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SPECIAL REPORT

Guidelines for mechanical lung function measurements in psychophysiology

THOMAS RITZ,[a] BERNHARD DAHME,[b] ARTHUR B. DUBOIS,[c] HANS FOLGERING,[d] GREGORY K. FRITZ,[e] ANDREW HARVER,[f] HARRY KOTSES,[g] PAUL M. LEHRER,[h] CHRISTOPHER RING,[i] ANDREW STEPTOE,[j] AND KAREL P. VAN DE WOESTIJNE[k]

[a]Department of Psychiatry and Behavioral Sciences, Stanford University, and the VA Palo Alto Health Care System Palo Alto, California, USA
[b]Psychological Institute III, University of Hamburg, Hamburg, Germany
[c]John B. Pierce Laboratory, New Haven, Connecticut, USA
[d]Department for Pulmonary Diseases, Dekkerswald Medical Center, University of Nijmegen, Nijmegen, The Netherlands
[e]Brown University/Rhode Island Hospital, Child and Family Psychiatry, Providence, Rhode Island, USA
[f]Department of Psychology, University of North Carolina, Charlotte, North Carolina, USA
[g]Psychology Department, Ohio University, Athens, Ohio, USA
[h]Department of Psychiatry, University of Medicine and Dentistry–Robert Wood Johnson Medical School, Piscataway, New Jersey, USA
[i]School of Sport and Exercise Sciences, University of Birmingham, Birmingham, UK
[j]Department of Epidemiology and Public Health, University College of London, London, UK
[k]Dienst Pneumologie, Universitaire Ziekenhuizen Gasthuisberg, Leuven, Belgium

Abstract

Studies in psychophysiology and behavioral medicine have uncovered associations among psychological processes, behavior, and lung function. However, methodological issues specific to the measurement of mechanical lung function have rarely been discussed. This report presents an overview of the physiology, techniques, and experimental methods of mechanical lung function measurements relevant to this research context. Techniques to measure lung volumes, airflow, airway resistance, respiratory resistance, and airflow perception are introduced and discussed. Confounding factors such as ventilation, medication, environmental factors, physical activity, and instructional and experimenter effects are outlined, and issues specific to children and clinical groups are discussed. Recommendations are presented to increase the degree of standardization in the research application and publication of mechanical lung function measurements in psychophysiology.

Descriptors: Mechanical lung function, Airway resistance, Respiratory resistance, Spirometry, Pneumotachography, Body plethysmography, Forced oscillation technique, Interrupter technique, Respiratory perception

Despite long-standing interest in respiration by psychophysiologists, it is surprising that little attention has been directed to methodological issues of specific measurement techniques in this area. Although occasional textbook chapters have been devoted to respiration measurements, a more in-depth discussion of techniques dealing with the assessment of mechanical lung function is still missing. Mechanical lung function relates to lung volumes and airflow through the respiratory system as well as the pressure–volume and pressure–flow characteristics as indicators of elastic and resistive properties of the airways, lungs, and chest wall. In clinical practice, mechanical lung function measures are widely used for the diagnosis and management of various pulmonary diseases, most importantly bronchial asthma and chronic obstructive pulmonary disease (COPD). In psychophysiology and behavioral medicine, potential associations between psychological or behavioral processes and mechanical lung function offer an exciting perspective both for basic research and clinical application. Due to the particular importance of the vagal system in governing airway function, the airways offer a unique window into the autonomic nervous system regulation of behavior, emotion, and cog-

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Address reprint requests to: Thomas Ritz, Department of Psychiatry and Behavioral Sciences, Stanford University and VA Palo Alto Health Care System (116F-PAD), 3801 Miranda Ave., Palo Alto, CA 94304, USA. E-mail: tritz@stanford.edu.

nition. Research has also shown that modifications of airway resistance by experimental suggestions can be abolished by parasympathetic blockade (for review, see Isenberg, Lehrer, & Hochron, 1992). Induced emotions, stress, and mood states have been linked to changes in the resistive properties of the airways in health and disease (e.g., Kotses, Westlund, & Creer, 1987; Ritz, Steptoe, DeWilde & Costa, 2000). Interest in these issues extends into the domain of clinical research and application, where the interaction between behavioral factors and lung mechanics can be expected to affect pathophysiological processes and outcomes of disease management, such as in emotion-induced asthma or in dyspnea perception in COPD. The study of mechanical lung function can also further the understanding of basic psychophysiological processes in other organ systems. Extensive research has been directed to the psychophysiology of the cardiovascular system. Despite a tight coupling of cardiac and pulmonary systems to meet the organism's metabolic demand, the latter system has been largely excluded from investigation under this perspective. Epidemiological studies have, however, uncovered the independent role of impaired mechanical lung function as a prognostic factor for the development of arteriosclerosis, myocardial infarction, stroke, and complications in diabetes (Ebi-Kryston, 1988; Klein, Moss, Klein, & Cruickshanks, 2001; Truelsen, Prescott, Lange, Schnohr, & Boysen, 2001; Zureik, Kauffmann, Touboul, Courbon, & Ducimetiere, 2001).

Considerable effort has been devoted to the standardization of lung function testing (American Association for Respiratory Care [AARC], 1994, 1996; American Thoracic Society [ATS], 1991, 1995, 1999; British Thoracic Society and the Association of Respiratory Technicians and Physiologists, 1994; European Respiratory Society [ERS], 1993, 1997). Furthermore, textbooks are available on the basics of lung function testing in pulmonary medicine (e.g., Forster, DuBois, Briscoe, & Fisher, 1986; Stocks, Sly, Tepper, & Morgan, 1996). The present guidelines are not meant to duplicate these efforts, but they aim to introduce and evaluate these techniques for a psychophysiological research context. The needs of psychophysiology often differ from those of clinical assessment of respiratory disorders. A main focus in psychophysiology is on dynamic changes in lung function associated with psychological or behavioral processes, rather than on static diagnostic assessment. The introduction of new techniques for continuous noninvasive lung function monitoring, which are not sufficiently covered by previous guidelines, has opened this area for an increasingly wider psychophysiological research community. The guidelines presented here reflect this development and serve to provide an introduction to the basic physiology of the airways and to measurement principles and procedures of mechanical lung function and its perception.

The present report will not review the whole range of respiratory processes that could be a potential subject of noninvasive investigation in psychophysiology. The analysis of timing, volume, and flow parameters of spontaneous breathing will only be covered where it is of direct relevance for determining the mechanical properties of the airways. Related methods and applications for psychophysiology have been discussed more frequently in previous years (e.g., Wientjes, 1992; Wilhelm & Roth, 1996). The measurement of respiratory gas exchange is also not included, as it would certainly require a separate in-depth discussion. The reader is referred to earlier introductions and reviews of this subject (e.g., Clark et al., 1992; Saisch, 1994). In addition, methods for measuring upper airway obstruction in sleep studies (American Academy of Sleep Medicine Task Force, 1999) will not be outlined here.

Anatomy and Physiology

Anatomy of the Airways

The airways can be divided into the upper airways (nose, nasopharynx, oropharynx, hypopharynx, and larynx), and the lower airways (from trachea to alveolar ducts). Airway resistance, when breathing through the nose, is approximately twice the resistance of mouth breathing. Nasal resistance is elevated even more during allergic, viral, or bacterial nasal infections.

Larynx. The larynx is made up of the thyroid, cricoid, and arithenoid cartilages. It contains the true and false vocal cords. Its rostral aperture can be closed by the epiglottis, for example, during swallowing, or reflexly due to strong stimulation of irritant receptors in the lower airways. It is innervated by the superior laryngeal nerve and the recurrent vagal nerve. One of the main functions of the larynx is phonation. Pathological narrowing of the larynx (in vocal cord dysfunction, enlarged thyroid) gives rise to an increased inspiratory resistance and stridor. This is in contrast with a narrowing of the intrathoracic airways, which causes a predominantly expiratory resistance. Increased upper airway resistance, in vocal cord dysfunction or in athletes during stressful performances, can mimic exercised-induced asthma.

Trachea. The trachea is a 10–12-cm-long tube, 2 cm in diameter. It is composed by a number of U-shaped cartilages connected by ligaments and banded together posteriorly, by a smooth muscle (pars membranacea). Directly posterior is the esophagus, sharing its anterior wall with the posterior wall of the trachea. The proximity of these two structures helps to explain why some asthmatic patients can experience a bronchoconstriction when ingesting very cold drinks or food: the cooling of the esophagus is conducted rather easily to the trachea, and stimulates irritant receptors. The trachea bifurcates into the left and right main bronchi (first generation bifurcation); the right main bronchus bifurcating again into three lobar bronchi; the left one into two lobar bronchi (second generation bifurcation). This branching goes on to the 24th generation, when the bronchioles end at the level of the alveoli. The number of airways increases from one trachea, via two main-stem bronchi, to $8 \cdot 10^7$ alveolar sacs, and $3 \cdot 10^8$ alveoli. The diameter of the individual airways decreases from 15–22 mm in the trachea to 0.4 mm in the alveolar sacs (Grippi, 1995).

The airways from the first generation to the 16th generation are called the conductive zone. No exchange of oxygen and carbon dioxide takes place in this part of the respiratory system. Therefore the conductive zone is referred to as the *anatomical deadspace*. The total resistance at various branches of the airways decreases inversely with the increase of the cumulative cross-sectional area at peripheral levels. The greatest cumulative resistance is in the region from the trachea to the segmental bronchi ($\sim$0.01 $kPa \cdot l^{-1} \cdot s$) versus the level of the terminal bronchioles ($\sim$0.0002 $kPa \cdot l^{-1} \cdot s$), where very many resistances are in parallel. Because the resistance of the peripheral airways is so much smaller than that of the central airways, the changes in airways resistance in the peripheral airways are very difficult to detect. This is why the peripheral airways are called the *silent zone*. They are suspended in the parynchyma of the lung tissue, which generates radial forces that keep these airways open. Resistance of the peripheral airways changes inversely with lung volume: It is lower during inspiration and in hyperinflation. Peripheral airways are compressed during forced expiration, because they do not have a cartilagenous structure in their walls (West, 1990).

Bronchial walls have rings of cartilage or cartilagenous plaques down to the 10th generation down to the beginning of the bronchioles. The bronchial walls contain an inner mucosal layer of pseudostratified epithelium and ciliated cells that transport mucus, produced by goblet cells, towards the mouth. Under the mucosa lies the basal membrane of the mucosa and the submucosal connective tissue. The submucosa contains mucus glands and smooth muscle cells that contract in response to neurogenic or chemical stimuli.

The airway smooth muscle layer is situated between the mucosal layer and the adventitial layer, with fibers running more or less diagonally over the airway. Contraction is via graded depolarization; action potentials occur almost only in asthmatic patients. Metabolites of the arachidonic metabolism (e.g., prostaglandins, histamine, leucotrienes) have direct contractive effects on the smooth muscle. Contraction of the smooth muscles, thickening of the mucosa by (allergic) inflammatory processes, and secretion of mucus all contribute to the increase in airway resistance seen in asthma (National Heart, Lung and Blood Institute [NHLBI], 1997).

Mechanics of Breathing

In breathing, the mechanical system is energized by the respiratory muscles, which in turn are governed by the higher central nervous system via the intercostal and phrenic nerves. Though most breathing is involuntary, a person may voluntarily take a deep breath, hold the breath, cough, sneeze, speak, sing, or use the chest muscles to lift heavy objects. The influence of voluntary control on mechanics of breathing makes the study of mechanics of interest in psychophysiology. For general reading on mechanics of breathing see Forster et al. (1986) or Fishman, Geiger, Macklem, and Mead (1986).

Respiratory muscle forces are expressed as pressure and are measured with a pressure transducer calibrated with a U-tube manometer containing water or mercury (for units see Table A1 in the Appendix). The greatest pressure a person can exert against such a manometer without using the cheek muscles is about 15 kPa above atmospheric on expiratory effort or 10 kPa below atmospheric during inspiratory effort. Clinical experience shows that patients who can exert less than 2 kPa are weak and may need assisted ventilation.

To expand the lungs during inspiration, muscular force has to overcome (a) the force of elastic recoil, a restorative force after stretching the tissue of the lungs, chest wall, and abdomen (proportional to the volume of expansion), (b) force of friction during airflow (proportional to the rate of inspiratory airflow), and (c) force to overcome the inertia of the respiratory system (proportional to mass acceleration).

