The structure and function of breathing

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THE STRUCTURE–FUNCTION CONTINUUM

Nowhere in the body is the axiom of structure governing function more apparent than in its relation to respiration. This is also a region in which prolonged modifications of function – such as the inappropriate breathing pattern displayed during hyperventilation – inevitably induce structural changes, for example involving accessory breathing muscles as well as the thoracic articulations. Ultimately, the self-perpetuating cycle of functional change creating structural modification leading to reinforced dysfunctional tendencies can become complete, from whichever direction dysfunction arrives, for example: structural adaptations can prevent normal breathing function, and abnormal breathing function ensures continued structural adaptational stresses leading to decompensation.

Restoration of normal function requires restoration of adequate mobility to the structural component and, self-evidently, maintenance of any degree of restored biomechanical integrity requires that function (how the individual breathes) should be normalized through re-education and training.

MULTIPLE INFLUENCES: BIOMECHANICAL, BIOCHEMICAL, AND PSYCHOLOGICAL

The area of respiration is one in which the interaction between biochemical, biomechanical, and psychosocial features is dramatically evident
Inappropriate breathing can result directly from structural, biomechanical causes, such as a restricted thoracic spine or rib immobility or shortness of key respiratory muscles. Causes of breathing dysfunction can also have a more biochemical etiology, possibly involving an allergy or infection which triggers narrowing of breathing passages and subsequent asthmatic-type responses. Acidosis resulting from conditions such as kidney failure will also directly alter breathing function as the body attempts to reduce acid levels via elimination of CO2 through the means of hyperventilation.

The link between psychological distress and breathing makes this another primary cause of many manifestations of dysfunctional respiration. Indeed, it is hard to imagine examining a person suffering from anxiety or depression without breathing dysfunction being noted.

Other catalysts which may impact on breathing function include environmental factors (altitude, humidity, etc.). Even factors such as where an individual is born may contribute to subsequent breathing imbalances: ‘People who are born at high altitude have a diminished ventilatory response to hypoxia that is only slowly corrected by subsequent residence at sea level. Conversely, those born at sea level who move to high altitudes retain their hypoxic response intact for a long time. Apparently therefore ventilatory response is determined very early in life.’ (West 2000).

How we breathe and how we feel are intimately conjoined in a two-way loop. Feeling anxious produces a distinctive pattern of upper-chest breathing which modifies blood chemistry, leading to a chain reaction of effects, inducing anxiety, and so reinforcing the pattern which produced the dysfunctional pattern of breathing in the first place.

Even when an altered pattern of breathing is the result of emotional distress, it will eventually produce the structural, biomechanical changes which are described below. This suggests that when attempting to restore normal breathing – by means of re-education and exercise for example – both the psychological initiating factors and the structural compensation patterns need to be addressed. (The psychological effects of breathing dysfunction are covered in greater detail in Chapter 5.)

**Homeostasis and heterostasis**

The body is a self-healing mechanism. Broken bones mend and cuts usually heal, and most health disturbances – from infections to digestive upsets – get better with or without treatment (often faster without!), and, in a healthy state, there exists a constant process for normalization and health promotion. This is called **homeostasis**.

However, the homeostatic functions (which include the immune system) can become overwhelmed by too many tasks and demands as a result of any, or all, of a selection of negative impacts, including nutritional deficiencies, accumulated toxins (environmental pollution, either as food or inhaled, in medication, previous or current use of drugs, etc.), emotional stress, recurrent or current infections, allergies, modi-
fied functional ability due to age, or inborn factors, or acquired habits involving poor posture, breathing imbalances and/or sleep disturbances, and so on and on ... (Fig. 1.2).

At a certain point in time the adaptive homeostatic mechanisms break down, and frank illness – disease – appears. At this time the situation has modified from homeostasis to heterostasis, and at this time the body needs help – treatment. Treatment can take a number of forms, which are usually classifiable as involving one of three broad strategies:

1. Reducing the load impacting the body by taking away as many of the undesirable adaptive factors as possible (by avoiding allergens, improving posture and breathing, learning stress coping tactics, improving diet, using supplements if called for, helping normalize sleep and circulatory function, introducing a detoxification program if needed, dealing with infections) and generally trying to keep the pressure off the defense mechanisms while the body focuses on its current repair needs.

2. Enhancing, improving, modulating the defense and repair processes by a variety of means, sometimes via specific intervention and sometimes involving non-specific, constitutional methods.

3. Treating the symptoms while making sure that nothing is being done to add further to the burden of the defense mechanisms.

Not all available therapeutic measures need to be employed, because once the load on the adaptation and repair processes has reduced sufficiently, a degree of normal homeostatic self-regulating function is automatically restored, and the healing process commences.

**Therapy as a stress factor**

A corollary to the perspective of therapy being aimed at the ‘removal of obstacles to self-healing’

When homeostatic adaptive capacity is exhausted treatment calls for:
1. Restoration of immune competence, enhancement of defense capabilities, support of repair functions
2. Reduction of as many of the multiple interacting stressors impacting the individual as possible
3. Attention to symptoms (ideally without creating new problems).

*Figure 1.2 Multiple stressors in fibromyalgia. (Reproduced with kind permission from Chaitow 1999.)*
is that, since almost any form of 'treatment' involves further adaptive demands, therapeutic interventions need to be tailored to the ability of the individual to respond to the treatment. Excessive adaptive demands made of an individual, already in a state of adaptive exhaustion, are bound to make matters worse.

A clinical rule of thumb adopted by one of the authors (LC) is that the more ill a patient is, the more symptoms are displayed, and the weaker is the evidence of vitality, the lighter, gentler, and more 'constitutional' (whole person) the intervention needs to be. (See Ch. 4 for discussion of adaptation exhaustion, and Zink's protocols.) Whereas a robust, vital individual might well respond positively to several simultaneous therapeutic demands, for example a change in diet together with medication, bodywork, and rehabilitation exercises, someone who is more frail and less vital might well collapse under such an adaptive therapeutic assault. For the frail patient, a single modification or therapeutic change might be called for, with ample time allowed to adapt to the change (whether this involves exercise, posture, diet, bodywork, medication, psychological intervention, or anything else).

In any given case it is necessary to focus attention on what seems to be the likeliest and easiest targets (perhaps using a team approach in which more than one therapist/therapy is being utilized) which will achieve this desirable end. In one person this may call for rehabilitation exercises accompanied by psychotherapy/counseling; in another, dietary modification and stress reduction could be merited; while in another, enhancement of immune function and structural mobilization using bodywork and exercise may be considered the most appropriate interventions. The 'art' of health care demands the employment of safe and appropriate interventions to suit the particular needs of each individual.

**OBJECTIVES AND METHODS**

The focus of this book is on normal versus abnormal respiratory patterns (function), and how best to restore normality once an altered pattern has been established. This commonly requires the removal of causative factors, if identifiable, and, if possible, the rehabilitation of habitual, acquired dysfunctional breathing patterns, and, in order to achieve this most efficiently, some degree of structural mobilization to restore the machinery of breathing towards normality.

If rehabilitation is attempted without taking account of etiological features or maintaining features – restricted rib articulations, shortened thoracic musculature, etc. – results will be less than optimal.

An example of extreme breathing pattern alteration is hyperventilation. Hyperventilation and its effects occupy a major part of the book. It will also be necessary to explore the widespread gray area in which normal patterning is clearly absent, even though patent hyperventilation is not demonstrable.

A perspective needs to be held in which function and structure are kept in mind as dual, interdependent features. The thoracic cage can be thought of as a cylindrical structure housing most major organs – lungs, heart, liver, spleen, pancreas, kidneys. The functions associated with the thorax (or with muscles attached to it) include respiration, visceral activity, stabilization, and movement (and therefore posture) of the head, neck, ribs, spinal structures, and upper extremity.

The causes of dysfunctional breathing patterns will be seen to possibly involve etiological features which may in nature be largely biomechanical (for example post-surgical or postural factors), biochemical (including allergic or infection factors), or psychosocial (chronic emotional states such as anxiety and anger). Etiology may also involve combinations of these factors, or established pathology may be the cause. In many instances, altered breathing patterns, whatever their origins, are maintained by nothing more sinister than pure habit (Lum 1994).

Where pathology provides the background to altered breathing patterns, the aim of this book is not to explore these disease states (e.g. asthma, cardiovascular disease) in any detail except insofar as they impact on breathing patterning (for example where airway obstruction causes normal nasal inhalation to alter to mouth breathing). The changes which concern this text are largely func-
tional in nature rather than pathological, although the impact on the physiology of the individual of an altered breathing pattern such as hyperventilation can be profound, possibly resulting in severe health problems ranging from anxiety and panic attacks to fatigue and chronic pain.

It is axiomatic that in order to make sense of abnormal respiration, it is essential to have a reasonable understanding of normality. As a foundation for what follows, this chapter will outline the basic characteristics of normal breathing. The biochemical and alveolar processes involved in respiration (as distinct from the biomechanical process of breathing) will be covered in later chapters.

NORMAL BREATHING

RESPIRATORY BENEFITS

Optimal respiratory function offers a variety of benefits to the body:

- It allows an exchange of gases involving — the acquisition of oxygen ($O_2$) — the elimination of carbon dioxide ($CO_2$).
- The efficient exchange of these gases enhances cellular function and so facilitates normal performance of the brain, organs, and tissues of the body.
- It permits normal speech.
- It is intimately involved in human non-verbal expression (sighing, etc.).
- It assists in fluid movement (lymph, blood).
- It helps maintain spinal mobility through regular, mobilizing, thoracic cage movement.
- It enhances digestive function via rhythmic positive and negative pressure fluctuations, when diaphragmatic function is normal.

Any modification of breathing function from the optimal is capable of producing negative effects on these functions.

THE UPPER AIRWAY (Figs 1.3–1.10)

To enter the air sacs of the lungs, air journeys through a series of passages: nose, nasopharynx, oropharynx, laryngeal pharynx, larynx, trachea, bronchi, and bronchioles. Disease and dysfunction can affect any of these segments and cause abnormal breathing patterns, and it is important to recognize and treat any such conditions before attempting to correct abnormal patterns such as hyperventilation.

The nose

The nose is an intriguing characteristic facial feature, taking on a variety of shapes and sizes, and changing with age. It is a complex structure with a number of vital functions:

- Air enters each narrow nostril (the external naris), streaming into a tall cave.
- Further turbulence is created by three curved bony plates (termed conchae) on the outer wall. These increase the mucosal surface area.
- Bristling hairs (vibrissae) inside the nostril trap large floating debris, while fine dust is arrested by a forest of fine hairs and a film of mucus floating on the nasal mucosa.
- In this fashion, the air is filtered, warmed, and humidified before leaving the nose via large apertures (choanae) above the hard palate.
- The mucosa has a rich blood supply providing heat and fluid, and is thickest over the tips of the conchae.
- During (for instance) a cold, the mucous membrane can swell, blocking the passage of air.
- Air in the upper reaches of the tall cave can enter attics (cool air sinuses) through narrow openings (ostia). Here, warming and further filtering takes place.
- These sinuses are named the frontal behind the forehead, the maxillary behind the cheek, and the ethmoid and sphenoid under the bridge of the nose.
- The dust-laden mucus can work forward toward the nostrils, where it dries and can be removed.
- There is a backward flow as well, and this collection can be swallowed or coughed out.

Box 1.1 gives details of the osseous and muscular components of the nasal apparatus, and Chapter 6 describes palpation/treatment exercises relating to them.
Box 1.1 Key facial and cranial structures associated with the nasal function

![Diagram of facial and cranial structures related to nasal function.]

