes successful, given sufficient self-observation. Also, teaching to limit the size of the sigh may work, plus keeping the mouth closed during a sigh. A quick swallow can terminate a sigh.

The relaxation response

The relaxation response is a term coined nearly 30 years ago by Mirian Klipper and the then associate professor of medicine at Harvard Medical School, Herbert Benson MD. It was used to

describe the conscious initiation in the body of a protective mechanism to counter the effects of anxiety and the 'fright, fight or flight' reaction to stress (Benson & Klipper 1988). First described by Dr Walter B. Cannon, a professor of physiology at Harvard Medical School at the turn of last century, the 'fight-or-flight' response was seen as an 'emergency reaction' where increases in blood pressure, heart rate, breathing and increased body metabolism were recorded. Benson believed that the more this reaction was activated the more likely high blood pressure was to develop, especially if the responses it was designed to provide – fighting or fleeing – were continually repressed.

Benson, who was also director of the hypertension section of Boston's Beth Israel Hospital, was curious to find the cause of 'essential hypertension' (of unknown cause) which affected a very high percentage of his patients. Often put down to 'stress' he determined to find out more about the then inadequately studied subject. An ill-defined and overused word, stress has come to mean different things in different areas of health care. But whatever its origin – emotional, environmental, occupational, or physiological – learning how to switch off the signs and symptoms of stress by evoking this response has been exhaustively researched and documented as well as put into a practical guide for patient use by Benson and his colleagues.

Taking centuries-old Eastern meditation techniques and the claims that physiological functions could be altered by practicing these methods was the basis of the Harvard group's exploration. Prior to this, it was generally accepted in orthodox medicine that control over the automatic functions of the body was beyond conscious control. But the Harvard studies revealed what practitioners of Eastern yogic and meditational techniques had understood empirically for many centuries – that mechanisms in the unconscious autonomic nervous system could be altered by both mental and physical relaxation techniques to slow heart, breathing, and metabolic rates and reduce both mental and physical tension. Acknowledging the age-old universality of the techniques examined, the group came up with a term and method acceptable to most skeptical Western stress sufferers.

The four basic elements for the effective practice of the relaxation response are:

1. A quiet environment to help reduce external stimuli
2. An object to dwell on, which can be either a word or sound to repeat, or something to gaze at such as a flower or symbol
3. A passive attitude by letting all thoughts and distractions float away (this appears to be the most important factor in eliciting the response)
4. A comfortable position in sitting to prevent falling asleep.

(These four elements put together while lying down help promote sleep.)

The recommended practice, developed at Harvard's Thorndike Memorial Laboratory, is to schedule two sessions daily of between 10 and 20 minutes. Sitting comfortably, the patient relaxes the muscles from the feet up, and then focuses on breathing in and out through the nose and silently saying the word 'one' at the end of each exhalation. A simple technique to use, without attachment to any cult or philosophy, the relaxation response is an acceptable method to teach patients who fear ancient or 'godless' practices or who cannot afford to pay to attend other types of courses.

Clinical tips

In practice, this technique is best taught once abdominal nose breathing patterns have been restored. Some patients may have already felt the relaxed pause at the end of exhalation, and the new sensation of 'letting the in-breath start itself,' and appreciate its calming qualities.

The majority of patients, when asked if they practice any form of relaxation, usually answer in the negative or exclaim they never have enough time to 'sit round doing nothing.' Reinforcing the need to take 'time out' can be made more acceptable if the patient perceives they are doing something (even if it is 'doing nothing').

As mentioned above, patients with strong religious affiliations may balk at any technique which

smacks of a philosophy other than their own. Packaging it as all-in-one method which includes:

- continued breathing retraining
- 'time out'
- relaxation

makes it acceptable to most. Accepting that many people have to work long hours in stressful jobs and find it hard to practice such commonly taught methods as transcendental meditation (TM), which requires 20-minute sessions twice a day, teaching 'mini relaxes' to practice throughout the day becomes an equally acceptable way of turning on the relaxation response and reducing hyperarousal levels. (This compares with the more recent exercise prescriptions stating several short sessions of exercise daily are as effective as the more prolonged sessions previously required.)