The pressure exerted on the respiratory system, P_{rs}, is a function of the resulting changes in volume (V), flow (V') and volume acceleration (V''). The relationship may be written:

$$P_{rs} = f_1(V) + f_2(V') + f_3(V''). \tag{1}$$

Pressure used to displace volume is called static pressure (P_{stat}), that generating V' or V'', dynamic pressure (P_{dyn}). If the respiratory system behaves linearly, which is a valid assumption over limited changes of V, V', and V'', equation (1) can be written:

$$P_{rs} = (V - V_0)/C_{rs} + R_{rs}V' + I_{rs}V''. \tag{2}$$

C_{rs} is the compliance, R_{rs} the resistance, I_{rs} the inertance of the system, describing respectively its elastic, frictional, and inertial (or inductive) properties. V_0 is the volume at which the elastic recoil pressure is zero. Similar equations apply to the various components of the respiratory system: chest wall, lungs, and airways. For the chest wall, the driving pressure is the pressure difference between outside and inside of the wall (transthoracic pressure, P_w; pull exerted by the muscles on the wall [or, if the muscles are passive, by outside pressure] minus pressure in the pleura, pleural pressure). For lung tissue, the driving pressure is the difference between pleural pressure and pressure at the level of the alveoli (alveolar P_{alv}; transpulmonary pressure, P_L). For the airways, it is the difference between P_{alv} and atmospheric pressure.

Compliance. In quiet breathing, expiration is brought about by elastic recoil. Inflation of the lungs and chest of a relaxed or paralyzed person to a new volume by a pump to create a pressure difference between mouth and outside the body (transthoracic pressure) allows measurement of a pressure–volume curve relating static pressure (P_{stat}) and change in volume (ΔV). Part of this pressure is needed to inflate the lungs (P_{Lstat}), and the remainder to enlarge the rib cage, diaphragm, and abdomen. The slope of the curve, $V - V_0$ versus P_{Lstat}, measured between end-expiratory volume and the maximum volume of a regular inspiration, is the compliance of the lungs (C_L). It is about 2 l/kPa, but can be half of this if part of the lungs cannot be expanded, or a tenth if the lung tissue is stiff. The lung's pressure–volume curve is S-shaped. This yields a low slope (low C_L, i.e., increased stiffness, or increased "elastance") when it is measured near the bottom or top of the curve.

Resistance. The faster the rate of inflation or deflation, the more dynamic pressure (P_{dyn}) is required to overcome the resistance, which is mostly due to dissipation of energy in the air stream. A small amount of pressure overcomes tissue friction. The ratio $\Delta P_{dyn}/\Delta V'$ is called *resistance to breathing* and for the chest as a whole amounts to 0.3 or 0.4 $\text{kPa}\cdot\text{l}^{-1}\cdot\text{s}$. Part of this pressure is dissipated in the airways and lungs, the remainder in the chest wall. P_{dyn} adds to the P_{stat} needed to inflate the chest (equation 1).

Inertance. The air column in the trachea has a small amount of inertia that has to be overcome by pressure to start it moving (accelerate it). This pressure is regained during deceleration. Similarly, some pressure is needed to start the tissues moving to overcome their inertia. The inertance of the respiratory system is so small that it can be neglected at normal breathing frequency. However, it cannot be neglected during measurements by the forced oscillation technique (FOT; see Forced Oscillation Technique).

Autonomic Nervous System and Humoral Regulation of the Airways

Efferent innervation. The airways and lungs are innervated by both the sympathetic and parasympathetic systems. In healthy individuals, the balance between sympathetic and parasympathetic activity is such that the airways are almost maximally dilated. However, due to a remaining vagal tone, further dilation is achieved when anticholinergics are given. The vagus nerve carries afferent as well as efferent fibers. The efferent parasympathetic system comes from the vagal nerve and is cholinergic, that is, uses the transmitter acetylcholine to induce a bronchoconstriction. The cholinergic system is the most important bronchoconstrictor pathway in man. There is a vago-vagal reflex: Stimulation of afferent vagal receptors (irritant receptors and c-fibers, laryngeal receptors), reflexively leads to increased efferent vagal activity and to bronchoconstriction. Histamine, bradykinine, and prostaglandins stimulate these receptors. Asthmatic patients show an exaggerated bronchoconstrictor response to cholinergic agonists. The cholinergic mechanisms seem to have some predominance in central airways, whereas the adrenergic mechanisms are said to have more peripheral effects (Barnes & Thomson, 1992). The mucus glands are also mainly stimulated by the cholinergic system.

The adrenergic-sympathetic effects on airway caliber are mediated by sympathetic nerves, circulating catecholamines, and α- and β-adrenergic receptors. The most important efferent sympathetic system is β_2-adrenergic and mediates a bronchodilation. There is no direct β-adrenergic innervation of airway smooth muscles. Beta-mimetics activate adenyl-cyclase and cyclic AMP. The latter in turn activates proteine kinase-A, which has a direct effect on the smooth muscle by lowering the intracellular calcium concentration. Beta-blocking agents are potent bronchoconstrictors in asthmatic patients, and therefore absolutely contraindicated. Alpha-adrenergic receptors in the airways mediate a bronchoconstriction. On the other hand, α-receptor stimulation also causes a vasoconstriction in the airway walls, and subsequently an increase in the airway diameter. The net effect is not always predictable, but there were instances where adrenaline administration in an asthma attack caused a fatal bronchoconstriction. Alpha-blocking agents do not substantially dilate the airways, so in the end, the relative importance of this system is rather low. Adrenaline, an α- and β-mimetic, in physiological doses, reduces airway resistance. Sympathetic and parasympathetic nerves both end on the peribronchial plexus in the wall of the airways. This plexus innervates the airway smooth muscle. Apart from the sympathetic and parasympathetic innervation, there is a nonadrenergic–noncholinergic system (NANC) in the efferent vagus nerve. The transmitters in this NANC system are substance P and vasoactive intestinal peptide; the former mediates contraction of the bronchial smooth muscle, the latter relax the airways. Sensory nerves in the airway walls contain substance P. Activation of these nerves either by local stimulation with drugs such as capsaicin, or tobacco smoke, causes local airway reactions such as hyperemia, edema and smooth muscle contraction, presumably by local axon reflexes. The mucus glands are stimulated mainly by the parasympathetic, cholinergic system, which can be blocked by atropine (Richardson, 1988). Figure 1 shows a diagram of the innervation of the airways.

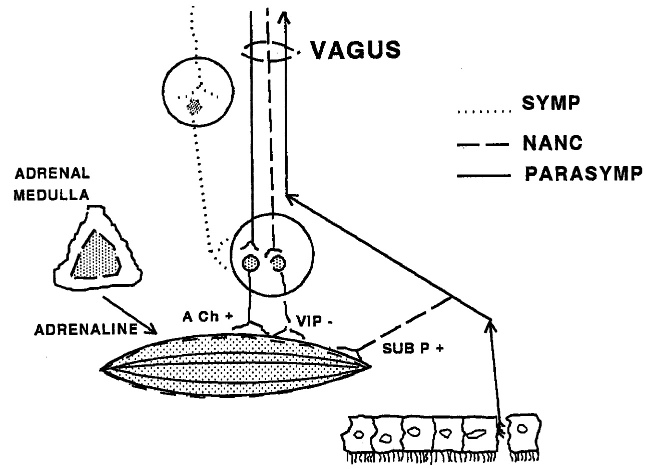

Figure 1. A diagram of the innervation of the airways and its musculature. The smooth muscle is innervated by the sympathetic nerves and by the vagal nerve. Acetylcholine (ACh), and substance P (from afferent nerves) generate an increase in muscular tone (+) and consequently a bronchoconstriction. Adrenaline and vasointestinal peptide (VIP) relax the airway smooth muscle (−) and cause a bronchodilation. The upper circle is the sympathetic ganglion; the lower circle is the intrapulmonary ganglion. Increased sympathetic activity is associated most of the time with increased levels of adrenaline in the blood.

Afferent innervation. The lungs and airways contain three types of receptors, having afferent innervation via different fibers in the vagal nerve. The afferents from bronchopulmonary receptors are thought to play a role in the sensation of dyspnea. However, it remains to be established to what extent stimulation of these afferent pathways can be perceived. The lungs of patients with transplanted lungs are denervated, yet these patients still can be dyspneic. This means that afferent vagal activity is not the sole source of dyspneic sensations.

Irritant receptors or rapidly adapting receptors (RARs) are located in the mucosa of the airways. Their afferent fibers in the vagus are myelinated and are stimulated by inhaled noxious gases or particles: tobacco smoke, ammonia, dust, cold air, and so forth. When the lungs are inflated, these receptors fire only during a short time, and decrease their firing frequency quite rapidly. When an inflammatory process has disrupted the tight junctions of the mucosa, these receptors can be reached more easily by the inhaled irritants. Increased activity in afferent vagal fibers reflexively leads to a bronchoconstriction via the efferent vagal nerves, in response to minor stimuli that would not give such effects on an intact mucosa in normal individuals. This is the definition of *bronchial hyperresponsiveness*. Furthermore, stimulation of these receptors reflexively causes tachypnea (respiratory frequency >12/min), tachycardia and hyperventilation (Meessen, van den Grinten, Luijendijk, & Folgering, 1997).

Stretch receptors or slowly adapting receptors (SARs) are located in the smooth muscles of the more peripheral airways. When the lungs are inflated, these receptors remain active, and hardly adapt. The afferent traffic of action potentials, via myelinated fibers, inform the "respiratory centers" about the inflation status of the lungs. At high inflations or at tidal volumes of more than 1 l, this afferent activity inhibits the inspiratory neurons in the brain stem, and thus initiates the expiration. This classical Breuer–Hering reflex is probably not very important in regulating the pattern of breathing in resting human individuals.

J-receptors, or Juxta capillary receptors, are located in the parynchyma of the lung. Their nonmyelinated afferent C-fibers are slow conducting. Stimulation of these fibers in animals with capsaicin or bradykinine causes a respiratory arrest, followed by rapid shallow breathing. In humans, pulmonary interstitial edema also stimulates these J-receptors in pneumonia, pulmonary embolism, and left heart failure, and leads to rapid shallow breathing.

Measurement of Pulmonary Function

Lung Volumes and Airflows

The measures reviewed in this section serve as indirect indicators of airway resistance (flows and volumes inspired or expired during a fixed time interval) during forced inspirations and expirations (i.e., flows executed at the highest possible speed). The volume of gas in the lung that can be displaced by inspiration and expiration is generally directly measured at the mouth as a volume, by means of a spirometer, or from the integration of airflow, by means of a pneumotachograph. To measure the volume of gas that cannot be displaced by respiratory movements, other methods such as gas dilution (not discussed) or body plethysmography (see Body Plethysmography) are required.

Spirometry

Spirometers register the volume of air displaced during either quiet or forced breathing. The classical water spirometer has now been replaced by "dry" spirometers that consist either of a bellows or an inner cylinder of light plastic inside an outer cylinder, with a flexible rolling seal allowing the inner cylinder to move. The movements are recorded mechanically or by a linear transducer. For ambulatory studies, pocket spirometers are available. They are suited for measuring the volume of a forced expiration and generally consist of a small turbine turned by the expiratory flow. To record breathing and forced respiratory maneuvers continuously over several minutes, spirometers should be mounted in a closed breathing circuit to allow CO_2 absorption and the addition of oxygen (see Laboratory Set-up: Equipment Maintenance and Quality Control).

Calibration, leaks, frequency characteristics. Calibration is performed regularly using a 3-l syringe. Leaks should be detected by loading the spirometer by at least 0.2 kPa: The record should remain level for at least 1 min. Most commercially available spirometers measuring directly volumes have frequency characteristics that are satisfactory for accurate recording of forced expiratory and inspiratory volumes, but not of maximal flows (specifically peak expiratory flow).

Correction for temperature and barometric pressure. Expired air is at body temperature and saturated with water vapor (BTPS). In the spirometer, the air cools down: This results in a volume decrease. If the spirometer has been calibrated with room air, a correction factor should be applied to restore the measured volume changes to those occurring in the lungs (same temperature and barometric pressure): For instance, if the spirometer is at 23°C (temperature under the spirometer bell), this correction factor will be $\times$1.084 (at sea level barometric pressure) (Quanjer et al., 1993).