Figure 1.3 A The right side of the septum of the nose, showing its constituent bones and cartilages. B The lateral wall of the right half of the nasal cavity: internal aspect. C An outline of the bones of the face, showing the positions of the frontal and maxillary sinuses. (Reproduced with kind permission from Gray 1989.)

**Sphenoid (see Fig. 1.4)**
- The body of the sphenoid – a hollow structure enclosing an air sinus – is situated at the centre of the cranium.
- Two 'great' wings, the lateral surfaces of which form the temples, are the only aspect of this bone palpable from outside the head.
- The anterior surfaces of the great wings form part of the eye socket.
- The anterior surfaces of the two lesser wings form part of the eye socket.
- Two pterygoid processes hang down from the great wings and are palpable intraorally posteromedial to the eighth upper tooth.
- The pterygoid plates form part of the pterygoid processes and are important muscular attachment sites.

- The sella turcica ('Turkish saddle') houses the pituitary gland.
- The sphenobasilar junction with the occiput is a synchondrosis which fuses in adult life.

**Articulations relevant to respiration**
- Anteriorly with the ethmoid.
- Inferiorly with the palatine bones.
- Inferiorly with the vomer.
- Anterolaterally with the zygoma.

**Relevant muscular attachments**
- The temporalis muscle attaches to the great wing and the frontal, parietal, and temporal bones, crossing important sutures such as the coronal, squamous, and frontosphenoidal.
Specifically, the attachments of temporalis are to the temporal bone, the zygomatic arch, the mandible, and the lateral and medial pterygoid plates of the sphenoid.

- Attaching to the internal pterygoid plate are buccinator as well as a number of small palate-related muscles.
- Medial pterygoid attaches to lateral pterygoid plate and palatine bones, running to the medial ramus and angle of the mandible.
- Lateral pterygoid attaches to the great wing of the sphenoid, the lateral pterygoid plate, and the anterior neck of the mandible.

**Neural associations**
The first six cranial nerves have direct associations with the sphenoid, with the 2nd (optic), 3rd (part of oculomotor), 4th (trochlear), 5th (nasociliary, frontal, lacrimal, mandibular, and maxillary branches of trigeminus), and 6th (abducens) all passing through the bone into the eye socket (the 1st, the olfactory nerve, runs superior to the lesser wings).

**Ethmoid** (see Fig. 1.5)
A tissue paper thin construction comprising a central horizontal plate (cribriform) which contains tiny openings for the passage of neural structures, surrounded by shell-shaped air sinuses forming a honeycomb framework to each side of the plate, which is crowned by: a thin crest (crista galli) formed by the dragging attachment of the faix cerebrif; thin, bony, plate-like structures which form the medial eye socket; additional projections and plates, one forming part of the nasal septum, with the perpendicular plate being a virtual continuation of the vomer (see below).
Articulations
There are interdigitated sutures with the sphenoid and non-digitated sutures with the vomer, nasal bones, palatines, maxillae, and frontal bone.

Reciprocal tension membrane relationships
- The falx cerebri attaches directly to the crista galli
- The inferior border connects with the nasal cartilage

Other associations and influences
Air passing through the shell-like ethmoid air cells is warmed before reaching the lungs and the alternation of pressures as air enters and leaves the ethmoid results in minor degrees of motion between it and its neighboring structures. Because in life its tissue paper like delicacy has a spongy consistency, it must be presumed that the structure acts as a local shock absorber.

The first cranial (olfactory) nerve lies superior to the cribriform plate and from this derive numerous neural penetrations of it which innervate mucous membranes which provide the olfactory sense.

Treatment protocols for the ethmoid will be found in Chapter 6.

Vomer
This is a plough-shaped sandwich of thin, bony tissue which houses a cartilaginous membrane, which forms the nasal cartilage. It is a junction point between the ethmoid and the maxillae, and the maxillae and the sphenoid.

Articulations
- Superiorly, it articulates with the sphenoid at a tongue-and-groove joint of spectacular beauty, as the vomer forms two wing-shaped expansions which dovetail with the receptacle offered by the inferior aspect of the centre of the sphenoid
- On the inferior aspect of the sphenoid the vomer also has minor articulation contacts with the palatine bones at the rostrum
- There is a direct, plain (not interdigitated) suture with the ethmoid at its anterolateral aspect, the vomer
Box 1.1 (Continued)

**being a virtual continuation of the ethmoid's perpendicular plate**

- The inferior aspect of the vomer articulates with the maxillae and the palatines
- There is a cartilaginous articulation with the nasal septum

**Muscular attachments**
- There are no direct muscular attachments.

**Associations and influences**
- As with the ethmoid, this is a pliable, shock-absorbing structure which conforms and deforms, depending on the demands made on it by surrounding structures. The mucous membrane covering the vomer assists in warming air in nasal breathing.

**Muscular attachments**
- Levator labii superioris arises from the frontal portion of the maxilla and runs obliquely laterally and inferior to insert partly in the greater alar cartilage and partly into the upper lip. Its actions are to raise and evert the upper lip and dilate the nostrils

**Associations and influences**
- The zygomatico-facial and zygomaticotemporal foramina offer passage to branches of the 5th cranial nerve (maxillary branch of trigeminal).

**Maxilla** (see Fig. 1.6)

This extremely complex bone is made up of:

- The body which houses an air sinus
- A superior concave orbital surface which forms part of the floor of the eye socket
- An infraorbital foramen and canal which offers passage to part of the 5th cranial (trigeminal) nerve and to the infraorbital artery
- An anterior spine to which the nasal septum attaches
- An aperture (maxillary hiatus) on the medial wall of the air sinus which is largely covered by the palatines posteriorly and the inferior conchae anteriorly
- A jutting superior projection which articulates by interdigitation with the frontal bone
- A notch (ethmoid notch) on the medial surface of this projection which articulates with the middle conchae
- A lateral zygomatic process which articulates with the zygoma at the dentate suture
- An inferiorly situated palatine process which forms most of the hard palate (anterior portion)
- A suture which runs transversely across the palate where the maxillary palate and the palate bone articulates (maxillopalatine suture)
- A central (Incisive) canal, placed inferiorly and anteriorly, for passage of the nasopalatine nerve
- The alveolar ridge, an anterior/inferior construction for housing the teeth

**Articulations**
- As described above, the maxillae articulate at numerous complex sutures with each other, as well as with the teeth they house, the ethmoid and vomer, the palatines and the zygoma, the inferior conchae and the nasal bones, the frontal bone and the mandible (by tooth contact), and sometimes with the sphenoid.

**Muscular attachments**
- Medial pterygoid runs from the palatine bones and the medial surface of the lateral pterygoid plate of the sphenoid and the tuberosity of the maxilla to the
Figure 1.6 A Left maxilla, lateral aspect, showing major features, articulations, and muscular attachment sites. B Left maxilla, medial aspect, showing major features and articulations. (Reproduced with kind permission from Chaitow 1998.)

Box 1.1 (Continued)
Box 1.1 (Continued)

lateral ramus of the mandible and deep to the coronoid process and upper ramus of the mandible

- Buccinator is a thin four-sided muscle which forms part of the cheek, occupying the space between the maxilla and the mandible. It attaches to the alveolar processes of the maxilla and the mandible, opposite the three molar teeth. Its fibers converge toward the angle of the mouth and the lips. Its action is to compress the cheeks against the teeth during chewing and it is involved in the act of blowing (‘buccinator’ means trumpeter)

- Of lesser importance but also attaching to the maxillae are various muscles, many of which have to do with facial expression and with mouth movement in eating, such as orbicularis oris, depressor anguli oris, levator labii superioris, levator labii superciliis alaeque nasi, levator anguli oris, nasalis, depressor septi nasi, risorius

- There are also strong influences from the muscles of the tongue, although these do not directly attach to the maxillae

Associations and influences

Because of the involvement of both the teeth and the air sinuses, the causes of pain in this region are not easy to diagnose. These connections (teeth and sinuses), as well as the neural structures which pass through the bone, plus its multiple associations with other bones and its vulnerability to trauma, make it one of the key areas for therapeutic attention where problems associated with the area are concerned.

Treatment protocols for the maxillae will be found in Chapter 6.

Palatines

This complex, extremely thin, hook-shaped structure includes:

- A perpendicular plate which forms part of the wall for the maxillary sinus
- A horizontal plate which makes up the posterior aspect of the hard palate as well as the floor of the nose
- A pterygoid process which articulates with the sphenoid
- An ethmoidal crest which articulates with the middle conchae of the ethmoid
- A ridge which articulates with the inferior conchae
- An orbital process which articulates with the maxilla, ethmoid, and sphenoid
- A sphenoidal process which articulates with the vomer and the inferior aspect of the sphenoid

Diaphragmatic and intercostal breathing

Breathing through the nose involves overcoming a resistance, and this favours the slow, rhythmic, diaphragmatic breathing of sleep, rest, and quiet activity. As exercise increases the nostrils at first widen, but when larger volumes of air are required the person resorts to mouth breathing, where there is much less resistance to flow. This breathing involves the intercostal and anterior neck muscles and is termed ‘intercostal breathing’.

- A nasal crest which is a continuation of the suture which links the two palatines (median palatine suture)
- A sulcus which houses the greater palatine nerve and the descending palatine artery

Articulations

- The conchal crest for articulation with the inferior nasal concha
- The ethmoidal crest for articulation with the middle nasal concha
- The maxillary surface has a roughened and irregular surface for articulation with the maxillae
- The anterior border has an articulation with the inferior nasal concha
- The posterior border is serrated for articulation with the medial pterygoid plate of the sphenoid
- The superior border has an anterior orbital process, which articulates with the maxilla and the sphenoid concha, and a sphenoidal process posteriorly, which articulates with the sphenoidal concha and the medial pterygoid plate, as well as the vomer
- The median palatine suture joins the two palatines

Muscular attachments

The medial pterygoid is the only important muscular attachment. It attaches to the lateral pterygoid plate and palatine bones running to the medial ramus and angle of the mandible.

Associations and influences

- These delicate shock-absorbing structures with their multiple sutural articulations spread force in many directions when any is exerted on them
- They are capable of deformation and stress transmission and their imbalances and deformities usually reflect what has happened to the structures with which they are articulating
- Great care needs to be exercised in any direct contact on the palatines (especially cephalad pressure) because of their extreme fragility and proximity to the sphenoid in particular, as well as to the nerves and blood vessels which pass through them (see cautionary note below).

\[\text{CAUTION: No guidelines are presented in this text}\]

for treatment involving the palatines due to reported iatrogenic effects resulting from inappropriate degrees of pressure being applied (McPartland 1996).
Sense of smell

The sense of smell, the second function, is served by an olfactory membrane lining the roof of the ethmoid sinuses. Protruding among supporting cells are tiny buttons, each with three to four fine hairs lying in a special secretion. Turbulence ensures that the scent of roses, for example, or the odor of a meal cooking, persists, enabling the cilia to sense the stimulus and activate the receptor cell below. An impulse is sent along the nerve fiber to the brain for deciphering, recognition, and initiating the appropriate response – for example irritating substances can cause violent sneezing which expels the offensive material. In humans there is a further mechanism elsewhere in the nose for the recognition of powerful and dangerous substances such as smoke and noxious chemicals.
which evoke violent alarm, coughing, and avoidance behavior. The sense of smell is much more sophisticated and sensitive in the dog, which has a much larger area of olfactory membrane than man.