Patients can learn to switch on their relaxation response while reclining by tuning in to light abdominal nose breathing, concentrating on the exhale and the silently repeated word 'one' (or whatever they choose to focus on).

Taking blood pressure readings before and after a relaxation session in patients who have hypertension may be helpful, although often the very sight of a sphygmomanometer cuff is enough to instantly raise the patient's blood pressure ('white coat fever').

A session discussing and practicing this technique in the sitting position for further daily practice equips the patient with physical coping skills to deal with stressors wherever they occur in daily life.

Cognitive therapy

An important step in this process is helping the patient to deal with the rapid, fairly automatic assumptions being made about the meaning of the symptoms. Rapid heartbeat, for instance, could represent impending heart attack. With that interpretation dominating, it is no wonder that a panic attack ensues. Mild dyspnea resulting perhaps from a tensed diaphragm muscle could be interpreted as impending suffocation, lightheadedness as about to lose consciousness, tingling fingers as an approaching stroke, and so on.

Helping the patient to reinterpret these 'symptoms' as mere sensations which result from the hyperventilation coupled with a cognitive proneness to catastrophizing, provides a very new angle on things.

The concept of automatic thoughts has been developed by the cognitive therapy school (Salkovskis 1988) and holds that such thoughts emerge from underlying assumptions about what things mean. The deepest level of belief might be 'I am at particular risk from heart attack because there is heart disease in my family.' Given that belief, the individual will be predisposed to react to benign chest sensations, heart palpitations, or tachycardia as a confirmation of these deeply held beliefs. The automaticity of this process means that the matter is not necessarily referred to the conscious mind for decision; it has already been decided. The conscious mind becomes occupied instead with the next step, such as deciding where the closest hospital is. This anxiety will feed back into the physiological system which is producing the symptom, sustaining the state or provoking it to even greater levels of alarm.

One should question the patient about the assumed meaning of symptoms related to hyperventilation. Teaching control of breathing without explanation may not be sufficient, even though the chest or neurological symptoms abate, if the patient covertly decides that a heart attack has been averted. Intervening at a higher cognitive level – challenging the assumption that the sensations signal approaching disaster – can interrupt a disruptive and very persistent vicious cycle. Clark (1986) and Salkovskis et al (1996) represent this clinical approach, and find that breathing retraining can often be bypassed when the implicit meaning of the hyperventilation symptoms is uncovered, challenged, and changed.

Cognitive therapy takes account of thinking errors, but tends to stay at the conscious level of mental life. Emotional interference with breathing can be quite complex; the state of confusion and disorientation resulting from hyperventilation can be rewarding or punishing. The act of hyperventilating can create depression, agoraphobia, and fear of death; the symptoms may lead to misdiagnosis of schizophrenia, hysteria,

conduct disorders, and hypochondria. It can aggravate pre-existing psychiatric disorders. Fensterheim (1994) reviews these matters and stresses the prevalence of fear of death in people who hyperventilate. This can include a general feeling of doom or imminent death, the fear of death or loss of a significant person, and fear of fatal medical illness.

Non-cardiac chest pain

When chest pain or tightness occur, it is usually attributed at first to problems in the heart, especially if there is a family history or other risk factors. If cardiac examination shows no heart problems, other explanations will be considered: asthma, muscle tension, esophageal disorders, or arthritis of the rib–sternal joints. The pain may be labeled 'angina pectoris' if associated with physical exertion. If the chest symptoms coincide with emotional stress, however, diagnosis becomes more complicated because stress is associated with a style of breathing which can generate pain or tightness in the chest. Although the discomfort occurs in the heart region, it is not necessarily related to the heart. Unpleasant sensations can arise from intercostal and other breathing muscles due to prolonged upper chest breathing, with or without hyperventilation.