Main parameters. In addition to the measurement of tidal volume (V_T), the volume of air displaced during spontaneous breathing, spirometers are mostly used for the recording of forced respiratory maneuvers and determining forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1; Figure 2). VC is the largest volume of air an individual can displace by exhaling slowly (or forcefully, FVC) starting from the maximal inspiratory level. FEV_1 is the largest volume displaced during the first second of a forced exhalation starting from the same maximal inspiratory level. Whereas VC is an expression of lung size, FEV_1 is influenced both by lung volume and the dimensions of the airways. The ratio FEV_1/VC (or FEV_1/FVC) is independent of lung size and is a measure of airway obstruction. Another expression of forced expiration is the mean expiratory flow during the middle half of the FVC ($FEF_{25\text{–}75\%}$), formerly called the maximal mid-expiratory flow ($MMEF$). When experiments are performed in a closed circuit (see Laboratory Set-up: Equipment Maintenance and Quality Control), V_T and VC may be measured in succession, allowing the separate determination of expiratory reserve volume (part of VC that can be expired from the level of spontaneous end-expiratory volume, and required for the determination of residual volume, RV).

Procedure. Detailed guidelines for measurement of lung volumes and maximal airflows have been published by both the ATS (1995) and Quanjer et al. (1993). Briefly, the participant should rest 15 min prior to the test. A mouthpiece is inserted between the teeth and lips and the nose is clipped. Preliminary explanation and forceful coaching during the performance are absolutely required. At least three satisfactory blows should be performed, with a maximum of eight blows if performance is faulty. Criteria of satisfactory performance of forced ventilatory maneuvers are detailed in the ATS and ERS recommendations. For clinical use, the largest values of $(F)VC$ and FEV_1 values are reported.

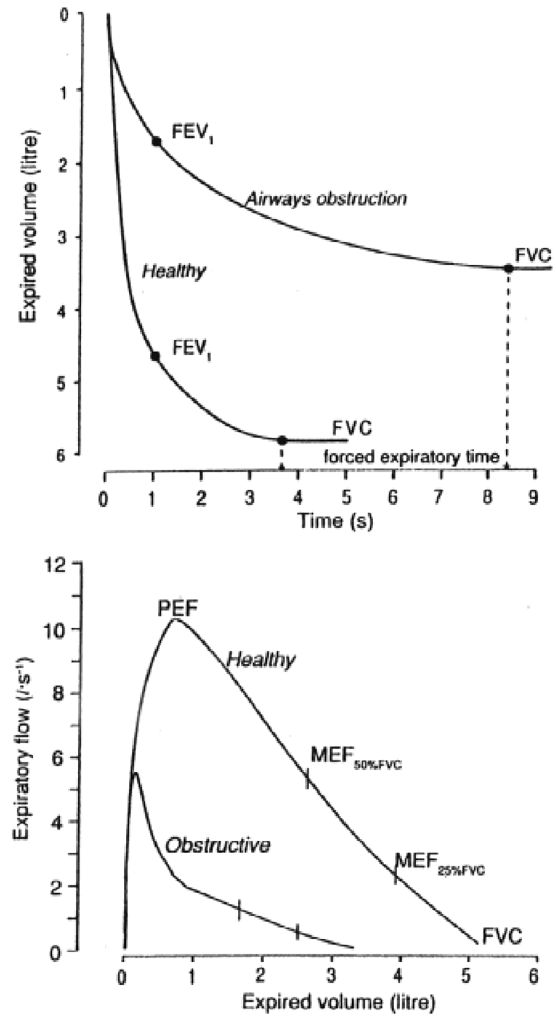

Figure 2. Idealized forced expiratory flow spirograms (volume–time curves; upper panel) and maximal expiratory flow-volume curves (superimposed at total lung capacity; lower panel) of healthy individuals and patients with airway obstruction. Note that a longer expiratory time is required for FVC measurement in patients (with permission from Quanjer et al., 1993, modified).

Pneumotachography

The pneumotachograph is an instrument used to measure airflow. Flow is derived from the measurement of pressure drop over a fixed resistance, consisting of a bundle of parallel capillary tubes (Fleisch type) or of a metal screen (Lilly type). The apparatus is designed in such a way that the air flowing through the resistance has a laminar profile, ensuring a direct proportionality between pressure drop over the resistance and flow. This condition is met only within a given range of flows. When this range is exceeded, the relationship between pressure drop and flow becomes nonlinear in the sense that the pressure drop increases progressively more for a given increase in flow. The limits of linearity should be known. These limits can be enhanced by increasing the diameter of the fixed resistance, resulting necessarily in an increase of the dead space of the apparatus. Hence the size of the pneumotachograph should be selected as a function of the maximum airflow developed during the measurements. For the recording of spontaneous breathing, devices should be linear to $1.5\ \mathrm{l \cdot s^{-1}}$ (e.g., pneumotachograph Fleisch No. 2 with a dead space of 40 ml). The frequency characteristics of the pneumotachographs allow a correct measurement of airflow during forced inspiratory and expiratory maneuvers (provided the limits of linearity are not exceeded). Volume is obtained from analog or digital integration of the flow signal.

Calibration. The calibration of integrated flow (volume) is performed with a 3-l gas syringe. The linearity of the device can be checked by delivering the gas both rapidly and slowly: The volume readings should be independent of gas flow rate. Generally, the pneumotachograph, heated to 30 °C, is calibrated with room air; the calibration factors are then applied, without further corrections to inspired and expired gas. In fact, under those conditions, only inspiratory flow (at room temperature) is determined correctly. Because of the difference in temperature, water vapor pressure, and composition of the expired gas, expired airflow will be underestimated by about 5%. A systematic underestimation of expired with respect to inspired volume will result in a linear drift of the baseline of the volume reading. The latter can be corrected for electronically or digitally. Some flow meters automatically perform this correction.

Main parameters. Recording of airflow during quiet breathing is generally used to measure V_T (by integration of the flow signal). Maximal expiratory (*MEF*) and inspiratory flows (*MIF*) over the course of forced respiratory maneuvers (during expiration: *FVC*) are used to detect airway obstruction. Those flows are generally related to a fixed percentage of *FVC* (to be exhaled) in a flow-volume curve: for example, $MEF_{75\%FVC}$, $MEF_{50\%FVC}$, $MEF_{25\%FVC}$ (Figure 2, lower panel). Measurement of maximal flows requires the use of a large pneumotachograph meeting the condition of linearity for the flows achieved during those forced maneuvers (up to $15\ \mathrm{l \cdot s^{-1}}$ in adults). In healthy individuals, maximal expiratory flows over the first half of the *FVC* mainly reflect the flow characteristics of large airways (trachea and central airways), whereas those over the second half mostly reflect the flow characteristics of the smaller intrathoracic airways (Quanjer et al., 1993): A bronchoconstriction at this level mainly influences $MEF_{50\%FVC}$ and $MEF_{25\%FVC}$. This is because a forced expiration is accompanied by a compression of the central intrathoracic airways: The stronger the individual's effort (the larger the pleural pressure), the more pronounced the compression up to a point. As a result, beyond peak expiratory flow, the increase in driving pressure, due to the increase in pleural pressure, is met by no increase of airflow through the compressed airways. In the further course of the expiration, *MEF* is thus effort independent (flow-limitation). The effective driving pressure is then the lung's elastic recoil, and the effective flow resistance that of the upstream airways. FEV_1 is less effort dependent and more reproducible than peak expiratory flow in patients with lower airway obstruction because it corresponds to the summation over time of *MEF* mainly of the proportion of *FVC* characterized by flow limitation (ATS, 1995; see also for a further discussion of reproducibility). In normal individuals, early expiration is effort dependent.

Procedure. When the pneumotachograph is used to measure maximal airflows the procedure is the same as for spirometry.

Peak Flow Measurements

A widely used parameter for the detection of airway obstruction is peak expiratory flow (*PEF*), the most rapid airflow produced during a forced expiration with maximal effort starting from a maximal inspiratory level. The value of *PEF* can be determined by means of a pneumotachograph of suitable size. A number of simple, inexpensive, portable devices have been developed to monitor directly (and solely) the *PEF*. Those devices consist of a hinged vane uncovering an orifice the size of which varies with airflow (Wright peak flow meter) or in a plastic piston moving within a cylinder, the wall of which has a rectangular slot to allow the air to escape. These devices are difficult to calibrate. Their performance varies. The measurement is similar to that of spirometry, except that a nose clip is not required. Guidelines have been issued both by the ATS (1995) and the ERS (1997). Peak flow meters are generally used as a tool for self-management of asthmatic patients or for occupational and epidemiological studies. In general, the long-term within-instrument stability of the measurements is more important than the its accuracy (Douma et al., 1997; Folgering, van den Brink, Van Heeswijk, & van Herwaarden, 1998). As an alternative to peak flow meters, a number of pocket spirometers have recently been developed, measuring both flows and volumes of a forced expiration (and inspiration).

Inductive Plethysmography (Respitrace)

In contrast to the previously mentioned devices, the Respitrace measures chest and abdominal wall movement rather than airflow or volume. It consists of a pair of insulated coils strapped over the chest and upper abdomen. The change in mean cross-sectional area of the coil caused by chest and abdominal wall displacement modifies the inductance of the coil. The apparatus is calibrated by recording simultaneously the outputs while the individual is breathing through a pneumotachograph (or spirometer). A multiple regression is performed on the digitized signals, relating the (calibrated) output of the pneumotachograph to the combined outputs (chest wall and abdomen) of the Respitrace. The regression coefficients of the latter outputs can then be used to reconstruct the volume signal (Loveridge, West, Anthonisen, & Kryger, 1983). The calibration should be repeated every time body posture is even slightly changed.

Potential and Limitations for Psychophysiology

The major advantages of spirometry are its reliability, validity, and widespread acceptance and availability as a tool to assess the clinical significance of variations in airway function. However, it provides only indirect information on the resistive properties of the airway passages. Ambulatory monitoring is one of the major fields of application, using peak-flow meters or miniature electronic spirometers (e.g., Hyland, 1990; Ritz & Steptoe, 2000). In the laboratory and in the clinic, spirometry has been used to assess effects of emotional states and relaxation therapy on asthma (e.g., Florin, Freudenberg, & Hollaender, 1985; Lehrer et al., 1994).

The primary disadvantage of spirometry is that the forced expiratory maneuvers take several minutes. Because the participant's effort and concentration may affect this measure, a minimum of three consecutive maneuvers are prescribed by current standards (ATS, 1995). Lack of motivation, fatigue, and deep relaxation may compromise a participant's effort. Compensation for this can be possible through adequate coaching. This should continue during each maneuver, to encourage maximum effort. Thus, the technique cannot be used to measure moment-to-moment changes in airway function produced by psychological tasks. Measurements are also susceptible to experimenter effects (see Instructional and Experimenter Effects).

Among individuals with asthma, forced expiratory maneuvers can produce bronchoconstriction (Orehek, Gayard, Grimaud, & Charpin, 1975). This is manifest by progressively decreasing values of FEV_1 with succeeding maneuvers. In such cases, it may not be possible to obtain reproducible curves. Also, changes in respiratory patterns can have important psychological effects. Assigned respiratory maneuvers can produce either relaxation or hyperventilation in some individuals, particularly when performed the first few times. Patients with asthma or other lung diseases may be less susceptible to these effects, because they are much more familiar with the technique from frequent use at home and in medical care settings. For inexperienced participants, thorough preexperimental training is necessary. An implication of the various side effects of spirometry is that, wherever measurements of spirometry are combined with measurements of lung function by body plethysmography or forced oscillations, these latter measurements should ideally precede spirometry. More research is needed on effects of multiple spirometric assessments in experiments, and the ideal number of records in the psychophysiological context.

Inductive plethysmography is the only technique that has the potential to leave the participant unaware of the fact that his/her breathing is recorded. It is minimally invasive and does not require a mouthpiece or nose clip (except for calibration). Breathing patterns can be altered systematically by participants' awareness that breathing is the target of the study, and by the use of a mouthpiece and nose clip (Han, Stegen, Cauberghs, & Van de Woestijne, 1997). The technique cannot inform about resistive or elastic properties of the airways, but it is a useful tool for monitoring the breathing pattern during continuous measurements of respiratory resistance (see Experimental Design: Controls and Confounds, Ventilation).

Airway resistance

As discussed in Mechanics of Breathing, the determination of airway resistance (R_{aw}) requires the measurement of the driving pressure, alveolar pressure (P_{alv}, the pressure difference between alveoli and mouth) versus V'. Whereas V' is measured at the mouth by means of a pneumotachograph, P_{alv} is not directly available. A technique for measuring it is body plethysmography, which is used as a reference technique for other noninvasive measurement techniques of mechanical lung function.