**Protective function**

A third function of the nose is as a defence against both viral and bacterial infections, protecting the lungs and the rest of the body against these organisms. The first reaction to invasion is a vascular engorgement, bringing phagocytes into the nasal mucosa to engulf the invading organisms. The blood also brings cells which stimulate and secrete antibodies against the invaders. The debris of phagocytes with their contents is carried via the lymphatics to small satellite lymph nodes. In many cases the infection is limited to a cold in the nose, a tonsillitis, or a pharyngitis.

**Tear duct drain into the nose**

The nasopharynx has a curved, sloping roof. Its floor is made up of a bony hard palate in the front and a soft palate behind. The soft palate ends in the uvula. In swallowing and vomiting, the soft palate and uvula rise to cut off the nasal cavity. On each side wall is the opening of the eustachian tube, draining the middle ear and equalizing the pressure with the external atmosphere. In childhood the opening is surrounded by the adenoids, a collection of lymphoid tissue.

**The oropharynx**

The oropharynx lies between the tip of the uvula above, and, below, the epiglottis, a cartilaginous flap at the back of the tongue. The mouth opens into the oropharynx (Fig. 1.7). On each lateral wall is a recess, housing the tonsil, which can vary in size. Tonsils are large in childhood, when recurrent infection and inflammation favor increased size. On the medial surface of each tonsil are 12–15 orifices of the narrow tonsilar crypts lined with stratified squamous epithelium with invading lymphocytes. Behind these are germinal centers, generating lymphocytes. The tonsils are part of the defense system of the upper airway.

The laryngeal pharynx lies between the epiglottis above, and the cricoid cartilage of the larynx below, merging with the larynx in front and the esophagus behind.

**Swallowing**

In the first, voluntary, stage of swallowing the front of the tongue is raised to press against the hard palate, pushing back the bolus of food to the soft palate, which descends to grip the bolus. With the rising of the back of the tongue, the food bolus is propelled into the oropharynx. Here the involuntary second stage ensues. The soft palate rises to close off the nasopharynx. The epiglottis is bent back to close off the entry to the larynx and the food bolus slips down into the esophagus, partly by gravity and partly by the action of the constrictor muscles of the pharynx.

**The larynx**

The larynx is the next segment in the air passages, joining the pharynx with the trachea. In its wall are nine cartilaginous plates with connecting muscles. The cavity has an upper pair of vestibular folds and a lower set, termed the vocal chords, which can be more tightly brought together. The whole structure forms a mechanism to produce speech by opposing the vocal folds to varying degrees. Alternatively the folds may be completely closed off to protect the airway from fluid and food or to enable the lungs to build up pressure to cough out sputum.

Air proceeds to the trachea, dividing into right and left bronchi and on through diminishing orders of bronchi and bronchioles to the terminal air sacs where gaseous exchange takes place.

**PATHOLOGICAL STATES AFFECTING THE AIRWAYS**

Chronic obstruction of the nose and oropharynx can arise from a deviated nasal septum, exuberant distorted conchae, enlarged adenoids, hay fever, cluster headaches, nasopharyngeal tumors,
or Wegener's granulomatosis. Obstruction increases the resistance to airflow, necessitating intercostal breathing and a switch to mouth breathing. The patient should be referred back to a general practitioner, or to an ear, nose, and throat surgeon, or perhaps an allergist. In obese people, redundancy and laxity of the mucosal folds can give rise to laryngeal obstruction and cause sleep apnea or the obstructive sleep syndrome. Tumors of the vocal chords can cause laryngeal obstruction. In acromegaly, where there is a pituitary tumor overproducing growth hormone, the vestibular folds enlarge and tend to obstruct breathing. A previous tracheotomy can give rise to a tracheal stenosis. Chronic, inadequately-treated asthma causes spasm of the bronchioles, producing an expiratory wheeze and breathlessness. Lung tumors are another cause of breathlessness, while emphysema and heart disease are more common causes.

The therapist who concentrates on psychological causes and on correcting abnormal breathing patterns in these patients is unlikely to succeed, since the major factor is not psychological but physical, is often life-threatening, and requires precise diagnosis and treatment. Such patients should be referred to an appropriate specialist – a general physician or an expert in respiratory diseases.

Is there such a thing as an optimal breathing pattern?

If structural modifications result from, and reinforce, functional imbalances (see Ch. 4 in particular) in respiration as in other functions, it is of some importance to establish whether an optimal, ideal, state is a potential clinical reality.

Since breathing function is, to a large extent, dependent for its efficiency on the postural and structural integrity of the body, the question can be rephrased: 'Is there an optimal postural state?' (Fig. 1.8).

NORMAL POSTURE AND OTHER STRUCTURAL CONSIDERATIONS

It is a truism worth repeating that in order to appreciate dysfunction, a clear picture of what lies within normal functional ranges is needed. For normal breathing to occur, a compliant, elastic, functional state of the thoracic structures, both osseous and soft tissue, is a requirement. If restrictions are present which reduce the ability of the rib cage to appropriately deform in response to muscular activity and altered pressure gradients during the breathing cycle, compensating adaptations are inevitable, always at the expense of optimal function.

In manual medicine it is vital that practitioners and therapists have the opportunity to evaluate and palpate normal individuals with pliable musculature, mobile joint structures, and sound respiratory function so that dysfunction can be more easily identified. Apart from standard functional examination, it is also important that practitioners and therapists acquire the ability to assess by observation and touch, relearning skills familiar to former generations of 'low-tech' health care providers. Assessment approaches will be outlined in Chapters 6 and 7.

Figure 1.8 Balanced posture (A) compared with two patterns of musculoskeletal imbalance which involve fascial and general tissue and joint adaptations. (Reproduced with kind permission from Chaitow 1996a.)
Is there an ideal posture?

Kuchera & Kuchera (1997) describe what they consider an ideal posture:

Optimal posture is a balanced configuration of the body with respect to gravity. It depends on normal arches of the feet, vertical alignment of the ankles, and horizontal orientation (in the coronal plane) of the sacral base. The presence of an optimum posture suggests that there is perfect distribution of the body mass around the centre of gravity. The compressive force on the spinal disks is balanced by ligamentous tension; there is minimal energy expenditure from postural muscles. Structural and functional stressors on the body, however, may prevent achievement of optimum posture. In this case homeostatic mechanisms provide for 'compensation' in an effort to provide maximum postural function within the existing structure of the individual. Compensation is the counterbalancing of any defect of structure or function.

This succinct description of postural reality highlights the fact that there is hardly ever an example of an optimal postural state, and, by implication, of optimal respiratory function. However, there can be a well-compensated mechanism (postural or respiratory) which, despite asymmetry and compensations, functions as close to optimally as possible. This is clearly an acceptable 'ideal' and approaches the reality normally observed in most symptom-free people. Where dysfunction is apparent, or symptoms are evident, a degree of adaptive overload will have occurred.

If postural features are a part of such a scenario it is necessary to take account of emotional states, occupational and leisure influences, proprioceptive and other neural inputs, inborn characteristics (for example an anatomical short leg), as well as habitual patterns of use (for example upper-chest breathing), along with clinical evidence of joint and soft tissue restrictions and imbalances. It is also necessary to be able to evaluate and assess patterns of use which indicate just how close to, or far from, an optimal postural or respiratory state the individual is. Examples of useful structural and functional assessment methods will be found in Chapters 4, 5, 6, and 7.

Further structural considerations

As described above, the cylindrical thoracic cage houses most major organs – the lungs, the heart, the liver, the spleen, the pancreas, and the kidneys. It has a number of functions associated with it (or with its muscular attachments), including visceral support and influence, stabilization, and movement (and therefore posture) of the head, neck, ribs, spinal structures, and upper extremity.

Since the volume of the lungs is determined by changes in the vertical, transverse, and antero-posterior diameters of the thoracic cavity, the ability to produce movements which increase any of these three diameters (without reducing the others) should increase respiratory capacity, under normal circumstances (i.e. if the pleura are intact).

Inhalation and exhalation involve expansion and contraction of the lungs themselves, and this takes place:

- By means of a movement of the diaphragm, which lengthens and shortens the vertical diameter of the thoracic cavity. This is the normal means of breathing at rest. This diameter can be further increased when the upper ribs are raised during forced respiration, where the normal elastic recoil of the respiratory system is insufficient to meet demands. This brings into play the accessory breathing muscles, acting rather like a reserve tank, including sternocleidomastoid, the scalenes, and the external intercostals.
- By means of movement of the ribs into elevation and depression which alters the diameters of the thoracic cavity, vertical dimension is increased by the actions of diaphragm and scalenes. Transverse dimension is increased with the elevation and rotation of the lower ribs ('bucket handle' rib action) involving the diaphragm, external intercostals, levatores costarum. Elevation of the sternum is provided by upwards pressure due to spreading of the ribs ('pump handle' rib action), and the action of sternocleidomastoid and the scalenes.
Kapandji’s model

Kapandji (1974), in his discussion of respiration, has described a respiratory model. A crude model can be created by replacing the bottom of a flask with a membrane (representing the diaphragm), and providing a stopper with a tube set into it (to represent the trachea) and a balloon within the flask at the end of the tube (representing the lungs within the rib cage). By pulling down on the membrane (the diaphragm on inhalation), the internal pressure of the flask (thoracic cavity) falls below that of the atmosphere, and a volume of air of equal amount to that being displaced by the membrane rushes into the balloon, inflating it. The balloon relaxes when the lower membrane is released, elastically recoiling to its previous position as the air escapes through the tube.

The human respiratory system works in a similar manner, while at the same time being much more complex and highly coordinated:

- During inhalation, the diaphragm displaces caudally, pulling its central tendon down, thus increasing vertical space within the thorax.
- As the diaphragm descends, it is resisted by the abdominal viscera.
- At this point, the central tendon becomes fixed against the pressure of the abdominal cavity, while the other end of the diaphragm's fibers pull the lower ribs cephalad, so displacing them laterally.
- As the lower ribs are elevated and simultaneously moved laterally, the sternum moves anteriorly and superiorly.
- Thus, by the action of the diaphragm alone, the vertical, transverse, and anteroposterior diameters of the thoracic cavity are increased.
- If a greater volume of breath is needed, other accessory muscles must be recruited to assist.
- Abdominal muscle tone provides correct positioning of the abdominal viscera so that appropriate central tendon resistance can occur. If the viscera are displaced, or abdominal tone is weak and resistance is reduced, lower rib elevation may be impeded and volume of air intake will be reduced.

STRUCTURAL FEATURES OF BREATHING

The biomechanical structures which comprise the mechanism with which we breathe include the sternum, ribs, thoracic vertebrae, intervertebral discs, costal joints, muscles, and ligaments. Structural and functional aspects of all of these are summarized below (see also Figs 1.9–1.12).

Put simply, the efficiency of breathing/respiration depends upon the production of a pumping action carried out by neuromuscular and skeletal exertion. The effectiveness of the pumping mechanism may be enhanced or retarded by the relative patency, interrelationships, and efficiency of this complex collection of structures and their activities:

- On inhalation, air enters the nasal cavity or mouth and passes via the trachea to the bronchi, which separate to form four lobar bronchi and subsequently subdivide into ever narrower bronchi until ‘At the 11th subdivision, the airway is called a bronchiole’ (Naifeh 1994).
- Normal nasal function in respiration includes filtration of the air as well as warming and humidifying it as it passes towards the trachea. This function is lost if there is obstruction of the airways involved, or in chronic mouth breathers. Quiet inhalation function should be effortless if all the mechanical characteristics of the structures involved are optimal and airways are patent. Altered compliance (the expansibility potential of the lungs and thoracic cage), tissue resistance (how elastic, fibrotic, mobile the structures are), and airway resistance all increase the amount of effort required to inhale.
- The structure of the trachea and bronchi includes supporting rings which are made up of varying proportions of cartilage – for rigidity – and elastic muscle. While the wider and more cephalad trachea has a larger proportion of cartilage, the narrower and more caudad bronchioles are almost entirely elastic.
- Gas exchange takes place in the alveoli (air sacs) which are situated toward the end of the
Figure 1.9 Anterior view of the thoracic cage. (Reproduced from Seeley et al 1995.)