Restriction of abdominal breathing forces the chest and shoulder muscles to do the work of breathing. This may come about from tight clothing, a habit of bracing the abdominal muscles, or from simply inhibiting abdominal expansion for reasons of personal appearance. Friedman (1945) did the definitive experiment in this area by tight strapping of the abdomen and lower ribs of normal volunteers. Chest pain developed within a day or two, and the trouble was exacerbated during exercise. Also, patients already suffering from chest pain (called in those days 'neurocirculatory asthenia') had their chests strapped to

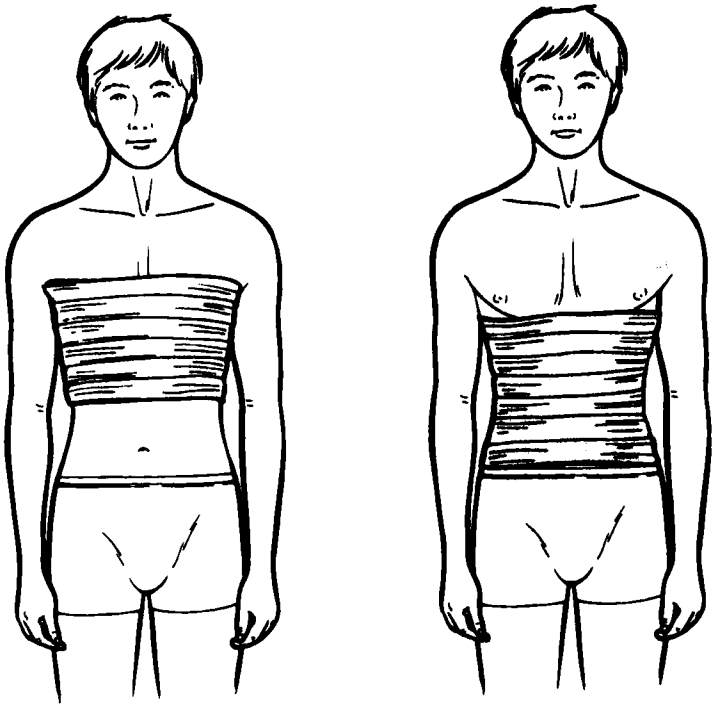

Figure 8.6 Non-cardiac pain related to faulty breathing. Habitual thoracic breathing can create angina-like chest pain from overuse of accessory breathing muscles. Such pain was abolished within 2 days by tight strapping of the upper chest to prevent expansion, and could be created in normal subjects by tight strapping of the abdomen and lower ribs. (Reproduced with kind permission from Gilbert 1999b.)

prevent the exaggerated thoracic breathing which Friedman considered was causing the pain. Forced to adopt more abdominal breathing, these patients lost their pain within 2 days, but it reappeared when the strapping was removed and their habitual breathing returned (Fig. 8.6).

This is not to suggest that incorrect breathing does not affect the heart; Nixon (1989, 1993a, 1994) has reviewed and documented the effect of chronic hyperventilation on the heart itself. He has focused on the physiological consequences of a continuing personal struggle to close the gap between existing performance and what seems required. He calls this the 'effort syndrome,' which stimulates hyperventilation via preparation for effort. This worsens the situation by unbalancing the autonomic nervous system toward the sympathetic end, triggering tachycardia, rise in blood pressure, and ultimately exhaustion as more effort is expended for less and less return. Homeostatic priorities are sacrificed. Hyperventilation over time disrupts the balance of potassium, magnesium, phosphorus and calcium which play intricate interlinked roles in maintaining proper cardiac function. Ultimately, the loss of alkaline buffer (see Ch. 3) and increase of lactic acid production reduces tolerance for exercise. This is apparent in the lower threshold for anaerobic muscle metabolism during standard exercise testing, indicating metabolic acidosis. Details can be found in Nixon (1994) and in Von Schéele & Van Schéele (1999).

A large number of cases of non-cardiac chest pain are eventually diagnosed as 'functional' and are most likely attributable to chronic hyperventilation (Bass & Wade 1984). Cardiovascular symptoms are reported by nearly all panic patients. An intervention study (De Guire et al 1992) attempted to reduce symptoms in patients who reported chest pain, palpitations, shortness of breath, arrhythmias, tachycardia, and paresthesias. Breathing retraining, the main intervention, consisted of teaching slower, more abdominal breathing. Some patients also received more involved explanations and training with respiratory strain gauges and CO_2 feedback. Significant improvement occurred in 6 weeks, and included reduced respiratory rate, increased end-tidal CO_2, and reduction in symptoms. Reduced breathing rate and the subject's belief that they were maintaining paced abdominal breathing were the strongest predictors of success. Exposure to the strain gauges and CO_2 feedback did not enhance the results.