Body Plethysmography

This method measures R_{aw}, which is mainly affected by middle-sized airways with smooth muscles. In addition, the technique can be used to measure the thoracic gas volume (TGV; the lung volume at which R_{aw} is determined), and the functional residual capacity (FRC). For more detailed descriptions of the methods involved in plethysmography, the reader is referred to original work by DuBois and coworkers (Briscoe & DuBois, 1958; DuBois, Botelho, Bedell, Marshall, & Comroe 1956; DuBois, Botelho, & Comroe 1956). The original and most commonly used method is the "constant volume box," which will be described here (for description of the "volume displacement box," see Jaeger & Otis, 1964).

Method and procedures. The basic idea is an application of Boyle's law, which states that the volume of a gas varies inversely with its pressure. The body plethysmograph is an air-tight chamber or box resembling a telephone booth. The person sits inside, rebreathing the air within it, and the small changes of volume (ΔV) resulting from compression of alveolar air during exhalation and expansion of alveolar air during inhalation (Figure 3) lead to reciprocal changes of pressure within the box (P_{box}). This pressure change is detected by a sensitive pressure transducer (linear range ±

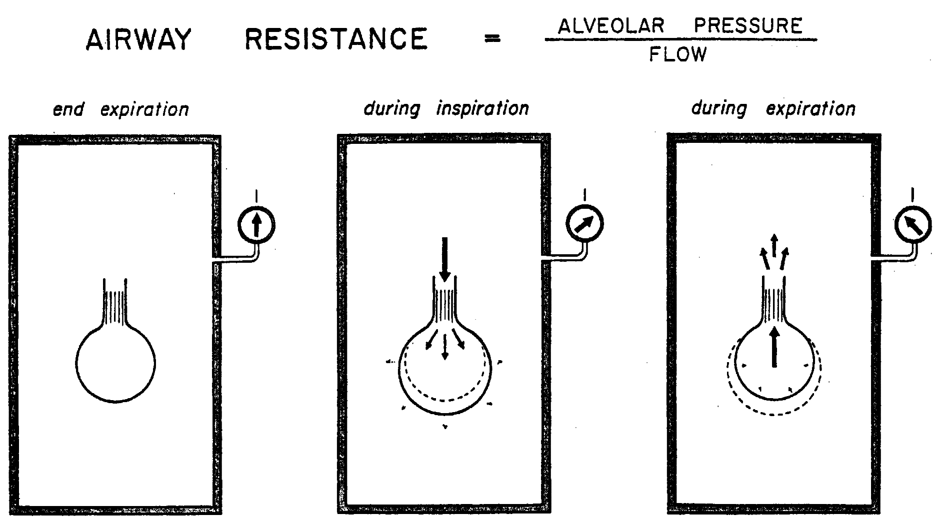

Figure 3. Measurement of airway resistance: body plethysmography technique. The rectangle represents the air-tight body plethysmograph. The patient is represented by alveoli and conducting airway, and his airway resistance by parallel lines in the conducting airway. The circle with pointer represents a sensitive gauge that continuously measures pressure in the box around the patient. During inspiration (center) the alveoli have enlarged from the original volume (broken line) to a new volume (solid line); during expiration (right), the alveoli have returned to their original volume. The small displacements of air in a pneumotachograph during expiration and inspiration will not modify the pressure inside the box because the displaced volumes simply shift from alveoli to box and vice versa. The volume changes, inducing changes in box pressure (and recorded by the gauge), are the result of the expansion and compression of alveolar gas produced by changes in alveolar pressure during breathing (with permission from Forster et al., 1986).

0.2 kPa). To measure V', the person breathes through a pneumotachograph (linear between ± 1.5 l/s). The ratio between V' (on the *y*-axis) and P_{box} (on the *x*-axis) forms the first element in the calculation R_{aw}. It is shown as a slope on the X–Y display. The slope can be measured with a protractor attached to the screen.

Two types of breathing methods can be used: (1) Panting method: The person makes rapid shallow breathing movements (panting), at about two cycles per second, through a warmed (37°C) flowmeter. Panting prevents the air from undergoing temperature and humidity changes or variations in oxygen and carbon dioxide exchange during the breathing cycle, by keeping the boundary between the air that the person breathes from the box, and the air inside the lungs, within the dead space of the pneumotachograph. In panting, the larynx stays wide open, whereas during the expiratory phase of normal tidal breathing it is often half-closed. (2) Quiet breathing method: A person for whom the panting procedure is inappropriate can obtain acceptable results by rebreathing tidal air at a normal breathing frequency in and out of a rubber bag containing warm (37°C), moist air, which helps to avoid the changes in temperature, humidity, and pulmonary gas exchange. Because the larynx is apt to partly close during expiratory airflow, the resistance slope is measured during inspiration rather than expiration.

To measure the second element in the calculation of R_{aw}, a shutter (solenoid valve) at the mouthpiece is closed and the person expands and compresses the gas in the chest by making voluntary respiratory efforts against the shutter. A strain gauge manometer (linear range ± 5 kPa) connected to the breathing tube on the mouth side of the closed shutter measures changes in mouth pressure (ΔP_{mo}). The assumption is that during shutter closure with the absence of airflow, ΔP_{mo} approximates ΔP_{alv}. When the airflow is stopped, P_{mo} on the *y*-axis and P_{box} on the *x*-axis are displayed, forming the second slope. Dividing the second slope (P_{alv}/P_{box}) by the first slope (V'/P_{box}) eliminates the P_{box} common to both slopes. The result is P_{alv}/V', or the resistance of the airway plus that of the pneumotachograph. From this sum we subtract the resistance of the pneumotachograph to find the R_{aw} of the individual.

During the measurement of R_{aw}, the person usually pants at a *TGV* slightly greater than the resting respiratory level (*FRC*). The actual *TGV* is important because R_{aw} is not independent of the lung volume at which it is measured. *TGV* can be calculated from the small changes in lung volume (V_L) by air compression and decompression while the person is breathing against the shutter, and from the ratio of ΔP_{mo} to $P_B - P_{H2O}$. P_B is barometric pressure (normally 101.3 kPa), and P_{H2O} is the partial pressure of water vapor at 37°C (normally 6.3 kPa). Then, $TGV = (\Delta V_L/\Delta P_{mo}) \times (P_B - P_{H2O})$.

Quality control. To calibrate, a volume of 30 ml is introduced into and withdrawn from the box at a frequency equal to the breathing frequency (about twice a second for panting, or less rapidly for normal breathing). A 5- or 10-l bottle or tank serves as a reference pressure for the backside of the box pressure (P_{box}) gauge. The mouth pressure gauge is calibrated using a water manometer. Lung volume may be simulated with a piston and a bottle containing froth or copper sponges for isothermal air compression. The pneumotachograph is calibrated with known rates of airflow. Commercially available body plethysmographs and flowmeters may contain algorithms that "correct" for phase lag, gas temperature, and humidity. If the cheeks bulge when the person is making respiratory efforts against the closed shutter, the breathing efforts should be slowed to avoid looping in the plethysmograph tracing. The cheeks may also be supported with the hands. Training of the operator is necessary to recognize and discard bad records (for examples, see DuBois & Van de Woestijne, 1969).

Parameters. When normal people are panting, the R_{aw} averages 0.13 $\text{kPa}\cdot\text{l}^{-1}\cdot\text{s}$ (range 0.08 to 0.24 $\text{kPa}\cdot\text{l}^{-1}\cdot\text{s}$) at a *TGV* of 3.5 l (Fisher, DuBois, & Hyde, 1968). If the person is given a bronchodilator, R_{aw} is reduced from its initial value to about 0.06 $\text{kPa}\cdot\text{l}^{-1}\cdot\text{s}$. A person who has mild asthma, and is unaware of bronchoconstriction, may have an R_{aw} of 0.3 $\text{kPa}\cdot\text{l}^{-1}\cdot\text{s}$. A moderate asthmatic might have 0.6, and a more severe asthmatic 1.0 $\text{kPa}\cdot\text{l}^{-1}\cdot\text{s}$. Because resistance varies inversely with lung volume, "specific R_{aw}" (sR_{aw}) is calculated by $R_{aw} \times TGV$. The reciprocal of R_{aw}, airway conductance (G_{aw}), is sometimes preferred, as it is directly proportional to *TGV* between and within individuals (unit: $\text{l}\cdot\text{s}^{-1}\cdot\text{kPa}^{-1}$). To correct for *TGV* changes, the "specific G_{aw}" (sG_{aw}) is calculated by G_{aw}/TGV. Normal values for the sG_{aw} have been found to average 2.4 $\text{s}^{-1}\cdot\text{kPa}^{-1}$ and fall within a range between 1.3 and 3.6 $\text{s}^{-1}\cdot\text{kPa}^{-1}$. Though sG_{aw} is often used to characterize or to follow changes in the conducting airways, it has the drawback of losing information about the *TGV* itself.

Other factors than bronchoconstriction or dilation may alter the pressure-flow relation. These include change in the width of the glottis or change in lung elastic tension caused by a change in *FRC* (Butler, Caro, Alcala, & DuBois, 1960). Emphysema reduces elastic recoil of lung tissues, increasing their *FRC* and *RV*. Their G_{aw} approaches zero at *RV* (or obstruction is complete in terms of R_{aw}), implying that air is trapped in the patient's lungs. Asthmatics may breathe at an increased *TGV* to decrease the resistance to breathing. People with pulmonary fibrosis have increased lung tissue tension and a G_{aw} larger than predicted from their *TGV*. In obesity, the diaphragm is pushed upward so that *RV* and G_{aw} approach zero at a volume close to *FRC*, interfering with ventilation at the base of the lungs.

Potential and Limitations for Psychophysiology

Body plethysmography has been used in various studies relevant to psychophysiology (e.g., Dahme, Richter, & Maß, 1996; McFadden, Luparello, Lyons, & Bleeker, 1969; Mathé & Knapp, 1971; Meyer, Kröner-Herwig, & Spörkel, 1990). The main advantage of this technique, which lies in the most accurate noninvasive monitoring of resistive properties of the airways, is often outbalanced by disadvantages in terms of cost efficiency and restrictions on study design. The equipment is expensive, heavy, and space demanding. Regular maintenance and consumables can make it a costly asset for the average laboratory. Intensive training is needed for the operator, and good technical support is essential. Studies can be implemented more efficiently in collaboration with hospital-based specialists in pulmonology.

The measurement setup with the participant seated in a closed cabin makes the application of psychophysiological tasks sometimes difficult. Although the walls are usually made of glass, allowing the use of projector screens and so forth, the straight view can be blocked for the participant by the pneumotachograph head. Equipment for tasks can be brought into the cabin; however, its temperature effects must be determined carefully and sealing of the cabin must not be impaired. Each additional device in the cabin has to be taken into account in the volume correction of R_{aw} and *TGV* measurements. Special constructions have been reported, for example, for bicycle ergometry (Guenard, Vieillefond, & Varene, 1977). The elaborate procedures needed to determine R_{aw} and *TGV* reduce the application of body plethysmography to studies looking at more stable, tonic influences on the airways. Including full determination of TGV and R_{aw}, the minimum time necessary for a measurement is approximately 90 s. Dropping TGV at the expense of volume correction and assessing only sR_{aw} (Dab & Alexander, 1976) can reduce the average time needed for the trained operator to 30 s. Particular maneuvers such as panting require concentrated efforts from participants and will interfere with most behavioral tasks. The respiratory maneuvers themselves can be expected to alter autonomic regulation and thus may abolish transient, phasic effects elicited by behavioral tasks. Measurements with the quiet breathing method produce less interference and require less attention from the participant; sR_{aw} is measured directly during experimental tasks with the quiet breathing method, whereas TGV is obtained from prior baseline measurements with the panting technique (von Leupoldt & Dahme, 2000). When the focus is on actual changes in R_{aw}, this approach must rely on the assumption that changes in TGV from baseline during the task are negligible, as it will be difficult to determine whether changes of sR_{aw} are due to changes in R_{aw} or TGV or both.

A point of concern from the psychological perspective are emotional effects of enclosure in the cabin (for cases studies, see Stein, 1962). The mobility of the participant's head is usually restricted by the fixed mouthpiece. Although the resulting relative fixation of body position is in the service of measurement accuracy, it can add to the psychological stress. Sufficient adaptation periods have to be provided at the beginning of each experiment. Routine checks of state anxiety and general mood changes attributable to the measurement procedures should accompany the experimental protocol. The actual extent of most of these influences and limitations on psychophysiological experiments has rarely been examined.