Figure 1.10 The rib and its vertebral articulation. A Articulation with the thoracic vertebra. B Posterior view of the rib. (Reproduced from Beachey 1998.)
bronchioles, mainly in the alveolar ducts (Fig. 1.13). These air sacs have fine membranous walls surrounded by equally thin-walled capillaries which allow gas exchange to occur.

- In order for the lungs to expand and contract, the thoracic cavity lengthens and shortens due to the rise and fall of the diaphragm as the ribs elevate and depress to produce an increase and decrease in the anteroposterior diameter of the rib cage. Any restrictions imposed by joint or soft tissue dysfunction will retard the efficiency of this pumping process.

Some of the thoracic activities described are under muscular control, whereas others result from elastic recoil. When the chest wall is opened during surgery the lungs have a continual elastic tendency to collapse, pulling away from the chest wall, whereas the chest wall tends

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**Figure 1.11** Chest wall dimension changes during breathing. The left column illustrates the pump-handle movement of the ribs. The right column illustrates the bucket-handle movement. (Reproduced from Beachey 1998.)

**Figure 1.12** Muscles of ventilation, including accessory muscles. (Reproduced from Beachey 1998.)
Figure 1.13 Branching of the conducting and terminal airways. Alveoli first appear in the respiratory bronchioles marking the beginning of the respiratory or gas exchange zone. BR bronchus, BL bronchiole, TBL terminal bronchiole, RBL respiratory bronchiole, AD alveolar duct, AS alveolar space, and Z order of airway division. (Reproduced from Beachey 1998.)

to recoil outwards. These movements, in opposite directions, are responsible for the development of negative pleural pressure when the respiratory system functions in the intact state’ (D’Alonzo & Krachman (1997)). In quiet breathing, at the end of exhalation, with the lungs partially inflated, an elastic recoil occurs which contracts and starts to empty the lungs. This passive elastic recoil which empties the lungs in quiet breathing should not involve any muscular activity. If more air is required than can be introduced by quiet breathing, accessory breathing muscles come into play. And with such deeper breathing, where muscular activity is used to overcome airway resistance, elastic recoil must also be overcome during inhalation. During exhalation from deeper breathing, the tendency for the thorax to increase its volume also has to be overcome by muscular effort.

If the accessory breathing muscles become shortened or fibrotic they negatively influence the efficiency of these processes.

LUNG VOLUMES AND CAPACITIES

- Total lung capacity (TLC) is the amount of air the lung can contain at the height of maximum inspiratory effort. All other lung volumes are natural subdivisions of TLC.
- Residual volume (RV) is the amount of air remaining within the lung after maximum exhalation. Inhaled at birth, it is not exhaled until death because the rib cage prevents total lung collapse. The volumes and capacities within these two limits are described in Figure 1.14.
- Lung volumes are measured in a variety of ways. From simple hand-held peak expiratory flow (PEF) meters used by patients with asthma to record air flow resistance to sophisticated laboratory equipment to establish both static and exercise lung capacities, volumes, and pressures, accurate information can be gained as to lung health or otherwise. For instance, vital capacity (VC) may be greatly reduced by limited expansion (restrictive disease) or by an abnormally large tidal volume (chronic obstructive airways disease or during asthma attacks). During vigorous exercise, tidal volume (Vt) may increase to half the VC to maintain adequate alveolar ventilation. Limitation of exercise capacity is often the first sign of early lung disease that limits VC (Berne & Levy 1998, p. 530).

Respiratory function (breathing) therefore demonstrably depends on the efficiency with which the structures constituting the pump mechanisms operate. At its simplest, for this pump to function optimally, the thoracic spine and the
attaching ribs, together with their anterior sternal connections and all the soft tissues, muscles, ligaments, tendons, and fascia, need to be structurally intact, with an uncompromised neural supply. Without an efficient pump mechanism all other respiratory functions will be suboptimal. Clearly, a host of dysfunctional patterns can result from altered airway characteristics, abnormal status of the lungs, and/or from emotional and other influences. However, even if allergy or infection is causal in altering the breathing pattern, the process of breathing can be enhanced by relatively unglamorous nuts-and-bolts features such as the normalization of rib restrictions or shortened upper fixator muscles (see Ch. 6 for further discussion).

The major structural components of the process of breathing are briefly outlined in the observations below. These notes discuss the fascia, the joints of the thoracic cage— including spinal and rib structures — and the musculature and other soft tissues of the region. Functional influences on these structures (e.g. gait and posture) are evaluated insofar as they impact on the efficiency of the respiratory process.

**FASCIA AND RESPIRATORY FUNCTION**

- Page (1952) describes the fascial linkage as follows:

  The cervical fascia extends from the base of the skull to the mediastinum and forms compartments enclosing oesophagus, trachea, carotid vessels and provides support for the pharynx, larynx and thyroid gland. There is direct continuity of fascia
THE STRUCTURE AND FUNCTION OF BREATHING

from the apex of the diaphragm to the base of the skull. Extending through the fibrous pericardium upward through the deep cervical fascia and the continuity extends not only to the outer surface of the sphenoid, occipital and temporal bones but proceeds further through the foramina in the base of the skull around the vessels and nerves to join the dura.

An obvious corollary to this vivid description of the continuity of the fascia is that distortion or stress affecting any one part of the structure will have repercussions on other parts of the same structure. For example, if the position of the cervical spine in relation to the thorax alters (as in a habitual forward-head position), or if the position of the diaphragm alters relative to its normal position (as in a slumped posture), the functional efficiency of the breathing mechanisms may be compromised.

- Rolfer Tom Myers (1997) has described what he terms the 'deep front line' - fascial connections linking the osseous and soft tissue structures which highlight clearly how modifications in posture involving spinal and/or other attachment structures will directly modify the fascia which envelops, supports, and gives coherence to the soft tissues of the breathing mechanism:
  - the anterior longitudinal ligament, diaphragm, pericardium, mediastinum, parietal pleura, fascia prevertebralis and the scalene fascia connect the lumbar spine (bodies and transverse processes) to the cervical transverse processes and via longus capitis to the basilar portion of the occiput
  - other links in this chain involve a connection between the posterior manubrium and the hyoid bone, via the subhyoid muscles and the fascia pretrachealis, between the hyoid and the cranium/mandible, involving the suprathyroid muscle as well as the muscles of the jaw linking the mandible to the face and cranium.
- Barral (1991) details additional fascial features of the respiratory mechanism, pointing out that there are five lung lobes (segments), three on the right and two on the left, wrapped in a membranous fascial structure, the pleura, which separates the lungs from the inner thoracic wall, and attaches to the thoracic structures superiorly at the hilum and inferiorly to the diaphragm. Barrell highlights the importance of this connection: 'The pleura is probably the structure most affected by the twenty-four thousand daily diaphragmatic movements, particularly in its superior attachments.' (Ideally, breathing rates are between 10 and 14 per minute; therefore, the normal 24-hour total would range from 14,000 to 20,000.) The suspensory attachment of the pleura (and pericardium), to the skeleton, is via a connective tissue dome comprising a variety of myofascial tissues and ligaments which attach to the spine and deep cervical aponeurosis close to the cervicothoracic junction. Barrell (1991) points out that while the mobile pleura require a point of stability, 'it is somewhat paradoxical that the cervical spine is much more mobile than the thorax, but at the same time serves as a superior fixed point for the pleural system.' Barrell observes that on dissection of degenerated lower cervical structures it is common to find associated excessive thickening and fibrotic change to the pleuropulmonary attachments: 'in view of the relationships between the pleural attachments and neurovascular system, it is easy to imagine the disorders which can arise in this strategic area, and their effects on nearby organs.'

In considering function and dysfunction of the respiratory system, fascial continuity should be kept in mind, since evidence of a local dysfunctional state (say of a particular muscle, spinal segment, rib, or group of ribs) can be seen to be capable of influencing (and being influenced by) distant parts of the same mechanism, as well as other areas of the body, via identifiable fascial connections.

THORACIC SPINE AND RIBS

The posterior aspect of the thorax is represented by a mobile functional unit, the thoracic spinal
column, through which the sympathetic nerve supply emerges:

- The degree of movement in all directions (flexion, extension, sideflexion, and rotation) allowed by the relatively rigid structure of the thorax is less than that available in the cervical or lumbar spines, being deliberately limited in order to protect the vital organs housed within the thoracic cavity.
- In most individuals the thoracic spine has a kyphotic (forward-bending) profile which varies in degree from individual to individual.
- The thoracic spinous processes are especially prominent, and therefore easily palpated.
- The transverse processes from T1 to T10 carry costotransverse joints for articulation with the ribs.

The thoracic facet joints, which glide on each other and restrict and largely determine the range of spinal movement, have typical plane-type synovial features, including an articular capsule.

**Facet orientation**

Hruby and colleagues (1997) describe a useful method for remembering the structure and orientation of the facet joints (of particular value when using mobilization methods, see Ch. 6):

The superior facets of each thoracic vertebrae are slightly convex and face posteriorly (backward), somewhat superiorly (up), and laterally. Their angle of declination averages $60^\circ$ relative to the transverse plane and $20^\circ$ relative to the coronal plane. Remember the facet facing by the mnemonic: ‘BUL’ (backward, upward, and lateral). This is in contrast to the cervical and lumbar regions where the superior facets face backwards, upwards, and medially (‘BUM’). Thus, the superior facets of the entire spine are BUM, BUL, BUM, from cervical, to thoracic to lumbar.

**Discs**

The disc structure of the thoracic spine is similar to that of the cervical and lumbar spine. The notable difference is the relative broadness of the posterior longitudinal ligament which, together with the restricted range of motion potential of the region, makes herniation of thoracic discs an infrequent occurrence. Degenerative changes due to osteoporosis and aging, as well as trauma, are relatively common in this region and may impact directly on respiratory function as a result of restricted mobility of the thoracic structures.

**Structural features of the ribs**

(see Fig. 1.9, above)

The ribs are composed of a segment of bone and a costal cartilage. The costal cartilages attach to the costochondral joint of most ribs (see variations below), depressions in the bony segment of the ribs.

Ribs 11 and 12 do not articulate with the sternum (‘floating ribs’), whereas all other ribs do so, in various ways, either by means of their own cartilaginous synovial joints (i.e. ribs 1–7, which are ‘true ribs’) or by means of a merged cartilaginous structure (ribs 8–10, which are ‘false ribs’).

The head of each rib articulates with its thoracic vertebrae at the costovertebral joint. Ribs 2–9 also articulate with the vertebrae above and below by means of a demifacet. Ribs 1, 11, and 12 articulate with their own vertebrae by means of a unifacet.

Typical ribs (3–9) comprise a head, neck, tubercle, angles, and shafts and connect directly, or via cartilaginous structures, to the sternum.

The posterior rib articulations allow rotation during breathing, while the anterior cartilaginous elements store the torsional energy produced by this rotation. The ribs behave like tension rods and elastically recoil to their previous position when the muscles relax. These elastic elements reduce with age and may also be lessened by intercostal muscular tension.

Rib articulations, thoracic vertebral positions, and myofascial elements must all be functional for normal breathing to occur. Dysfunctional elements may reduce the range of mobility, and, therefore, lung capacity.