At follow-up 3 years later, the improvements had held, with an average 10% rise in CO_2 levels and a drop from 16 to 10 breaths per minute, and corresponding drop in symptom occurrence. Potts and colleagues (1999) obtained similar success with 60 patients having chest pain but normal coronary arteries. Belief in their efficacy contributed to symptomatic improvement in this study also; those who still attributed their chest pain to heart disease had poorer outcomes.

The conclusion from these studies seems to be that teaching improved breathing to people having non-cardiac chest symptoms is quite feasible in a matter of weeks, and can bring substantial relief. Expectation and belief in one's ability to control breathing are involved, though this may interact with success in learning the breathing techniques.

Respiratory sinus arrhythmia (RSA)

This term refers to the cyclic rise and fall of the heart rate in rhythm with breathing. Laymen tend to consider a steady, unvarying heartbeat a good sign, perhaps because skipped beats, palpitations, extra beats, and a racing heart all seem to involve deviations from clock-like steadiness. The truth is actually the opposite: a heartbeat that is steady like a metronome is not generally a sign of health, and if chronic, usually indicates high stress and/or cardiac damage.

In a relaxed, healthy person, the heart rate normally rises part way through each inhalation, and then drops with the exhalation. The range of variation in beats per minute can range from fewer than 5 to more than 30, but averages between 8 and 14. RSA is often used as an index of general vagal tone since there is a waxing and waning of parasympathetic influence with each breath when the autonomic nervous system is properly balanced. Deeper breathing amplifies the variability; atropine, a parasympathetic

inhibitor, will abolish RSA, as will anxiety and other influences which tip the balance toward sympathetic dominance.

Much basic physiological research is being done now in this area, and a certain amount of clinical research as well. The general assumption is that learning to increase the RSA can promote a healthier balance between sympathetic and parasympathetic dominance. Chronic disorders which involve autonomic imbalance, such as asthma, do seem to respond to the slow steady breathing involved in RSA training. Current research often uses the term 'resonant frequency biofeedback training,' which refers to using heart rate feedback for seeking a resonance between the breathing rhythm and the Traub-Hering-Mayer wave, which is tied to blood pressure regulation. The ideal breathing rate should be determined for each individual, but in most people a rate around six breaths per minute, or one complete breath every 10 seconds, establishes the resonance. That frequency is often mentioned as optimal by yoga practitioners and others who work with breathing relaxation.

Lehrer et al (1997) trained a number of asthmatics to increase RSA amplitude via RSA biofeedback, and compared their outcomes to others who learned an enhanced abdominal breathing technique by other means. Respiratory impedance (a measure of bronchoconstriction) was reduced in the RSA-trained group only. This provided confirmation for results reported from the Center for Medical Rehabilitation for Children in St Petersburg, Russia, where RSA biofeedback has been used for several years to treat thousands of children with bronchial asthma (Smetankin 1997) with a claimed 70% rate of success. Another study (Lehrer et al 2000) found improvements in spirometic measures in asthmatic children trained with RSA biofeedback.

The theory behind why RSA training should work has to do with exercising baroreceptor reflexes which regulate blood pressure. This is hypothesized to strengthen homeostatic activity, and this in turn helps modulate and limit stress-induced perturbations which may affect the entire autonomic nervous system. By this means, 'parasympathetic rebound' following stress-induced sympathetic arousal would be inhibited. This rebound phenomenon is thought to be a factor in asthma. Sympathetic dominance provokes bronchodilation (thus, adrenaline as an emergency treatment for asthma attacks) but when the body strives to recover its balance the rebound can go too far toward parasympathetic dominance, which is associated with bronchoconstriction.

Monitoring this variable can involve very elaborate electrocardiographic equipment, but any device which can display heart rate and updates rapidly can give rough biofeedback. Simply palpating the pulse will give an indication of whether the heartbeat rises and falls with the breathing. More details on this technique and other cardiovascular aspects of breathing can be found in Gilbert (1999b).