Respiratory Resistance

Forced Oscillation Technique (FOT)

Measurement principle. The FOT determines the impedance of the total respiratory system. In the conventional setup, sinusoidal oscillations are imposed at the mouth by means of a loudspeaker: Induced pressure and flow signals are recorded at the mouth by means of a transducer and a pneumotachograph, respectively. The impedance, Z, is the ratio of the amplitudes of pressure and flow and corresponds to the total pressure drop required to oscillate, that is to develop a flow in the respiratory system (rs): $Z_{rs} = |P|/|V'|$, $|P|$ and $|V'|$ corresponding to the amplitudes of pressure and flow, respectively. Generally, depending on the mechanical characteristics of the system, P and V' signals are not exactly in phase[1]: the phase shift between the two signals, φ, is used to partition Z_{rs} in a so-called "real" (or resistance), R_{rs}, and "imaginary" (or reactance), X_{rs} component:

$$R_{rs} = Z_{rs} \cos \varphi$$

$$X_{rs} = Z_{rs} \sin \varphi.$$

In a simple linear model of the respiratory system consisting of a resistance connected in series to a capacitance (or compliance), R_{rs} and X_{rs} correspond to the resistance and the elastance (the inverse of the compliance) of the system respectively. By modifying the frequency of oscillations, R_{rs} and X_{rs} are thus measured at several frequencies. In practice, the frequencies of interest are combined. The loudspeaker is then driven by a complex signal containing several discrete frequencies. When the succession of the frequencies is randomized but the amplitude of the various frequencies is equal, a so-called pseudo-random noise (PRN) signal (usually the fundamental frequency plus its harmonics) is generated. The various frequencies are separated afterwards by submitting pressure and flow signals to a Fourier analysis. The requirement for the latter analysis is that the relationship between the two signals is linear. This condition is generally met. Indeed, notwithstanding the fact that the pressure–volume and pressure–flow relationships of the respiratory system are curvilinear, the amplitude of the forced oscillations is small: The imposed displacements of the system do not exceed the limits of linearity. In addition, this requirement of the application of the Fourier analysis can be checked easily by modifying the amplitude of the signals. In a linear system, this procedure will not influence the values of impedance. If, in addition, care is taken to keep the frequency of the oscillatory signal higher than the breathing frequency (e.g., by a factor of 5 or more) there will be no interference between oscillatory and breathing signals, and it becomes possible to perform the measurements during spontaneous breathing, the lowest frequency at which measurements with FOT are still feasible being 2 Hz.

Generally, measurements are performed between 2 and 32 Hz, the signal being made up of a fundamental frequency of 2 Hz and its harmonics: 4, 6, 8, … 32 Hz. To cover several breathing cycles during the measurements, the latter are performed over 16 s, thus yielding average values of R_{rs} and X_{rs} over about four breathing cycles. As a check for the linearity and the signal-to-noise ratio of the measurements, a coherence function is calculated. The latter is the equivalent in the frequency domain of a correlation coefficient in the time domain. The coherence should be at least 0.90. For a more detailed outline of the technique, the reader is referred to DuBois, Brody, Lewis, and Burgess (1956), Lándsèr, Nagels, Demedts, Billiet, and Van de Woestijne (1976), and Michaelson, Grassman, and Peters (1975).

Devices. The device consists of a loudspeaker connected via a wide tube to a pneumotachograph (Fleisch or Lilly type, linear up $1.5\ \mathrm{l} \cdot \mathrm{s}^{-1}$) and a pressure transducer (linear up to 0.2 kPa). The individual breathes via the pneumotachograph into a wide-bore side tube acting, because of its inertive properties, as a low impedance pathway for the subject's breathing but as a high impedance for the forced oscillations (Figure 4). The technical requirements of the device, of input signal and of data processing have been described (Van de Woestijne, Desager, Duiverman, & Marchal, 1994). During the measurements, the individual, wearing a nose clip, breathes quietly via a mouthpiece, and supports his cheeks and the mouth floor with both hands.

A modification of the technique using pressure impulses has been described by Lándsèr et al. (1976). The loudspeaker generates short impulses containing various harmonics of 2 Hz repeated every 0.5 s, instead of presenting the harmonics in a random order. Earlier comparisons with the multiple frequency technique showed a less favorable signal-to-noise ratio. Recently, a new device has been developed generating five 45-ms impulses per second, allowing the determination of Z_{rs}, R_{rs}, and X_{rs} at frequencies 5–35 Hz (Hellinckx, Cauberghs, DeBoek, & Demedts, 2001; Kohlhäufl et al., 2001). Further research will have to determine its accuracy and validity.

A simplified device, operating at one frequency, was designed by Korn, Franetzki, and Prestele (1979). Oscillations are produced

[1]The frequency at which pressure and flow are exactly in phase is called natural or resonant frequency (fn) of the system.

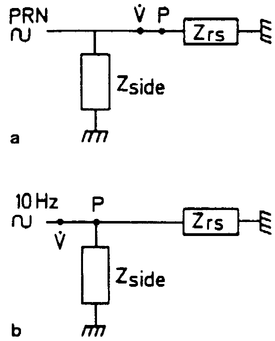

Figure 4. Schematic representation of the set-up of forced oscillation technique: **a:** The impedance of the respiratory system (Z_{rs}) is measured by forcing oscillations (pseudo-random noise, PRN, signal) generated by a loudspeaker, at the subject's mouth. Pressure, *P*, and airflow, V', are measured at the mouth. The subject breathes via a side tube (Z_{side}). **b:** Simplified setup: A small pump produces a constant oscillatory (10 Hz) V'. *P* is recorded at the level of the mouth and side tube (see text; with permission from Cauberghs & Van De Woestijne, 1984)

by a small pump, developing a known flow signal. Pressure is recorded at the entrance of a Y-tube connected on one side to the mouth and on the other to the side tubing (Figure 4). Because the impedance of the latter is known and the oscillatory flow is constant, Z_{rs} can be calculated from the pressure signal only. The device determines Z_{rs} (or R_{rs} and X_{rs} if, in addition to Z_{rs}, the phase angle φ is measured) at a frequency of 10 Hz.[2] The advantage with respect to PRN devices is that Z_{rs} values are obtained continuously during the breathing cycle.

Calibration. A reference impedance, the values of which are comparable to those one wants to measure within the range of applied frequencies, should be available. Techniques have also been described to test the linearity of the device (Desager, Cauberghs, & Van de Woestijne, 1997).

Artifacts. The major problem of the FOT is that its results are influenced by the shunt characteristics of the upper airway. Part of the oscillations applied at the mouth is "lost" in movements of the cheeks and mouth bottom. This shunt does not influence markedly the measurements in healthy individuals, but will play a progressively larger role when the impedance of the respiratory system is abnormally large. As a consequence of this shunt, R_{rs} will be underestimated and will show a negative frequency dependence (resistance will decrease with frequency), and X_{rs} will decrease with a corresponding increase of the resonant frequency. Support of the cheeks by means of the palms of the hands reduces this influence by stiffening the cheeks (Cauberghs & Van de Woestijne, 1989; Michaelson et al., 1975). A modification of the technique prevents the shunt from biasing the results: When the oscillations are applied around the head of the person, the cheeks are not oscillated any more because pressure at the inside and the outside of the upper airway and mouth are nearly identical (Peslin, Duvivier, Didelon, & Gallina, 1985). This technique is quite successful but rather invasive (enclosure of the person's head in a canopy).

Interpretation of the results. These should take into account the influence of the upper airway shunt. This artifact does not modify the measurements in healthy adults, however. In the latter, an increase of Z_{rs} will be correctly detected, though the size of this increase may be influenced by the upper airway shunt. In a recent study, Farré, Rotger, Marchal, Peslin, and Navajas (1999) suggest expressing the results in admittance (the reverse of impedance) rather than in impedance. They demonstrate that this procedure allows, when changes of admittances in a same individual are compared (e.g., before and after bronchial challenge), the elimination of the influence of the upper airway shunt on the observed changes in admittance. Besides the upper airway shunt, the measurements mainly reflect the impedance of the larger airways (pharynx, larynx, trachea, large bronchi). The technique is very sensitive to changes in glottis aperture. At low frequencies (<4 Hz), R_{rs} is also influenced by the viscoelastic properties of the respiratory system, and at higher frequencies (>12 Hz), by the inertance of the air in the central airways. It is likely that the frequency best corresponding to R_{aw} is $\cong$10 Hz (Bates, Daroczy, & Hantos, 1992).

Potential and limitations for psychophysiology. The FOT has often been used in the psychophysiological context, such as in studies on stress and emotion induction (e.g., Levenson, 1979; Ritz et al., 2000) or biofeedback of respiratory resistance (Maß, Dahme, & Richter, 1993). The equipment is relatively light and space-saving, the handling is easy, and the maintenance is economical. The technique allows the participant to breathe spontaneously, thus reducing demands on preexperimental skills training and on attention during an experimental protocol. As a consequence, it is also less susceptible than spirometry to experimenter and instructional bias. It provides a continuous measure of resistance and thus allows for an investigation of dynamic resistance changes during ongoing psychological or behavioral processes. However, it shares the disadvantages of all equipment for direct measurements of respiration by necessitating the use of mouthpiece and nose clip. These can be a source of distraction from tasks and can interfere with other physiological measurements or observations such as electromyography of the facial muscles (e.g., Ritz, George, & Dahme, 2000). The sensitivity of the FOT to the upper airway shunt (cheeks and mouth floor) necessitates additional accessories such as padded clamps or elastic bands. Support of these areas with the palms of the hands is not feasible for longer measurements, as the stability of this control is not guaranteed and it will distract considerably from the performance of specific tasks. Although breathing through the tube increases dead space slightly, it is usually well tolerated if sufficient adaptation time is provided. Nevertheless, the duration of continuous measurements should be restricted, as collection of saliva or drying of the upper airways can become unpleasant and lead to secondary physiological adjustments. Although resistance can be measured continuously, only the single-frequency method and the impulse method allow for a breath-by-breath analysis. The output of the PTN technique provides averages over successive 16-s epochs, thus limiting the resolution for an analysis of phasic processes. Due to its sensitivity to glottis movements, tasks that are likely to activate the vocal cords (e.g., mental arithmetic) can produce artifactual increases in values.

Interrupter Technique

Measurement principle. The interrupter technique for the measurement of R_{rs} was pioneered by von Neergaard and Wirz (1927). The technical difficulties in measuring driving pressure for a calculation of respiratory resistance are solved by a technique of short

[2]The values displayed as R_{os} on the device are approximate values of oscillatory resistance, indicated on the pressure scale, and estimated from Z_{rs} (φ is not measured).

interruptions of airflow during spontaneous breathing. It is assumed that immediately after airflow interruption, P_{mo} equilibrates with pressure in the alveoli. The equipment consists of an interrupter valve and pressure transducer that is connected to the airway by a mouthpiece or face mask. Pressure is measured at the mouth during interruption of the airflow by the closure of a valve. Interrupter resistance (R_{int}) is then calculated by the ratio of change of P_{mo} to flow at the time of occlusion. Its reciprocal is interrupter conductance (G_{int}). Usually, the measurements are made during a series of five or more interruptions while the participant breathes through the mouthpiece. Pressure changes almost instantaneously after interruption, and, if the characteristics of the valve are satisfactory (closure time $\leq$ 10 ms; leakage flow after occlusion: $<$ 0.6 ml/s; Kessler, Mols, Bernhard, Haberthür, & Guttmann, 1999), the rapid pressure transient recorded at the mouth is an approximation of the P_{alv} that existed immediately before interruption. Curves of P_{mo} plotted against time ($P_{mo}(t)$) after airflow interruption consist of an initial rapidly changing phase often with high frequency oscillations and then a secondary slowly changing phase. An estimate of P_{alv} at the time of occlusion can be derived from these $P_{mo}(t)$ curves by backwards extrapolation (Phagoo, Watson, Pride, & Silverman, 1993).

Theoretical studies suggest that resistance is underestimated by R_{int} when there is airflow obstruction, due to compliance of the extrathoracic airways, which may delay equilibration between P_{alv} and P_{mo}. However, supporting the cheeks and pharynx, thereby minimizing upper airway compliance by improving rigidity, and a greater closing speed of the shutter nowadays improves accuracy of the interrupter method (Oswald-Mammosser et al., 1997; Phagoo, Watson, Silverman, & Pride, 1995; Sly & Bates, 1988). Likewise, Bates et al. (1988) estimated that an increase in airway resistance of up to 10 times is still correctly measured by the interrupter method, as long as compliance of the upper airways is not excessively high.