**Atypical ribs**

Atypical ribs and their key features include:

- Rib 1, which is broad, short and flat, is the most curved. The subclavian artery and cervi-
cal plexus are anatomically vulnerable to compression if the 1st rib becomes compromised in relation to the anterior and/or middle scalenes, or the clavicle.

- Rib 2 carries a tubercle which attaches to the proximal portion of serratus anterior.
- Ribs 11 and 12 are atypical due to their failure to articulate anteriorly with the sternum or costal cartilages.

Rib dysfunction and appropriate treatments are discussed in Chapter 6.

**Intercostal musculature**

Stone (1999) amplifies the generally understood role of the intercostal muscles and their functions: ‘For many years the intercostals were attributed with a very complex biomechanical effect, such that the internal intercostals were considered expiratory muscles and the external intercostals inspiratory muscles’ (Kapandji 1974). Stone continues by explaining that the processes involved are far more complicated and that they relate to air and fluid movement within the thoracic cavity: ‘During inspiration and expiration there are cascades of action within the intercostal muscles, which start at one end of the rib cage and progress to the other to produce the required changes in rib cage shape.’

De Troyer & Estenne (1988) showed that during inhalation the external intercostals are activated from superior to inferior, while during forced exhalation the internal intercostals are activated from inferior to superior. The implication is that, on inhalation, stabilization of the upper ribs is required, involving scalenes (De Troyer 1994) to allow the sequential intercostal contraction wave to progress inferiorly. In contrast, during forced exhalation the lower ribs require stabilization – by quadratus lumborum – to allow the superiorly directed wave to occur. Muscular imbalances (shortness, weakness, etc.) could therefore impact on normal breathing function. (Assessment and treatment of muscular imbalances are discussed in Chapters 6 and 7.)

**Structural features of the sternum**

(see Fig. 1.9, above)

There are three key subdivisions of the sternum:

1. The manubrium (or head) which articulates with the clavicles at the sternoclavicular joints. The superior surface of the manubrium (jugular notch) lies directly anterior to the 2nd thoracic vertebra. The manubrium is joined to the body of the sternum by means of a fibrocartilaginous symphysis, the sternal angle (angle of Louis), which lies directly anterior to the 4th thoracic vertebra (Fig. 1.15).

2. The body of the sternum provides the attachment sites for the ribs, with the 2nd rib attaching at the sternal angle. This makes the angle an important landmark when counting ribs.

3. The xiphoid process is the ‘tail’ of the sternum, joining it at the xyphisternal symphysis (which fuses in most people during the fifth decade of life), usually anterior to the 9th thoracic vertebra.

**Posterior thorax**

In regional terms, the thoracic spine is usually divided into (White & Panjabi 1978): upper (T1-4), middle (T5-8), lower (T9-12):

- The total range of thoracic flexion and extension combined (between T1 and T12) is approximately 60° (Liebenson 1996)
- The total range of thoracic rotation is approximately 40°
- Total range of lateral flexion of the thoracic spine is approximately 50°.

**Palpation landmarks**

A useful way of identifying the thoracic vertebrae involves the so-called ‘rule of threes’. This ‘rule’ is simply an approximate generalization, but it positions the palpating fingers in the estimated positions for locating individual thoracic vertebrae (Hruby et al 1997):

- The spinous processes of T1-3 project directly posteriorly so that the tip of each spinous process is in the same plane as the transverse process of the same vertebra.
- The spinous processes of T4–6 project caudally so that the tip of each spinous process is in a plane that is approximately halfway between the transverse processes of its own vertebra and those of the vertebra immediately below.
- The spinous processes of T7–9 project more acutely caudally so that the tip of each spinous process is in the same plane as the transverse processes of the vertebra immediately below.
- T10 spinous process is similar to T7–9 (same plane as the transverse processes of the vertebra immediately below).
- T11 spinous process is similar to T4–6 (in a plane that is approximately halfway between the transverse processes of its own vertebra and those of the vertebra immediately below).
- T12 spinous process is similar to T1–3 (in the same plane as the transverse process of the same vertebra).

Box 1.2 describes some research into respiratory synkinesis and Box 1.3 describes the segmental coupling that takes place during compound spinal movements.

**NEURAL REGULATION OF BREATHING**

Respiratory centres in the most primitive part of the brain, the brainstem, unconsciously influence and adjust alveolar ventilation to maintain arterial blood oxygen and carbon dioxide pressures ($P_{CO_2}$) at relatively constant levels in order to sustain life under varying conditions and requirements.

There are three main groups:

1. The **dorsal respiratory group** is located in the distal portion of the medulla. It receives input
Biomechanical coupling of segments occurs during compound movements of the spine. During side-flexion an automatic rotation occurs due to the planes of the facets. In the thoracic spine this coupling process is less predictable than in the cervical region, where from C3 downwards type 2 coupling (also known as 'non-neutral') is the norm (i.e. side-bending and rotation occur to the same side).

Upper thoracic coupling is typically neutral/type 2 (i.e. side-bending and rotation take place towards the same side) and generally occurs as low as T4 ... [whereas] ... middle thoracic coupling is commonly a mix of neutral/type 1 and non-neutral/type 2 movements, that may rotate to either the formed convexity [type 1] or concavity [type 2]. Lower thoracic coupling is more apt to accompany lumbar neutral/type 1 mechanics.

Box 1.2 Respiratory synkinesis

Numerous adaptive combinations are possible in the thoracic spine, partly as a result of the compound influences and potentials of the muscles attaching to each segment. There seems to be a potential for compensatory patterning at all thoracic spinal levels. Lewit (1999) describes some research by Gaymans (1980) into respiratory synkinesis. This explains the apparent alternating inhibitory and mobilizing effects on spinal segments during inhalation and exhalation which appear to follow a predictable pattern in the cervical and thoracic spine during side-flexion. For example:

- On inhalation, resistance increases to side-flexion in the even segments (occiput–atlas, C2 etc. T2, T4, etc.) while in the odd segments there is a mobilizing effect (i.e. they are more free)
- On exhalation, resistance increases to side-flexion in the odd segments (C1, C3, etc., T3, T5, etc.) while in the even segments there is a mobilizing effect (i.e. they are more free).
- The area involving C7 and T1 seems 'neutral' and uninvolved in this phenomenon
- At the cervicocranial junction, the restrictive and mobilizing effects to inhalation and exhalation respectively seem to involve not just side-bending but all directions of motion
- The 'mobilizing influences' of inhalation as described above, diminish in the lower thoracic region.

The clinical value of this knowledge would be apparent during mobilization of segments in which side-flexion is a component of the restriction. In the thoracic region in particular there would be value in encouraging the appropriate phase of respiration when applying specific segmental mobilization techniques.

Box 1.3 Segmental coupling

Biomechanical coupling of segments occurs during compound movements of the spine. During side-flexion an automatic rotation occurs due to the planes of the facets. In the thoracic spine this coupling process is less predictable than in the cervical region, where from C3 downwards type 2 coupling (also known as 'non-neutral') is the norm (i.e. side-bending and rotation occur to the same side).

Hruby and colleagues (1997) state:

Upper thoracic coupling is typically neutral/type 2 (i.e. side-bending and rotation take place towards the same side) and generally occurs as low as T4 ... [whereas] ... middle thoracic coupling is commonly a mix of neutral/type 1 and non-neutral/type 2 movements, that may rotate to either the formed convexity [type 1] or concavity [type 2]. Lower thoracic coupling is more apt to accompany lumbar neutral/type 1 mechanics.

from peripheral chemoreceptors and other types of receptors via the vagus and glosso-pharyngeal nerves. These impulses generate inspiratory movements and are responsible for the basic rhythm of breathing.

2. The pneumotaxic centre in the superior part of the pons transmits inhibitory signals to the dorsal respiratory centre, controlling the filling phase of breathing.

3. The ventral respiratory group, located in the medulla, causes either inspiration or expiration. It is inactive in quiet breathing but is important in stimulating abdominal expiratory muscles during levels of high respiratory demand.

The Hering-Breuer reflex prevents overinflation of the lungs and is initiated by nerve receptors in the walls of the bronchi and bronchioles sending messages to the dorsal respiratory centre, via the vagus nerves. It 'switches off' excessive inflation during inspiration, and also excessive deflation during exhalation.

Chemical control of breathing

The central role of respiration is to maintain balanced concentrations of oxygen (O₂) and carbon dioxide (CO₂) in the tissues. Increased levels of CO₂ act on the central chemosensitive areas of the respiratory centres themselves, increasing inspiratory and expiratory signals to the respiratory muscles. O₂, on the other hand, acts on peripheral chemoreceptors located in the carotid body (in the bifurcation of the common carotid arteries) via the glossopharyngeal nerves, and the aortic body (on the aortic arch) which sends the appropriate messages via the vagus nerves to the dorsal respiratory centre.

Voluntary control of breathing

Automatic breathing can be overridden by higher cortical conscious input (directly, via the spinal neurons which drive the respiratory muscles) in response to, for instance, fear or sudden surprise. Speaking requires voluntary control to interrupt the normal rhythmicity of breathing, as does singing and playing a wind instrument. There is evidence that the cerebral cortex and thalamus also supply part of the drive
for normal respiratory rhythm during wakefulness (cerebral influences on the medullary centres are withdrawn during sleep). Breathing pattern disorders (BPDs) and hyperventilation syndromes (HVSs) probably originate from some of these higher centres (Timmons 1994, p. 35).

**THE AUTONOMIC NERVOUS SYSTEM**

(Table 1.1)

This system enables the automatic unconscious maintenance of the internal environment of the body in ideal efficiency, and adjusts to the various demands of the external environment, be it sleep with repair and growth, quiet, or extreme physical activity and stress. The nerves innervate the smooth muscle of the alimentary canal causing propulsion of food, while the nerves to exocrine glands initiate secretion of digestive juices. The system is also concerned with the emptying of the bladder and with sexual activity.

Innervation of smooth muscle in the walls of the arterioles varies their caliber, permitting the maintenance of blood pressure and the switching up and down of various parts of the circulation according to whether digestion, growth and repair, heat conservation or loss, or strong muscular activity are required. The maintenance of an adequate circulation also depends on the heart rate, the strength of the cardiac muscle contraction thereby varying the cardiac output.