Van Dixhoorn

In the area of cardiac rehabilitation, one of the largest studies of cardiac rehabilitation including breathing based relaxation was done in The Netherlands by Jan van Dixhoorn (van Dixhoorn et al 1989, van Dixhoorn & Duivenvoorden 1989). In this study 156 survivors of recent heart attacks were randomly divided into two groups. All patients participated in standard aerobic conditioning exercises, but members in one group received an additional 6 hours of individual instruction in a simple relaxation technique which centered on breathing. This was a short-term intervention, a matter of weeks, but follow-up data were collected for several years afterward.

The main conclusions relevant here were that incidence of subsequent heart trouble was lower in the relaxation-plus-exercise compared with the exercise-only group. The breathing-based relaxation procedure provided a clear advantage over aerobic training alone. Breathing rate at follow-up was reduced, and respiratory sinus arrhythmia was stronger in the relaxation group, indicating lowered sympathetic tone. The average heart rate of relaxation-trained patients was lower, as was the incidence of ST-segment depression (meaning less evidence of myocardial ischemia). The changes in breathing persisted at

a 2-year follow-up. Serious cardiac incidents occurred in 37% of the exercise group, compared to 17% in the relaxation group. A 5-year follow-up analysis showed that hospitalizations for cardiac problems were still lower in the relaxation group, and that the cost of the extra relaxation training intervention had been more than offset by the reduction of medical costs. All this resulted from 6 hours of instruction.

Method used The way that the author Jan van Dixhoorn taught relaxation via breathing is unique (Box 8.1). The essence of the technique consisted of comparing one's state (physical, mental, emotional) before and after the relaxation procedure, and noting the difference. Improved body awareness, rather than performance of a particular physical routine, was considered central to the entire intervention, and the desired respiratory change was coupled to passive awareness and self-observation. Breathing-based relaxation was integrated into daily routines by practicing in a variety of positions and situations.

Box 8.1 Van Dixhoorn's breathing techniques used in cardiac rehabilitation

- Deemphasize the idea of 'exercises' and 'performance'
- Emphasize self-observation and self-awareness, with many brief interventions throughout the day
- Practice both 'active relaxation' (during movement) and 'passive relaxation'
- Separate the intervention into two components: relaxed breathing and self-observation
- Encourage awareness and involvement of entire body in breathing
- Use manual touch to facilitate awareness of abdominal expansion, muscular tension, and participation of the ribs and back
- Use EMG biofeedback from the forehead
- Encourage experimentation with breathing variables (depth, rate, location, ease, regularity)
- Patient's hand placed on abdomen, with the direction: 'The hand notices what the body does'
- Directing attention to subtle body changes during exhale vs. inhale, such as the distance between sternum and pubic bone, magnitude of lumbar and cervical curves

Van Dixhoorn's recommended rest breaks were defined as exercises in self-awareness rather than 'relaxation' which his population of cardiac patients would have considered synonymous with being lazy or 'falling behind.' Van Dixhoorn's intervention neatly sidestepped this conflict by assigning a 'before and after' cognitive comparison. To do this requires entering an objective state that disrupts ongoing emotional states which contribute to stress.

In the six individual training sessions van Dixhoorn and his colleagues used several methods to initiate a self-awareness which was considered the key to restoring homeostatic balance (van Dixhoorn & Duivenvoorden 1989). In his words: 'The patient learned to elicit and observe a "shift" in the respiratory pattern, such that inspiration expanded both the lower abdomen and the costal margin, and expiration was moderated and slow' (van Dixhoorn et al 1990). Pursed-lips breathing to make the air movement audible increased breathing awareness further.

Van Dixhoorn went beyond encouraging diaphragmatic movement to improving the coordination of diaphragm action with other respiratory muscles in the abdomen, back, and chest. Efficient breathing actually involves movement of the whole trunk, the pelvis, spine, and sternum. He noted, for instance, that the top of the pelvis rotates subtly forward during inhalation, that the lumbar curve increases while the cervical curve decreases, and that the distance between the symphysis pubis and the sternum increases. Individualized instruction sometimes included manual touch to focus attention and to provide tactile feedback. For instance, he would place his hands on the abdomen or lower back and instruct the patient to 'breathe into my hands.' This gentle and personal instruction downplayed prescribing the 'proper' proper way to breathe. He encouraged patients instead to experiment with depth, rate, location, and timing, and then observe any differences in feeling state.

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