Potential and limitation for psychophysiology. The technique has rarely been used in the psychophysiological research setting, although it shares some of the advantages of the FOT. Continuous breath-by-breath analysis of resistance is possible, but, in practice, limited to achieving higher accuracy by averaging across respiratory cycles. Most of the necessary cautions for artifact avoidance in FOT (reduction of upper airway shunt) apply for the interrupter method, too. The rapid occlusions can usually be heard, but interference with normal breathing is only small. The duration of the shutter closure is critical in reducing sensations of discomfort. Recently, a small hand-held version has become available that would allow ambulatory measurements of airway resistance with an interrupter (Bridge, Lee, & Silverman, 1996). Validation studies are needed to determine whether this technique can increase sensitivity of lung function self-measurements in field studies compared to the commonly used spirometry or *PEF* techniques.

Comparison between Methods of Airflow Resistance Measurement

Partitioning of Flow Resistances

Measurements of airway and respiratory resistance include contributions from different parts of the respiratory tract. Body plethysmography measures R_{aw}, which includes resistances of the upper airways. These are reduced when measurements are made during panting due to the wider glottic opening. FOT and interrupter technique both measure R_{rs}, which includes additional resistances of lung and thoracic tissue. Table 1 shows summarized

Table 1. *Average Estimates of Resistance in Units of $kPa \cdot l^{-1} \cdot s$ (and Percentages of the Total Resistance) for Partitioning of Flow Resistance in Measurements of Total Airway, Pulmonary, and Respiratory Resistance in Healthy Adults at a High Spontaneous Breathing Frequency (19–20 Cycles/Min)*

	Total airway resistance		Total pulmonary resistance		Total respiratory resistance	
Upper airways[a]	0.06	(45%)	0.06	(30%)	0.06	(15%)
Lower airways	0.08	(55%)	0.08	(40%)	0.08	(20%)
Lung tissue	—	—	0.06	(30%)	0.06	(15%)
Chest wall	—	—	—	—	0.20	(50%)
Total (100%)	0.14	—	0.20	—	0.40	—

[a]Values refer to mouth breathing.

and rounded estimates for fractions of flow resistance from studies on healthy individuals, in which measurements were performed during spontaneous breathing through the mouth at *FRC* and V_T in the sitting position (Bachofen, 1966; Barnas et al., 1992; Ferris, Mead, & Opie, 1964; Hantos, Daróczy, Suki, Galgoczy, & Csendes, 1986; Hyatt & Wilcox, 1961; Jaeger & Otis, 1964; McIlroy, Mead, Selverstone, & Radford, 1955).[3] The estimates were obtained by different combinations of these techniques with more invasive methods such as the esophagus balloon catheter or pressure recording via a needle inserted into the extrathoracic part of the trachea. Most authors reported a considerable interindividual variation in estimates, probably due to anatomic differences. Estimates also depend on disease states (e.g., the lower airways account for a higher proportion in asthma or pulmonary emphysema), flow (e.g., during panting the contribution of the upper airways is reduced), and on the respiratory cycle. During inspiration, the upper airway component is smaller, probably due to the wider inspiratory glottis opening.

Spirometry versus Body Plethysmography, FOT, and Interrupter Technique

Indices derived from forced expiratory maneuvers allow only for an indirect measurement of airflow resistance, as compared to body plethysmography, FOT, and interrupter technique. Because the bronchial geometry is quite different during quiet breathing and forced expirations, one does not a priori expect good correlations between values of resistance (quiet breathing or panting) and parameters of forced expiration, except in disease states characterized by large increases in peripheral airway resistance. Physiological factors determining measurement outcome in forced expirations are the dimensions of the intra- and extrathoracic airways, resistive and elastic properties of the lungs and thorax, and the performance of the respiratory muscles (mainly abdominal expiratory muscles). These factors can be operative to a different degree in respiratory patients and healthy individuals (ERS, 1997). With respect to disease states, the forced expiratory maneuver can provide indices that reflect predominantly either larger (central) or smaller (peripheral) airway functioning (see Pneumotachography, Main Parameters). It has also been attempted, on the basis of

[3]Note that breathing through the nose (by use of a face mask) would markedly modify the relative contributions.

model studies, to derive information concerning small airways from the frequency dependence of R_{rs}, determined by FOT. Though theoretically attractive, conclusions of that type are only possible if the influence of the upper airway shunt has been corrected for (see Forced Oscillation Technique).

On an empirical level, moderate to high correlations between baseline FEV_1 and R_{aw} or sG_{aw} have been observed (van Noord, Clément, Van de Woestijne, & Demedts, 1991). On the other hand, correlations between FEV_1 and R_{rs} by single or multiple frequency techniques are only low to moderate. Indices of spirometry usually compare well with R_{rs} in the detection of bronchodilation and bronchoconstriction. Table 2 provides examples of studies comparing measures of spirometry with R_{rs} (FOT, interrupter method) under baseline conditions, bronchodilation (usually by β-adrenergic inhaler), or bronchoconstriction (by inhaled histamine, methacholine, or cold air) in clinical samples. The selected studies are for illustration only, as agreement can depend on a number of factors, such as age, clinical status, or sample size.

FOT versus Body Plethysmography

In principle, one expects R_{rs} or Z_{rs} values larger than R_{aw}, as lung and thoracic tissue resistances are included in R_{rs}. This has been observed in healthy individuals (Làndsér et al., 1976). In addition, glottic narrowing will increase R_{rs} measured during tidal breathing, as compared to R_{aw} obtained during panting. When Z_{rs} is increased in patients or children (above 0.6–0.8 $\text{kPa}\cdot\text{l}^{-1}\cdot\text{s}$), upper airway shunt will become more prominent and reduce values of R_{rs}. In multiple frequency FOT, this is indicated by a negative frequency dependence of R_{rs}. Under these circumstances, values of resistance when measured at higher frequency (FOT) are lower than at spontaneous breathing or panting frequency (body plethysmography). Overall, studies usually show good agreement between FOT and body plethysmography (Table 2).

Interrupter Technique versus FOT and Body Plethysmography

Similar to FOT, the interrupter technique measures R_{rs}; thus R_{int} values are expected to be higher than R_{aw}. It is also influenced by the shunt properties of the upper airways. Compared to the interrupter technique, the advantage of FOT is that it allows a separation between the influence of tissue deformation and of airway resistance (at lower frequencies), and gives, in addition, values of X_{rs} providing information about tissue (lungs and thorax) compliance (at low frequencies), and (mainly gaseous) inertance (at higher frequencies). In healthy individuals, R_{aw} is best approximated with R_{int} calculated from the initial rapid drop of pressure in the interrupter technique (Bates et al., 1992). The ensuing slow pressure drop corresponds to the tissue deformation resistance (Jackson, Milhorn, & Norman, 1974). In the presence of ventilation unevenness, resistance measured by R_{int} will be underestimated and will correspond in fact to the resistance of the better-ventilated lung segment. Although a breath-by-breath measurement of resistance can be provided by R_{int}, it is usually only determined at distinct times during the breathing cycle (inspiratory and/or expiratory), compared to resistance measurement across the whole cycle provided by FOT.

Correlations between baseline R_{aw} and R_{int} are usually high in health and in low to moderate airway obstruction. Research using the interrupter technique has mainly focused on children, showing overall good agreement with R_{aw} for bronchodilation and -constriction (Table 2). Although values of R_{int} and R_{rs} by FOT usually show a tight relationship, sensitivity to detecting changes in resistance is sometimes higher with FOT (e.g., Delacourt et al., 2001). A higher within-individual variability has often been observed with the interrupter method than with other techniques.

Choice of Measurement Technique for Psychophysiology

Table 3 provides an overview of features relevant to the psychophysiological research setting, and the different potentials of techniques. *Selectivity* relates to the capability of yielding information on airway resistance selectively, with body plethysmography as the gold standard. *Nonintrusiveness* relates to the interference with normal psychological and physiological function, in particular with normal breathing. *Practicability* in terms of interference with the experimental protocol is greater in the FOT and interrupter technique. These techniques also allow for the *continuous monitoring* often important in psychophysiological research. *Experimenter effects* (see Instructional and Experimenter Effects) are of concern in spirometry; however, the technique has clear advan-

Table 2. *Examples of Studies Investigating the Agreement between Respiratory Resistance (FOT, Interrupter Method) and Airway Resistance (Body Plethysmography) or Forced Expiratory Flows (Spirometry) for Baseline Measurements and Reactivity to Bronchodilators or Bronchoconstrictors*

	Forced oscillation technique				Interrupter method			
	Agreement	Age	*N*	Source	Agreement	Age	*N*	Source
Spirometry								
Baseline	+	A	75	Van Noord et al. (1991)	+++	C	107	Carter et al. (1994)
Bronchodilation	+++	C	118	Delacourt et al. (2001)	○	C	25	Bridge et al. (1996)
Bronchoconstriction	+++	C	31	Lebeque et al. (1987)	○	C	21	Bisgaard & Klug (1995)
Body plethysmography								
Baseline	+++	A,C	125	Van Noord, Smeets, Clément, Van de Woestijne, & Demedts (1994)	+++	A	43	Chowienczyk et al.(1991)
Bronchodilation	++	A,C	125	Van Noord et al. (1994)	+	A,C	37	Worth et al. (1987)
Bronchoconstriction	+++	A,C	53	Van Noord, Clément, Van de Woestijne, & Demedts (1989)	*	C	20	Klug & Bisgaard (1996)

Note. Agreement is based on correlations between absolute or change scores (+ $<$ 0.40, ++ $>$ 0.40 to $<$ 0.60, +++ $>$ 0.60), or comparisons of dose-response curves or sensitivity indices (differences: $*$ significant, ○ nonsignificant).
A = adult, C = children and adolescents.

Table 3. *Comparison between Different Measurement Techniques of Airflow Resistance in Terms of Features Relevant for Research in Psychophysiology*

	Spirometry	Pneumotachography	Body plethysmography	Forced oscillations	Interrupter technique
Selectivity	− [a]	− [a]	+++	++	+
Nonintrusiveness	−	−	−	++	++
Experimental practicability	−	−	−	+	+
Continuous measurement	○	○	○	+++	++
Experimenter effects	−	−	+	+	+
Economy	+++	++	−	++	++
Ambulatory application	+++	○ [b]	○	○ [c]	+
Clinical application	+++	+++	+	−	−

+++ = excellent, ++ = good, + = satisfactory, − = not satisfactory, ○ = feature not available.
[a] Spirometry and pneumotachography only yield indirect estimates of airway resistance (by forced expiratory maneuvers).
[b] ++ for recently developed pocket devices that share essential features of pneumotachographs.
[c] Initial tests of an ambulatory device have been reported recently by Rigau et al. (2002).

tages in terms of *economy*, *ambulatory monitoring*, and *clinical application* for diagnosis and therapy monitoring. The FOT and interrupter technique are less established in clinical practice, and lack sensitivity in states of stronger airway obstruction.

Assessment of Perception of Mechanical Lung Function Changes

Respiratory Sensation

Increases in the resistance to breathing due to acute airflow obstruction, which are documented with lung function tests, are often compared to the perceived intensity of obstruction using either the Borg Scale or Visual Analogue Scales (VAS). Borg (1970) first described a scale ranging from 6 to 20 to measure perceived exertion during physical exercise. The scale was later modified to a 10-point scale with verbal expressions of severity anchored to specific numbers. Additional terms at the ends of the scale anchor the responses, thus facilitating absolute comparisons to stimuli and enabling direct interindividual comparisons. Although care must be taken to provide consistent and specific instructions when using the scale, extensive reports demonstrate the reliability and validity of Borg ratings of breathlessness (ATS, 1999). The VAS consists of a line, usually 100 mm in length, placed either horizontally or vertically on a page with anchors to indicate extremes of a sensation (Aitken, 1969). Frequently used anchors include *not breathless at all* to *extremely breathless* and *no shortness of breath* to *shortness of breath as bad as can be*. Scoring is accomplished by measuring the distance from the beginning of the scale to the level indicated by the individual. Both the reliability and validity of the VAS as a measure of breathing discomfort have been reported (Gift, 1989).

Since Rubinfeld and Pain (1976), the relationship between perceived and objectively measured declines in lung function has been analyzed in two general ways: (1) between-individuals correlations using pairs of observations obtained from a number of patients exhibiting a range of symptom scores and lung function values, and (2) within-individual correlations between symptoms and lung function measured repeatedly in the same individual. Between-individual correlations are surprisingly high, whereas within-individual correlations are more variable, but also yield more meaningful interpretations about the correspondence between airway perception and abnormalities in lung function (Harver & Mahler, 1998).