The hypothalamus in the brainstem has a key role in coordinating the diverse functions of the autonomic nervous system, as well as controlling the release of hormones from the pituitary gland, to which it is connected by a stalk carrying the releasing factors. Some pituitary hormones directly control the biochemical processes of the body. On the other hand, the pituitary trophic hor-

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**Table 1.1 Comparison of the autonomic and somatic motor nervous systems (from Beachey 1998)**

<table>
<thead>
<tr>
<th>Features</th>
<th>Somatic motor nervous system</th>
<th>Autonomic nervous system</th>
</tr>
</thead>
<tbody>
<tr>
<td>Target tissues</td>
<td>Skeletal muscle</td>
<td>Smooth muscle, cardiac muscle, and glands</td>
</tr>
<tr>
<td>Regulation</td>
<td>Control of all conscious and unconscious movements of skeletal muscle</td>
<td>Unconscious regulation, although influenced by conscious mental functions</td>
</tr>
<tr>
<td>Response to stimulation</td>
<td>Skeletal muscle contracts</td>
<td>Target tissues are stimulated or inhibited</td>
</tr>
<tr>
<td>Neuron arrangement</td>
<td>One neuron extends from the central nervous system (CNS) to skeletal muscle</td>
<td>Two neurons in series; the preganglionic neuron extends from the CNS to an autonomic ganglion, and the postganglionic neuron extends from the autonomic ganglion to the target tissue</td>
</tr>
<tr>
<td>Neuron cell body location</td>
<td>Neuron cell bodies are in motor nuclei of the cranial nerves and in the ventral horn of the spinal cord</td>
<td>Preganglionic neuron cell bodies are in autonomic nuclei of the cranial nerves and in the lateral horn of the spinal cord; postganglionic neuron cell bodies are in autonomic ganglia</td>
</tr>
<tr>
<td>Number of synapses</td>
<td>One synapse between the somatic motor neuron and the skeletal muscle</td>
<td>Two synapses; first in the autonomic ganglia, second at the target tissue</td>
</tr>
<tr>
<td>Axon sheaths</td>
<td>Myelinated</td>
<td>Preganglionic axons myelinated, postganglionic axons unmyelinated</td>
</tr>
<tr>
<td>Neurotransmitter substance</td>
<td>Acetylcholine</td>
<td>Acetylcholine released by preganglionic neurons; either acetylcholine or norepinephrine released by postganglionic neurons</td>
</tr>
<tr>
<td>Receptor molecules</td>
<td>Receptor molecules for acetylcholine are nicotinic</td>
<td>In autonomic ganglia, receptor molecules for acetylcholine are nicotinic; in target tissues, receptor molecules for acetylcholine are muscarinic, whereas receptor molecules for norepinephrine are either α- or β-adrenergic</td>
</tr>
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mones control the activity of target endocrine glands, such as the thyroid and adrenals, in secret-
ing their own hormones, which in turn control other metabolic processes. The hypothalamus has connections with the preganglionic neurons further down the brainstem and the spinal cord.

The system has a set of receptors from which afferent nerve fibers carry impulses to the central nervous system for integration and action. This action is mediated by impulses transmitted down a set of efferent nerves to effector organs which are mainly smooth muscles and glands. Besides the afferent and efferent pathways, there are two divisions:

- The sympathetic, mainly concerned with preparing the body for, and handling stressful situations, adapting to the needs of the external environment
- The parasympathetic, serving visceral functions such as digestion, absorption, and growth (see Tables 1.2 and 1.3).

**Sympathetic division** (Tables 1.2 and 1.3; Figs. 1.16, 1.17)

- The sympathetic outflow from preganglionic neurons in the spinal cord is carried by way of their axons through the ventral routes of the first thoracic, all the way down to the third or fourth lumbar spinal nerves, some 15-16 pairs. They make up the white rami communicantes to the paravertebral sympathetic ganglionic chain, where they synapse with postganglionic neurons. These in turn send fibers along the arteries to autonomic effectors, be they glands or smooth muscle.
- The sympathetic ganglion chain has at its upper end the superior, middle, and stellate ganglia, housing the postganglionic neurons which supply effectors in the head, neck, and heart.
- The outflow from thoracic segments 1–4 supplies the larynx, trachea, bronchi, and lungs, while thoracic segments 5–12 fire the greater and small splanchnic nerves, enter the celiac and superior mesenteric ganglia. The inferior mesenteric ganglion is supplied by lumbar segments 1–3, 4. Nerve fibers from these three ganglia supply the organs in the abdomen and the genitalia.

**Parasympathetic division** (Tables 1.2 and 1.3; Fig. 1.16)

- The parasympathetic outflow occurs in two regions: cranial with the preganglionic axons traveling in the oculomotor, facial, glosso-pharyngeal, and vagus nerves to ganglia situated close to the effector mechanisms; the sacral outflow supplies the pelvic organs through the second to fourth sacral nerves coming together as the pelvic nerve.
- The lungs are entirely governed by autonomic sensory and motor nerves: there is no voluntary motor control over airway smooth muscles.

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<thead>
<tr>
<th>Feature</th>
<th>Sympathetic division</th>
<th>Parasympathetic division</th>
</tr>
</thead>
<tbody>
<tr>
<td>Location of preganglionic cell body</td>
<td>Lateral horns of spinal cord gray matter (T1–L2)</td>
<td>Brainstem and lateral horns of spinal cord gray matter (S2–S4)</td>
</tr>
<tr>
<td>Outflow from central nervous system</td>
<td>Spinal nerves</td>
<td>Cranial nerves</td>
</tr>
<tr>
<td></td>
<td>Sympathetic nerves</td>
<td>Pelvic nerves</td>
</tr>
<tr>
<td></td>
<td>Splanchnic nerves</td>
<td></td>
</tr>
<tr>
<td>Ganglia</td>
<td>Sympathetic chain ganglia along spinal cord for spinal and sympathetic nerves; collateral ganglia for splanchnic nerves</td>
<td>Terminal ganglia near or on effector organ</td>
</tr>
<tr>
<td>Number of postganglionic neurons for each preganglionic neuron</td>
<td>Many</td>
<td>Few</td>
</tr>
<tr>
<td>Relative length of neurons</td>
<td>Short preganglionic</td>
<td>Long preganglionic</td>
</tr>
<tr>
<td></td>
<td>Long postganglionic</td>
<td>Short postganglionic</td>
</tr>
</tbody>
</table>
Table 1.3 Comparison of the sympathetic and parasympathetic divisions (from Beachey 1998)

<table>
<thead>
<tr>
<th>Organ</th>
<th>Effect of sympathetic stimulation</th>
<th>Effect of parasympathetic stimulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Muscle</td>
<td>Increased rate and force (b)</td>
<td>Slowed rate (c)</td>
</tr>
<tr>
<td>Coronary arteries</td>
<td>Dilated (b), constricted (a)*</td>
<td>Dilated (c)</td>
</tr>
<tr>
<td>Systemic blood vessels</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abdominal</td>
<td>Constricted (a)</td>
<td>None</td>
</tr>
<tr>
<td>Skin</td>
<td>Constricted (a)</td>
<td>None</td>
</tr>
<tr>
<td>Muscle</td>
<td>Dilated (b), constricted (a)</td>
<td>None</td>
</tr>
<tr>
<td>Lungs</td>
<td>Dilated (b)</td>
<td>Constricted (c)</td>
</tr>
<tr>
<td>Bronchi</td>
<td>Glucose released into blood (b)</td>
<td>None</td>
</tr>
<tr>
<td>Liver</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skeletal muscles</td>
<td>Breakdown of glycogen to glucose (b)</td>
<td>None</td>
</tr>
<tr>
<td>Metabolism</td>
<td>Increased up to 100% (a, b)</td>
<td>None</td>
</tr>
<tr>
<td>Glands</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adrenal</td>
<td>Release of epinephrine and norepinephrine</td>
<td>None</td>
</tr>
<tr>
<td>Salivary</td>
<td>Constriction of blood vessels and slight production of a thick, viscous secretion (a)</td>
<td>Dilation of blood vessels and thin, copious secretion (c)</td>
</tr>
<tr>
<td>Gastric</td>
<td>Inhibition (a)</td>
<td>Stimulation (c)</td>
</tr>
<tr>
<td>Pancreas</td>
<td>Decreased insulin secretion (a)</td>
<td>Increased insulin secretion (c)</td>
</tr>
<tr>
<td>Lacrima</td>
<td>None</td>
<td>Secretion (c)</td>
</tr>
<tr>
<td>Sweat</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Merocrine</td>
<td>Copious, watery secretion (c)</td>
<td>None</td>
</tr>
<tr>
<td>Apocrine</td>
<td>Thick, organic secretion (c)</td>
<td>None</td>
</tr>
<tr>
<td>Gut</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wall</td>
<td>Decreased tone (b)</td>
<td>Increased motility (c)</td>
</tr>
<tr>
<td>Sphincter</td>
<td>Increased tone (a)</td>
<td>Decreased tone (c)</td>
</tr>
<tr>
<td>Gallbladder and bile ducts</td>
<td>Relaxed (b)</td>
<td>Contracted (c)</td>
</tr>
<tr>
<td>Urinary bladder</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wall</td>
<td>Relaxed (b)</td>
<td>Contracted (c)</td>
</tr>
<tr>
<td>Sphincter</td>
<td>Contracted (a)</td>
<td>Relaxed (c)</td>
</tr>
<tr>
<td>Eye</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ciliary muscle</td>
<td>Relaxed for far vision (b)</td>
<td>Contracted for near vision (c)</td>
</tr>
<tr>
<td>Pupil</td>
<td>Dilated (a)</td>
<td>Constricted (c)</td>
</tr>
<tr>
<td>Erector pili muscles</td>
<td>Contraction (a)</td>
<td>None</td>
</tr>
<tr>
<td>Blood</td>
<td>Increased coagulation (a)</td>
<td>None</td>
</tr>
<tr>
<td>Sex organs</td>
<td>Ejaculation (a)</td>
<td>Erection (c)</td>
</tr>
</tbody>
</table>

\[\text{a} \text{ Mediated by } \alpha\text{-adrenergic receptors; } \beta, \text{ mediated by } \beta\text{-adrenergic receptors; } \gamma, \text{ mediated by cholinergic receptors.}
\]

\[\text{*Sympathetic stimulation of the heart normally increases coronary artery blood flow because of increased cardiac muscle demand for oxygen.}\]

- A further subdivision of the autonomic nervous system is on the basis of the chemical transmission across the synapses.

**NANC system**

Researches have recognized a 'third' nervous system regulating the airways called the non-adrenergic noncholinergic (NANC) system, containing inhibitory and stimulatory fibers; nitric oxide (NO) has been identified as the NANC neurotransmitter (Snyder 1992).

- NANC inhibitory nerves cause calcium ions to enter the neuron, mediating smooth muscle relaxation and bronchodilation.
- NANC stimulatory fibers – also called C-fibers – are found in the lung supporting tissue, airways, and pulmonary blood vessels, and appear to be involved in bronchoconstriction following cold air breathing and in exercise-induced asthma (Beachey 1998, p. 30).

Box 1.4 gives an example of autonomic responses following stress/distress.
Figure 1.16 Schema of the autonomic innervation (motor and sensory) of the lung and the somatic (motor) nerve supply to the intercostal muscles and diaphragm. (Reproduced from Scanlan et al 1995.)

Box 1.4 An example of autonomic responses

A patient has become alarmed at the sight of blood and the pain from a wound. The immediate reaction to this stress is a strong vagal response, slowing the heart rate. As blood flow slows down in the vertebral arteries supplying the brainstem, a feeling of nausea and faintness supervenes. Pressor receptors in the carotid arteries sense the fall in blood pressure and its threat to the cerebral circulation. From the receptors, impulses travel up the afferents in the glossopharyngeal nerves to the cardiorespiratory center in the medulla. This in turn sends a cascade of efferent impulses down the spinal cord and out along the thoracic spinal nerves. The sympathetic and adrenergic outflow speeds up the heart and constricts the blood vessels of the body. In this way, normal blood pressure is restored and blood flow to the brain and brainstem increases, thereby preventing a faint. Moreover, there is a discharge in the adrenal medulla releasing epinephrine and norepinephrine which cause a further increase in heart rate and narrowing of the arterioles. These receptors allow the development and use of drugs that either stimulate certain receptors (agonists) or block some receptors (antagonists or blockers). For example, beta-blockers are used in the control of hypertension, favoring the dilation of arterioles and slowing of the heart. A number of beta agonists are useful in asthma, relaxing the bronchospasm of the bronchial musculature set up by allergic responses or infection.
THE MUSCLES OF RESPIRATION

The extrinsic thoracic musculature is responsible for positioning the torso, and therefore, also the placement in space of the shoulders, arms, neck, and head. The intrinsic thoracic muscles move the thoracic vertebrae or the rib cage (and possibly the entire upper body) and/or are associated with respiration.
The deeper elements of the thoracic musculature represent a remarkable system by means of which respiration occurs. Some of these muscles also provide rotational components which carry similar, spiraling, lines of oblique tension from the pelvis (external and internal obliques) through the entire torso (external and internal intercostals), almost as if the ribs were ‘slipped into’ this supportive web of continuous muscular tubes. Rolfer Tom Myers (1997) describes the physical continuity which occurs between these muscles (obliques and intercostals). Above the pelvic crest this myofascial network creates a series of crossover (X-shaped) patterns:

The obliques tuck into the lower edges of the basket of ribs. Between each of the ribs are the internal and external intercostals, which taken all together form a continuation of the same ‘X’, formed by the obliques. These muscles, commonly taken to be accessory muscles of breathing, are seen in this context to be perhaps more involved in locomotion [and stability], helping to guide and check the torque, swinging through the rib cage during walking and running. (Myers 1997)

Richardson and colleagues (1999) describe research into the behaviour of the abdominal muscles during quiet breathing. They found that the abdominal musculature was activated towards the end of exhalation, and noted that ‘Contraction of the abdominal muscles contributes towards the regulation of the length of the diaphragm, end-expiratory lung volume and expiratory airflow’.