Airflow limitation produced by a variety of stimuli is perceived similarly, and patients experience "just noticeable symptoms" when the FEV_1 declines to around 70–75% of predicted normal FEV_1 values following spontaneous or methacholine-induced bronchoconstriction. The intensity of breathlessness for a given level of airflow obstruction can also be expressed as a perception score: the rating on a modified Borg scale corresponding to a 20% decline in FEV_1 (PS_{20}; Boulet, Leblanc, & Turcotte, 1994). For histamin provocation, this perception score is similar for both genders, increases with age, and is normally distributed in a clinical population with respiratory symptoms. The ability of individuals to detect acute increases in airflow obstruction is susceptible to learning. Young adults, with or without asthma, improve in their ability to discriminate between the presence or absence of airflow increases (Harver, 1994), or between levels of airflow increases (Stout, Kotses, & Creer, 1997) in training tasks involving feedback or fading-plus-feedback.

There have been a number of attempts to uncover the ways in which individuals use language to describe respiratory sensations, and to relate specific descriptors to distinct pathophysiological states and respiratory pathophysiology (e.g., Harver, Mahler, Schwartzstein, & Baird, 2000; Kinsman, Luparello, O'Banion, & Spector, 1973). For example, in relation to changes in mechanical properties of the lungs, chest tightness seems to be specifically related to asthma and mild to moderate states of airway obstruction, whereas a sense of effort or increased work of breathing is more related to stronger increases in airway resistance, decreases in compliance of the lungs or chest wall, or stronger mechanical load on the ventilatory muscles (Manning & Schwartzstein, 2001).

Load Compensation and Load Perception

Added resistive or elastic loads impose an extrinsic or "extrapulmonary" load to breathing that alters normal ventilatory parameters (e.g., the rate at which air flows to and from the lungs). *Resistive loads* mimic the type of flow limitation experienced by patients with obstructive lung disease by altering normal pulmonary pressure–flow relationships. They are constructed generally of porous material (e.g., metal or plastic filters, or metal or nylon mesh screens) and connected in series, or steps, to provide a fixed range of increases in flow resistance. (Imagine breathing through a series of straws, each with a lumen narrower than the one before.) Resistive load intensities are determined in units of

$kPa \cdot l^{-1} \cdot s$ by passing air through the resistors at various known flows and simultaneously measuring the drop in pressure across the resistor that results. By comparison, elastic loads mimic the type of volume restriction experienced by patients with interstitial lung disease and are produced by requiring individuals to breathe from airtight containers of various sizes joined in parallel with tubing and normally vented to atmosphere. These loads are quantified by measuring the pressure within the container as known quantities of air are injected or withdrawn from the circuit. The ratio between the change in pressure for a given change in volume describes the elastance of the container (kilopascals per liter; see also Davenport & Revellete, 1996; Zechman & Wiley, 1986).

Two main goals have stimulated the study of added loads in humans (Pengelly, Rebuck, & Campbell, 1974). One goal relates to the regulation of ventilation (i.e., *load compensation*), and in the consequences of disturbances in the mechanics of breathing in lung disease, on respiratory homeostasis. The other purpose relates to the quantification of the relationship between mechanical perturbations and respiratory sensations (i.e., *load perception*; for review, see Harver & Mahler, 1998).

Laboratory Setup: Equipment Maintenance and Quality Control

Cleaning and Precautions against Contamination

Direct transmission of infections via pulmonary function testing has not been documented. Still, there is potential for transmission of infection through direct contact or aerosol droplets. Accordingly, all equipment parts that routinely come in contact with the oropharynx or its secretions should be cleaned and disinfected (or sterilized) or discarded after use. This applies to mouthpieces, tubing, valves and connections, and to any piece of equipment with visible condensation from expired air. The manufacturers should provide recommendations about the cleaning and disinfection of their equipment. In-line filters are available for patients with known transmissible infectious diseases. These filters may modify the frequency characteristics of the measuring device. Detailed guidelines for hygiene and infection control have been issued (AARC, 1996; ATS, 1995; Mahler & Loke, 1989).

Quality Control

Calibration

Calibration should be available for all measured variables (volume, flow, pressure, etc.) and should be performed on a regular basis. Some instruments are provided with an internal calibration system. The latter is helpful to verify the reproducibility of the recordings and may be used as check for accuracy. However, calibration with an independent primary "physical" is an absolute requirement for accurate measurements. Corrections and calculations performed by computer algorithms should also be checked.

Respiratory Circuit

When a respiratory circuit is put together, three factors should be carefully considered: leaks, dead-space, and impedance of the circuit.

1. Leaks are avoided in the assembly of a circuit by pushing tubing over connections rather than stuffing it inside. Scotch tape or sticking plaster should never be used to cover a leak.

2. Dead space is the volume of gas common to the inspiratory and expiratory sides. It consists generally of the mouthpiece, the respiratory valve, and any interposed instrument (e.g. pneumotachograph). The dead space should be kept to a minimum (less than 100 ml). Face masks have necessarily a larger dead space than a mouthpiece and are often the source of leaks.

3. Impedance of the circuit. The distensibility of tubing is generally not a problem unless large pressure changes are produced (exercise circuits). The resistance of the tubing should be kept as low as possible by using tubing as short and as wide as possible. For a respiratory circuit, the internal diameter should be 2.5 cm for recording of breathing at rest, more for exercise testing (Freedman, 1983). It should be taken into account that the frequency response of the measuring device is influenced by the characteristics of the circuit.

There are two types of circuits: open and closed. In an *open circuit*, gas is inspired from one source (room air, gas reservoir) and expired through another route. Inspired and expired air are separated by a valve connected to the mouthpiece. Following expansion, the gas is kept in a neoprene bag (50–100 l) or in a meteorological latex balloon (300 l). Valves should be leak-free, of low dead space, and of low resistance. In a *closed circuit*, gas is recirculated by means of a pump (or a low-resistance one-way valve). The composition of the gas is kept constant by a continuous supply of oxygen to the circuit and by absorption of exhaled CO_2 by means of soda-lime. If the recording time is short (less than 1 min), control of gas composition is not necessary. However, the circuit should be flushed with room air between successive measurements. Every time the circuit is disassembled and/or cleaned, calibration of the instrument should be performed.

Experimental Design: Controls and Confounds

Ventilation

Effects of ventilation on mechanical lung function are of particular concern, as emotional or behavioral influences on ventilation are numerous (Boiten, Frijda, & Wientjes, 1994). Higher lung volumes are inversely related to resistance (Briscoe & DuBois, 1958). In body plethysmography, *TGV* is controlled for in indices such as sG_{aw}. There is little experience with similar indices in the FOT or interrupter method (see, e.g., Fisher et al., 1968). However, concomitant monitoring of end-expiratory volume changes (ΔFRC) by pneumotachograph trace (Maß et al., 1991) or calibrated inductance plethysmography belts can assist the interpretation of these measurements. Measurements of the FOT are also positively flow dependent (Tomalak, Peslin, & Duvivier, 1998). In diagnostic settings, measurements are usually taken during quiet, relaxed breathing; however, this is not necessarily the case in the psychophysiological experiment.

Resistance changes throughout the respiratory cycle, with higher values during expiration (Cauberghs & Van de Woestijne, 1992). Thus, changes in the respiratory timing parameters alone can alter basal resistance values: If greater parts of the respiratory cycle are occupied by the inspiratory fraction, resistance averaged for the whole respiratory cycle will be lower. This is of importance for measurements by FOT that monitor resistance throughout the respiratory cycle. A separation of R_{rs} components according to components of the respiratory cycle can assist the interpretation.

Direct influences on airway smooth muscle tone can be exerted by lung inflation: Activation of lung stretch receptors triggers a relaxation (Nadel & Tierney, 1961). This mechanism could impede the demonstration of bronchoconstriction in a behavioral task that also stimulates ventilation. It can also reduce the utility of forced expiratory maneuvers in spirometry for an assessment of task-related bronchoconstriction. Changes in blood gas tension can be another sources of influence on the airways. The fall in PCO_2 related to hypocapnia has bronchoconstrictive effects, but there is conflicting evidence on effects of hypercapnia and hypoxia (Simon, Zanaboni, & Nyhan, 1997; van den Elshout, van Herwaarden, & Folgering, 1991).

Recommendation. Continuous measurements of ventilation should be included in psychophysiological studies of mechanical lung function. This is of particular importance when inferences should be drawn to putative autonomic mechanisms underlying airway responses.

Instructional and Experimenter Effects

Measurements of mechanical lung function in the context of behavioral settings must take into account additional potential sources of unwanted variance. Extensive research has demonstrated a particular susceptibility of airway resistance to suggestions (Kotses, Rawson, Wigal, & Creer, 1987; for review, see Kotses, 1998). Experimenter effects have long been recognized as a major factor in forced expiratory maneuvers. Instructions motivating the participant to do better results in improvement in both inspiratory capacity and FEV_1 (Harm, Marion, Creer, & Kotses, 1984), and variations in instructions can have marked effects (Harm, Marion, Creer, & Kotses, 1985).

Recommendations for elimination of these confounding effects consist of strict standardization of instruction and, ideally, the use of double-blind procedures.

Medication

Studies including patients with respiratory diseases must take into account medication effects that can modify basal airway tone, airway response to stimulation, perceptions from the respiratory system, or general autonomic function. Therefore drug intake should be carefully recorded. As the actual extent of the confounding effects is often difficult to establish, and withdrawal is not always justified for ethical reasons, the researcher might consider studying subgroups of patients with homogeneous types of medication or patients with stable asthma who do not need medication at that moment. Table 4 gives an overview of common drugs that target the airways and recommended periods of wash-out before an experiment.

Other Confounding Variables

Major confounds for comparison of baseline lung function between individuals are sex, age, height, and weight (especially in children), which are taken into account in reference values (for reviews, see ATS, 1991; Quanjer et al., 1989, 1993; for R_{rs} at 10 Hz, see Ducharme, Davis, & Ducharme, 1998; Gimeno, van der Weele, Koeter, & van Altena, 1992; for R_{int}, see van Altena & Gimeno, 1994; Merkus, Mijnsbergen, Hop, & de Jongste, 2001). Tables 5 and 6 list other potential sources of influence on baseline lung function and airway reactivity.

Research on Special Populations

Children

In objective assessment of pulmonary function in children, two important factors that demand attention are (1) the motivational level in effort-dependent testing such as spirometry, and (2) the degree of understanding of the test requirements. Motivation can be enhanced by computer programs tied to performance (e.g., "blow down the wall" program, Pulmonary Data Service, Louisville, Colorado). An experienced researcher can judge the degree of effort expended and use enthusiastic coaching to provoke a maximal response. However, instructions for any test cannot be standardized for the pediatric population because of the differences in language and cognitive abilities. For suitable instructions and procedures to achieve reliable spirometry test results in preschool children see Eigen et al. (2001).

The FOT and the interrupter technique have several advantages for use with children (e.g., Carter, Stecenko, Pollock, & Jaeger, 1994). Children can be anxious about entering the box and adding a companion can be distracting (Lindemann, 1979). FOT measurements are not effort dependent, and repeated assessment does not lead to fatigue. Physical growth issues are critical when working with children (Fritz & Wamboldt, 1998). As lung capacity varies directly with size, normal expected values vary greatly from child to child (for an in-depth discussion of measurement issues in children, see Stocks et al., 1996; Zapletal, Samanek, & Paul, 1987).