With a voluntary increase in exhalation force, all the muscles of the abdomen contract simultaneously. However, when increased exhalation force occurs involuntarily, transversus abdominis is recruited before the other abdominal muscles (rectus abdominis, obliquus externus abdominis), producing enhancement of inspiratory efficiency by increasing the diaphragm’s length and permitting an elastic recoil of the thoracic cavity.

Like the erector system of the posterior thorax, the abdominal musculature plays a significant role in positioning the thorax and in rotating the entire upper body. It is also now known to play a key part in spinal stabilization and intersegmental stability, particularly transversus abdominis (Hodges 1999). The rectus abdominis, external and internal obliques and transversus abdominis are also involved in respiration due to their role in positioning the abdominal viscera as well as depression of the lower ribs, assisting in forced expiration (especially coughing).

Additional soft tissue influences and connections

- The soft tissue links between the thoracic region and the pelvic region include major structures such as quadratus lumborum, transversus abdominis, and psoas, which merge with the diaphragm and therefore have the potential to influence breathing function.
- The internal and external obliques (usually described as trunk rotators) also merge with the diaphragm and lower ribs and can have marked influences on respiratory function. It is worth reflecting that this works in reverse, and that diaphragmatic and respiratory dysfunction is bound to affect these associated muscles.
- The primary inspiratory muscles are the diaphragm, the more lateral external intercostals, parasternal internal intercostals, scalene group, and levatores costarum, with the diaphragm providing 70–80% of the inhalation force (Simons et al 1998).
- These muscles are supported, or their role is replaced, by the accessory muscles during increased demand (or dysfunctional breathing patterns): sternocleidomastoid (SCM), upper trapezius, pectoralis major and minor, serratus anterior, latissimus dorsi, serratus posterior superior, iliocostalis thoracis, subclavius, and omohyoid (Kapandji 1974, Simons et al 1998).

INSPIRATORY AND EXPIRATORY MUSCLES (Box 1.5)

The muscles associated with breathing function can be grouped as either inspiratory or expiratory, and are either primary in that capacity or provide accessory support. It should be kept in mind that
the role that these muscles might play in inhibiting respiratory function (due to trigger points, ischemia, etc.) has not yet been clearly established, and that their overload, due to dysfunctional breathing patterns, is likely to impact on cervical, shoulder, lower back, and other body regions.

Box 1.5 lists the muscles of breathing, both inspiratory and expiratory.

Since expiration is primarily an elastic response of the lungs, pleura, and ‘torsion rod’ elements of the ribs, all muscles of expiration could be considered to be accessory muscles as they are recruited only during increased demand. They include internal intercostals, abdominal muscles transversus thoracis, and subcostales. With increased demand, iliocostalis lumborum, quadratus lumborum, serratus posterior inferior, and latissimus dorsi may support expiration, including during the high demands of speech, coughing, sneezing, singing, and other special functions associated with the breath. In addition:

- The intercostal muscles, while participating in inhalation (external intercostals) and exhalation (internal intercostals), are also responsible for enhancing the stability of the chest wall, so preventing its inward movement during inspiration.
- Quadratus lumborum (QL) acts to fix the 12th rib, so offering a firm attachment for the diaphragm. If QL is weak, as it may be in certain individuals, this stability is lost (Norris 1999).
- Bronchial obstruction, pleural inflammation, liver or intestinal encroachment and ensuing pressure against the diaphragm, as well as phrenic nerve paralysis are some of the pathologies which will interfere with diaphragmatic and respiratory efficiency.

Box 1.6 describes some muscle characterization models and the dual roles of some specific muscles.

**GAIT INFLUENCES**

The major postural muscles of the body (anterior and posterior aspects) are shown in Figure 1.18A and B. Gait involves the spine in general, and the thoracic spinal muscles in particular, and by inference can impact on respiratory function. Grakovetsky (1997) reports: ‘In walking, the hip extensors fire as the toe pushes the ground. The muscle power is directly transmitted to the spine and trunk via two distinct but complementary pathways’:

- Biceps femoris has its gait action extended by the sacrotuberosous ligament, which crosses the superior iliac crest and continues upward as the erector spinae aponeurosis, iliocostalis lumborum, and iliocostalis thoracis (among others), and linking directly with the contralateral latissimus dorsi.
- Gluteus maximus force is transmitted superiorly via the lumbodorsal fascia and latissimus dorsi

In this way, an oblique muscle–tendon–fascial sling is created across the torso, providing a
Richardson and colleagues (1999) have categorized muscles capable of controlling one joint or one area of the spine as being ‘monoarticular’. These could also be referred to as ‘local’ muscles. They also describe ‘multijoint’ muscles which are capable of moving several joints at the same time. These muscles are also phylogenetically the oldest. They can be referred to as ‘global’ muscles. This nomenclature allows clinical disciplines to communicate with other basic science disciplines in order to facilitate research and learning.

Other muscle categorization models include ‘postural/dual roles of specific muscles
Research shows that many of the muscles supporting and moving the thorax and/or the spinal segments (including erector spinae) prepare to accommodate for subsequent movement as soon as arm or shoulder activity is initiated, with deep stabilizing activity from transversus abdominis, for example, occurring miniseconds before unilateral rapid arm activity (Hodges & Richardson 1997). Stabilization of the lumbar spine and thorax has been shown to depend, to a large extent, on abdominal muscle activity (Hodges 1999). Transversus abdominis is categorized as a local, stabilizing structure. Richardson and colleagues (1999) note that the direct involvement of this muscle in respiration – where it contributes to forced exhalation – leads to a potential conflict with its role as a spinal stabilizer. They have identified a clear linkage between dysfunction of transversus abdominis and low back pain.

mechanism for energy storage, to be utilized in the next phase of the gait cycle. As Lee (1997) points out: ‘Together, these two muscles [gluteus maximus and latissimus dorsi] tense the thoracodorsal fascia and facilitate the force closure mechanism through the sacro-iliac joint.’

Gracovetsky (1997) continues: ‘As a consequence, firing hip extensors extends and raises the trunk in the sagittal plane. The chemical energy liberated within the muscles is now converted by the rising trunk, into potential energy stored in the gravitational field. When a person is running, so much energy needs to be stored that the necessary rise in the centre of gravity forces the runner to become airborne.’

The intrinsic thoracic muscles are largely responsible for movement of the thoracic spinal column or cage, as well as respiratory function. Though many of these muscles have very short fibers, and therefore may appear relatively unimportant, they are strategically placed to provide, or initiate, precisely directed movement of the thoracic vertebrae and/or ribs. They therefore demand due attention in evaluation of restrictions within these structures.

UPPER THORACIC MUSCLES
(see Fig. 1.18A and B)

When viewing the posterior thorax, the trapezius is immediately obvious as it lies superficially and extensively covers the upper back, shoulder and neck. In addition to trapezius, latissimus dorsi, which superficially covers the lower back, as well
as the rhomboids, serratus anterior, and pectoralis major and minor, are of possible clinical significance in breathing imbalance. A complex array of short and long extensors and rotators lies deep to the more superficial trapezius, latissimus dorsi, and rhomboids. Platzer (1992) breaks these two groups into lateral (superficial) and medial (deep) tracts, each having a vertical (intertransverse) and diagonal (transversospinal) component. This subdivision offers a useful mode when assessing rotational dysfunctions, as the superficial rotators are synergistic with the contralateral deep rotators.

**Thoracic musculature**

- Iliocostalis lumborum extends from the iliac crest, sacrum, thoracolumbar fascia, and spinous processes of T11–L5 to attach to the inferior borders of the angles of the lower 6–9 ribs.
- Iliocostalis thoracis fibers run from the superior borders of the lower 6 ribs to the upper 6 ribs and the transverse process of C7.
- Longissimus thoracis shares a broad thick tendon with iliocostalis lumborum, and fiber attachments to the transverse and accessory processes of the lumbar vertebrae and thoracolumbar fascia, which then attaches to the tips of the transverse processes and between the tubercles and angles of the lower 9–10 ribs.
- The thoracic component of the erector spinae system has numerous attachments onto the ribs.
- Trigger points in these vertical muscular columns refer caudally and cranially across the thorax and lumbar regions, into the gluteal region, and anteriorly into the chest and abdomen.
NOTES ON SPECIFIC MUSCLES

Spinalis thoracis
Attachments. Spinous processes of T11–L2 to the spinous processes of T4–T8 (variable).
Innervation. Dorsal rami of spinal nerves.
Function. Acting unilaterally, flexes the spine laterally; bilaterally, extends the spine.
Synergists. For lateral flexion: ipsilateral semispinalis, longissimus and iliocostalis thoracis, iliocostalis lumborum, quadratus lumborum, obliques, and psoas.
Antagonists. To lateral flexion: contralateral semispinalis, longissimus and iliocostalis thoracis, iliocostalis lumborum, quadratus lumborum, obliques, and psoas.

Semispinalis thoracis
Innervation. Dorsal rami of thoracic nerves.
Function. Acting unilaterally, it rotates the spine contralaterally; bilaterally, it extends the spine.
Synergists. For rotation: multifidi, rotatores, ipsilateral external obliques and external intercostal, and contralateral internal obliques and internal intercostals. For extension: posterior spinal muscles (precise muscles depending upon what level is being extended).
Antagonists. To rotation: matching contralateral fibers of semispinalis as well as contralateral multifidi, semispinalis, external obliques, and external intercostals, and the ipsilateral internal oblique and internal intercostal. For extension: spinal flexors (precise muscles depending upon what level is being extended).
Possible signs of dysfunction of semispinalis.
Possible impact on breathing function due to association with intercostal musculature
Chronic instability of associated vertebral segments
Reduced flexion of spine
Restricted rotation (sometimes painfully)
Pain along spine
Vertebral scapular border pain (referral zone).

Multifidi
Attachments. From the posterior surface of the sacrum, iliac crest, and the transverse processes of all lumbar, thoracic vertebrae and articular processes of cervicals 4-7, these muscles traverse 2-4 vertebrae and attach superiorly to the spinous processes of all vertebrae apart from the atlas.
Innervation. Dorsal rami of spinal nerves.
Function. When these contract unilaterally they produce ipsilateral flexion and contralateral rotation; bilaterally, they extend the spine.
Synergists. For rotation: multifidi, semispinalis muscles, ipsilateral external obliques and external intercostal, and contralateral internal obliques and internal intercostals. For extension: posterior spinal muscles (precise muscles depending upon what level is being extended).
Antagonists. To rotation: matching contralateral fibers of rotators as well as contralateral multifidi, semispinalis, external obliques, and external intercostals, and the ipsilateral internal oblique and internal intercostal. For extension: spinal flexors (precise muscles depending upon what level is being extended).
Possible signs of dysfunction of multifidi.
Possible impact on breathing function due to association with intercostal musculature
Chronic instability of associated vertebral segments
Reduced flexion of spine
Restricted rotation (sometimes painfully)
Pain along spine
Vertebral scapular border pain (referral zone).

Rotatores longus and brevis
Attachments. From the transverse processes of each vertebra to the spinous processes of the second (longus) and first (brevis) vertebra above (ending at C2).
Innervation. Dorsal rami of spinal nerves.
Function. When these contract unilaterally they produce contralateral rotation; bilaterally, they extend the spine.
Synergists. For rotation: multifidi, semispinalis muscles, ipsilateral external obliques and
external intercostal, and contralateral internal obliques and internal intercostals. For extension: posterior spinal muscles (precise muscles depending upon what level is being extended).

**Antagonists.** To rotation: matching contralateral fibers of rotatores as well as contralateral multifidi, semispinalis, external obliques, and external intercostals, and the ipsilateral internal oblique and internal intercostal. For extension: spinal flexors (precise muscles depending upon what level is being extended).

**Possible signs of dysfunction of rotatores.**
- Pain and tenderness as associated vertebral segments, tenderness to pressure or tapping applied to the spinous processes of associated vertebrae
- Possible impact on breathing function due to association with intercostal musculature.

**Spinal musculature: implications for thoracic function**

Multifidi and rotatores muscles comprise the deepest layer and are responsible for fine control of the rotation of vertebrae. They exist through the entire length of the spinal column, and the multifidi also broadly attach to the sacrum after becoming appreciably thicker in the lumbar region.

These muscles are often associated with vertebral segments which are difficult to stabilize and should be addressed throughout the spine when scoliosis is presented along with the associated intercostal muscles and pelvic positioning.

Trigger points in rotatores tend to produce rather localized referrals, whereas those in the multifidi refer locally and also to the suboccipital region, medial scapular border, and top of shoulder. These local (for both) and distant (for multifidi) patterns of referral continue to be expressed through the length of the spinal column. In fact, the lower spinal levels of multifidi may even refer to the anterior thorax or abdomen.

**Multifidus and the abdominals**

Multifidus should co-contract with transversus abdominis to assist in low back stabilization (Richardson & Jull 1995) which suggests that any chronic weakness (or atrophy) of multifidus is likely to impact strongly on spinal stability – and potentially on breathing function. While shortness and tightness of a muscle are obvious indicators of dysfunction, it is also important when considering muscular imbalances to evaluate for weakness. Actual atrophy of the multifidi has been reported in a variety of low back pain settings. As Liebenson (1996) observes: The initial muscular reaction to pain and injury has traditionally been assumed to be an increased tension and stiffness. Data indicates inhibition is at least as significant. Tissue immobilisation occurs secondarily, which leads to joint stiffness and disuse muscle atrophy.

**Serratus posterior superior**

**Attachments.** Spinal processes of C7–T3 attach to the upper borders and external surfaces of ribs 2–5, lateral to their angles.

**Innervation.** Intercostal nerves (T2–5).

**Function.** Uncertain role but most likely elevates the ribs (Gray 1995).

**Synergists.** Diaphragm, levatores costarum brevis, scalenus posterior.

**Antagonists.** Internal intercostals.

**Possible signs of dysfunction of serratus posterior superior.**
- Pain that seems to be deep to the scapula
- Pain may radiate over the posterior deltoid, down the back of the arm and the ulnar portion of the hand, and to the smallest finger
- Numbness into the ulnar portion of the hand
- Possible impact on breathing function via association with diaphragm and ribs.

**Serratus posterior inferior**

**Attachments.** Spinal processes of T11–L3 and the thoracolumbar fascia to the inferior borders of the lower four ribs.

**Innervation.** Intercostal nerves (T9–12).

**Function.** Depresses lower four ribs and pulls them posteriorly, not necessarily in respiration (Gray 1989).

**Synergists.** Internal intercostals.

**Antagonists.** Diaphragm.
Possible signs of dysfunction in serratus posterior inferior.
Leg length differential
Rib dysfunction in lower four ribs
Lower backache in area of the muscle
Scoliosis.

**Levatores costarum longus and brevis** (Fig 1.19)

**Attachments.** *Longus:* tips of transverse processes of T7–T10 to the upper edge and external surface of the tubercle and angle of the second rib below. *Brevis:* tips of transverse processes of C7–T11 to the upper edge and external surface of the tubercle and angle of the next rib below.

**Innervation.** Dorsal rami of thoracic spinal nerves.

**Function.** Elevate the ribs: contralateral spinal rotation, ipsilateral flexion, and bilaterally extends the column.

**Synergists.** For rib elevation: serratus posterior superior, external intercostals, diaphragm, scalenes.

Antagonists. Internal intercostals, serratus posterior inferior, elastic elements of thorax.

Possible signs of dysfunction of levatores costarum.
Rib dysfunction
Breathing dysfunctions, especially ribs locked in elevation
Vertebral segmental facilitation
Scoliosis.

**Special notes on levatores costarum.** The levatores costarum appear innocuous in their small, short passage from the transverse process to the exterior aspect of the ribs. However, this advantageous placement, directly over the costovertebral joint, places them in a powerful position to rotate the ribs during inhalation. Simons and colleagues (1998) state: 'They elevate the rib cage with effective leverage. A small upward movement of the ribs so close to the vertebral column is greatly magnified at the sternum.'

**Intercostals** (Fig. 1.12)

**Attachments.** External, internal, and innermost lie in three layers, with the external outermost and attach the inferior border of one rib to the superior border of the rib below it. (See special notes below for direction of fibers.)

**Innervation.** Corresponding intercostal nerves.


Possible signs of dysfunction in intercostals.
Respiratory dysfunctions, including dysfunctional breathing patterns and asthma
Scoliosis
Rib dysfunctions and intercostal pain
Cardiac arrhythmia.

Special notes on intercostals. Whereas the internal intercostal muscles attach to the ribs and fully to the costal cartilages, the external intercostals attach only to the ribs, ending at the lateral edge of the costal cartilages with the external intercostal membrane expanding the remaining few inches to the sternum.

The external and internal intercostal fibers lie in opposite directions to each other with the external fibers angling inferomedially and the internal fibers coursing inferolaterally when viewed from the front. These fiber directions coincide with the direction of external and internal obliques and provide rotatorial movement of the torso and postural influences in addition to respiratory responsibilities (Simons et al 1998).

The role these muscles play in quiet breathing is uncertain, with some texts suggesting involvement only during forced respiration (Platzer 1992). Simons and colleagues (1998) discuss progressive recruitment depending upon degree of forced respiration. Intercostals may also provide rigidity to the thoracic cage to prevent inward pull of the ribs during inspiration.

The subcostalis muscles (when present) are usually only well developed in the lower internal thoracic region. Their fiber direction is the same as that of internal and innermost intercostals and they span across the internal surface of one or two ribs rather than just the intercostal space. They probably have a similar function to the deeper intercostal muscles (Gray 1995, Platzer 1992, Simons et al 1998).

Interior thorax

Diaphragm

Attachments. Inner surfaces of lower 6 ribs and their costal cartilages, posterior surface of xiphoid process (or sternum), and the body of the upper 1–4 lumbar vertebrae, vertebral discs and the arcuate ligaments, thereby forming a circular attachment around the entire inner surface of the thorax.


Function. Principal muscle of inspiration by drawing its central tendon downward to stabilize it against the abdominal viscera, at which time it lifts and spreads the lower ribs.

Synergists. Accessory muscles of inhalation.

Antagonists. Elastic recoil of thoracic cavity and accessory muscles of exhalation

Possible signs of dysfunction in the diaphragm.
Dyspnea or any breathing difficulty (after ruling out more sinister causes)
Dysfunctional breathing patterns
Chronic respiratory problems (asthma, chronic cough, etc.)
'Stitch' in the side on exertion
Chest pain
Hiccup
External compression from tight-waisted clothing.

Special notes on the diaphragm

The diaphragm is a dome-shaped muscle with a central tendon whose fibers radiate peripherally to attach to all the margin of the lower thorax, thereby forming the floor of the thoracic cavity. It attaches higher in the front than in the side or the back. When this muscle contracts, it increases vertical, transverse, and anteroposterior diameter of the internal thorax (Kapandji 1974) and is therefore the most important muscle in inspiration.

A brief summary of some of the diaphragm’s key attachments and features indicates the complex nature of this muscle:

- The sternal part of the diaphragm arises from the internal surface of the xiphoid process (this attachment is sometimes absent)
- The costal part arises from the internal aspects of the lower six ribs, 'interdigitating with the transverse abdominis' (Gray 1995)
- The lumbar part arises from two aponeurotic arches (medial and lateral lumbocostal
arches or arcuate ligaments) as well as from the lumbar vertebrae by means of two crura (pillars).

- The lateral crura is formed from a thick fascial covering which arches over the upper aspect of quadratus lumborum, to attach medially to the anterior aspect of the transverse process of L1, and laterally to the inferior margin of the 12th rib.
- The medial crura is tendinous in nature and lies in the fascia covering psoas major.
- Medially it is continuous with the corresponding medial crura, and also attaches to the body of L1 or L2. Laterally it attaches to the transverse process of L1.
- The crura blend with the anterior longitudinal ligament of the spine, with direct connections to the bodies and intervertebral discs of L1, 2 and 3.

- The crura ascend and converge to join the central tendon (Fig. 1.20).
- With attachments at the entire circumference of the thorax, ribs, xiphoid, costal cartilage, spine, discs, and major muscles, the various components of the diaphragm form a central tendon with apertures for the vena cava, aorta, thoracic duct, and esophagus.
- When all these diaphragmatic connections are considered, the direct influence on respiratory function of the lumbar spine and ribs, as well as psoas and quadratus lumborum, becomes apparent.

**Transversus thoracis** (Fig. 1.21)

**Attachments.** Inner surface of the body of sternum and xiphoid process superiolaterally to the lower borders of the 2nd–6th costal cartilages.
Innervation. Intercostal nerves (2–6).

**Function.** Depresses the costal cartilages during exhalation, ribs 2–6.

**Synergists.** Muscles of exhalation.

**Antagonists.** Muscles of inhalation.

**Possible signs of dysfunction of transversus thoracis**

Inadequate lifting of the sternum during inhalation, if shortened
Inadequate excursion of upper ribs during exhalation (‘elevated ribs’), if lax.

**Special notes on transversus thoracis**

This muscle (also called the sternocostalis or triangularis sterni) lies entirely on the interior chest and is not available to direct palpation. It varies considerably, not only from person to person, but also from side to side in the same person (Gray 1995), and is sometimes absent (Platzer 1992).

Latey (1996) reports that the transversus thoracis muscle has the ability to generate powerful sensations, with even light contact sometimes producing reflex contractions of the abdomen or chest with feelings of nausea and choking, as well as anxiety, fear, anger, laughter, sadness, weeping, and other emotions. Latey believes that its closeness to the internal thoracic artery is probably significant since, when it is contracted, the muscle can exert direct pressure on the artery. He believes that physiological breathing involves a rhythmical relaxation and contraction of this muscle and that rigidity is often seen where ‘control’ dampens the emotions which relate to it.

In the following chapter, patterns of dysfunctional breathing will be described. Functional changes always have structural implications, either as part of the etiology, or as a consequence. Keeping in mind the structure while considering functional and dysfunctional behaviour allows for clinical choices and strategies to evolve.
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