Particular considerations are necessary when studying perception of added resistive loads in children. A small 7-year-old may have a basal resistance four times greater than a large 14-year-old, necessitating adding resistances as a percentage of basal resistance to obtain meaningful comparative data (McQuaid, Fritz, Yeung, & Mansell, 1996). To obtain a valid description of respiratory symptoms experienced by children is more problematic than with adults, in particular the numerical quantification of the level of dyspnea. The problems stem from children's lack of subtle language for description, attention problems, and difficulty abstracting their experience to fit onto scales. A recent review has suggested four methods for symptom experience assessment, each with advantages and disadvantages (Fritz et al., 1996). (1) Prediction of *PEF*:

Table 4. *Common Drugs for Respiratory Disease and Recommended Wash-Out Periods Prior to an Experimental Protocol*

Type of medication	Type of action	Patient groups	Withdrawal prior to experiment
Beta-2 adrenergic agonists, short-acting	Bronchodilation	Asthma, COPD	8 hr
Beta-2 adrenergic agonists, long-acting	Bronchodilation	Asthma, COPD	16 hr
Anticholinergic agonists	Bronchodilation	COPD, Asthma	8 hr
Theophyllines	Anti-inflammatory, Bronchodilation	Asthma	to be continued
Corticosteroids	Anti-inflammatory	Asthma	to be continued
Cromoglycates	Anti-inflammatory	Asthma	to be continued
Leukotriene antagonists	Anti-inflammatory, Bronchodilation	Asthma	24 hr

Table 5. *Confounding Variables in Measurements of Mechanical Lung Function*

Confounding variable		Lung function	Source
Sex	Male > female		ATS (1991)
Height	Increasing height	⇑	ATS (1991)
Weight	Childhood: increasing weight	⇑	
	Adulthood: increasing weight	⇓	ATS (1991)
Life-span	Childhood: increasing age	⇑	
	Adulthood: increasing age	⇓	Kerstjens, Rijcken, Schouten, & Postma (1997)
Race	Non-Caucasian	⇓	ATS (1991)
Time of day	Asthma: nighttime, morning	⇓	Lebowitz, Krzyzanowski, Quackenboss, & O'Rourke (1997)
Season	Spring summer > autumn, winter		McKerrow & Rossiter (1968)
Climate	Cold and/or dry air	⇓	McFadden (1992)
Air pollution	Ozone, sulfur dioxide, acid aerosols, nitrogen dioxide, particles	⇓	Committee of the Environmental and Occupational Health Assembly, ATS (1996)
	Tobacco smoke	⇓	Kerstjens et al. (1997)
	Asthma: airborne allergens	⇓	Pearlman & Lemanske (1996)
Illness	Respiratory infection	⇓	Nicholson, Kent, & Ireland (1993)
Physical activity	Posture: standing > sitting, supine		Michels, Decoster, Derde, Vleurinck, & Van de Woestijne (1991)
	Dynamic exercise	⇑	Warren, Jennings, & Clark (1984)
	Static muscle tension	⇑	Ritz, Dahme, & Wagner (1998)
	Asthma: exercise	⇓	Godfrey (1997)

Most asthma patients who monitor *PEF* at home are familiar with their personal range and have had sufficient practice and feedback to help shape subjective assessments. Healthy children require more training and have less variability, making this approach more problematic. (2) VAS: A number of child-specific VASs have been developed, such as the Brown Asthma VAS (Fritz, Spirito, Yeung, Klein, & Freedman, 1994). In this scale, both verbal anchors amid cartoon depictions are used to help orient a child to the use of the scale. (3) Categorial descriptors are commonly used in everyday life and may be more understandable for children than a VAS. Categorical scales can employ verbal or pictorial descriptors for young children, such as the FACES scale used by children to quantify pain (McGrath, 1987). (4) Treatment indicated: In this approach, the symptoms are categorized on the basis of the extent of treatment needed: for example, inhaler versus nebulized medications versus urgent visit to the physician. The advantage to such a rating system is that it is clinically relevant and easily understood by patients who are knowledgeable in self-management techniques. A major disadvantage is that it confounds treatment knowledge (and judgment) and perception of illness.

Clinical Populations

Asthma

The individual measurement techniques have sufficient to excellent capability to detect clinically significant degrees of airway narrowing in asthma. Commonly used criteria for clinical relevance include a 12–20% decrease in *PEF* or FEV_1 (NHLBI, 1997), or a 40% increase in R_{aw} (Cropp, 1979). The clinical significance of the bronchodilator effect of a drug usually is set at a 12% change in FEV_1, with a minimum increase of 200 ml. In pharmacological challenge tests that assess airway hyperreactivity (Sterk et al., 1993) as one of the defining aspects of asthma, a provocation concentration of histamine or methacholine is measured, which leads to a 20% fall in FEV_1 (PC_{20}).

Among asthmatic patients, there is a group of nonperceivers who do not seem to sense their bronchoconstriction or stimulation of irritant receptors. These patients are at risk for severe exacerbations, because they do not adapt their use of medication, nor do they seek adequate medical help during severe bronchoconstrictions. Life-threatening asthma attacks are rarely seen in the psychophysiology laboratory, although clinically significant changes may occur, and should be treated with great caution. In general, measurement procedures are considered to be safe, even for patients with severe asthma. Although spirometry tests can produce limited bronchoconstriction in asthma, spirometry is not contraindicated. Regular home *PEF* assessments are assigned for asthma management, *particularly* during exacerbation of symptoms. Patients should carry their bronchodilator medication during laboratory and ambulatory measurements. Repeated testing should not be done when the test produces bronchoconstriction. Laboratory test-

Table 6. *Confounding Variables in the Measurement of Airway Reactivity*

Confounding variable		Airway reactivity	Reference
Age	Childhood: increasing age	⇓	
Time of day	Nighttime	⇑	van Aalderen, Postma, Koeter, & Knol (1989)
Air pollution	Ozone, nitrogen dioxide	⇑	Committee of the Environmental and Occupational Health Assembly of the ATS (1996)
	Tobacco smoke	⇑	Mullen, Wiggs, Wright, Hogg, & Paré (1986)
	Asthma: airborne allergens	⇑	Pearlman & Lemanske (1996)
Diet	High salt	⇑	Gotshall, Mickleborough, & Cordain (2000)
Illness	Respiratory infection	⇑	Cockroft (1997)

ing should be abandoned and bronchodilator medication should be given if patients show signs of asthma exacerbation.

COPD

In general, patients with chronic airway obstruction can experience similar problems with forced expiratory maneuvers as asthmatics. The airways of moderate to severe patients might present a strong tendency to collapse, which can produce sudden "amputations" of the expiratory tracing. Some laboratories report that the most severe patients might present signs of a reduced cardiac output at the end of prolonged and forced expirations, with symptoms of dizziness, lightheadedness, or even presyncope. This is probably due to the very long expiration time needed to empty the lungs entirely (6 s for forced expiratory maneuvers is the standard requirement) plus high intrathoracic pressures, both leading to reductions of venous return. Therefore, mood and willingness to cooperate can be affected in these patients.

Anxiety Disorders

Lung function parameters are of particular interest in studying anxiety disorders because of their association with dyspnea and possible hyperventilation (Carr, Lehrer, & Hochron, 1992; Roth, Wilhelm, & Trabert, 1998). Baseline lung function can differ from healthy individuals (e.g., Carr, Lehrer, Jackson, & Hochron, 1996). Body plethysmographic measurements involving the enclosure in a sealed cabin can be particularly problematic for anxiety patients. In addition, elevated levels of chest muscle tension in panic patient (Lynch, Bakal, Whitelaw, & Fung, 1991) could influence R_{os} and R_{int} measures that include resistance of the chest wall.

Vocal Cord Disorder

Patients with a functional vocal cord disorder could produce spurious increases in resistance measures by narrowing of the upper airway passage. Psychological factors such as anxiety or trauma have been linked to this syndrome (Gavin, Wamboldt, Brugman, Roesler, & Wamboldt, 1998; Selner, Staudenmeyer, Koepke, Harvey, & Christopher, 1987), and it is yet unknown whether qualitatively similar upper airway effects can be expected in healthy individuals in anxiety or stressful situations. Usually these effects are most apparent during inhalation, and the disorder is often diagnosed by abnormal inspiratory flow in spirometry tests. Some patients may also have additional pulmonary disease, such as asthma (Newman, Mason, & Schmaling, 1995).

Issues in Statistical Design and Data Analysis

The choice of study design is linked to varying importance of confounding variables in the analysis of lung function. Between-individuals designs with comparison of baseline assessments must take into account age, sex, height, or weight (see Instructional and Experimenter Effects). These factors are likely to contribute to a good short-and long-term stability of basal lung function measures, which is comparable to other commonly studied parameters such as heart rate (Ritz, Wiens, & Dahme 1998). They will play a minor role for studies involving only within-individual variation. However, between-individual comparisons of reactivity scores (arithmetic difference of task score minus baseline) can be compromised, if these confounding factors affect airway reactivity.

Stability of reactivity scores for basic tasks such as isometric arm exercise or voluntary deep breathing has been shown to be relatively low for R_{os}, and task repetitions would be needed to increase stability to an acceptable level (Ritz, Wiens, & Dahme, 1998). No comparable research is available with other measurement techniques or for more complex psychological tasks. The distribution of reactivity scores has been examined in several ways. Earlier research into the psychological impact on the airways has focused on a reactor–nonreactor dichotomy, based on varying standards of clinically significant change (Isenberg et al., 1992). However, although dichotomic analysis may be appropriate for clinical purposes, it may miss some of the psychophysiologically relevant variation. Research has revealed qualitatively similar response patterns to psychological tasks in patients and nonclinical groups (e.g., Lehrer et al., 1996; Ritz, Steptoe, et al., 2000), suggesting a continuum of responding from health to illness. Airway hyperreactivity as tested by methacholine or histamine provocation is known to be normally distributed (Cockroft, 1997). In search of underlying mechanisms of psychophysiological responsiveness of the airways, it is important to include the whole continuum of responding.

Both ceiling and floor effects have been discussed for reactivity values of lung function measurements (e.g., Chung, Morgan, Keyes, & Snashall, 1982; Freedman, 1992). Theoretically, a positive initial-value dependence of resistance changes can be expected: As resistance varies inversely proportional to the fourth power of the radius of a tube, the same amount of change in airway smooth muscle tone will necessarily lead to stronger increases in resistance in individuals with a smaller airway caliber (Benson, 1975). Thus, higher bronchial reactivity values are found in children compared to adults, and in patients with asthma or COPD compared to healthy individuals. However, the theoretical relationship holds only for fully established laminar flow. In physiology, the profiles are generally unknown and vary along the bronchial tree (laminar in the periphery, turbulent in the central airways during forced expiration). In general, estimates of the importance of geometric factors in reactivity measures vary (Sterk et al., 1993). Recommendations on reporting change scores exist for spirometry in bronchodilator tests (ERS, 1997). Future research has to examine baseline dependency of various techniques and indices, and the adequacy of correction or data transformation procedures.

Conclusion

The available research reflects the complexity of applying and interpreting measures of pulmonary function in psychophysiology. The evaluation of measurement techniques is an ongoing process. Knowledge on the relative contribution of different levels of the airways to obstructions measured by different techniques has to be advanced. The continuous control of upper airway artifacts by means of noninvasive methods remains a major challenge for validation studies. From a psychophysiological point of view, many areas of airway function remain unknown. Very little is known about the differential susceptibility of various indices of pulmonary function to psychological factors such as mood states, habits, or personality. More research using continuous measurement techniques is needed to explore effects of psychosocial demands on response parameters such as latency, rise time, peak duration, and recovery. Transient or phasic responses in health might be in contrast with a tonic character of responding in clinical groups (e.g., Levenson, 1979). No research has yet been reported on the differential sensitivity of components of resistance, such as thoracic resistance versus lung resistance, or different anatomic locations in the airways to psychological processes. Similarly, no research is available on the sensitivity of airway elastance to these processes, although autonomic nervous system influences on elastic properties have been known for quite some time (Woolcock, Macklem, Hogg, & Wilson, 1969). Where links to psychological processes become apparent, their putative biological function in the context of basic behavioral strategies remains to be investigated. Apart from an insight into basic organismic functioning, such research could further our understanding of pulmonary diseases, where patients' perceptions of symptoms, lung function, disease states, or resulting restrictions on everyday life can bear little association with actual pulmonary function. A whole plethora of potential research issues has been suggested (e.g., Busse et al., 1995; Lehrer, Isenberg, & Hochron, 1993), but actual research is still lagging behind. We hope that the present report can motivate and facilitate research efforts, and thereby contribute to a wider appreciation of these issues. Psychophysiology could ultimately contribute to greater success in prevention, therapy, self-management, and rehabilitation of pulmonary diseases. A more detailed understanding of psychological factors in lung function testing will be vital for progress in these fields. It has become clear that an implementation of these methods in the psychophysiological research setting requires specific considerations and cautions, and a more systematic investigation of these aspects is clearly needed.

REFERENCES

Aitken, R. C. (1969). Measurement of feelings using visual analogue scales. *Proceedings of the Royal Society of Medicine*, *62*, 989–993.

American Academy of Sleep Medicine Task Force. (1999). Sleep-related breathing disorders in adults: Recommendations for syndrome definition and measurement techniques in clinical research. *Sleep*, *22*, 667–689.

American Association for Respiratory Care. (1994). AARC clinical practice guideline. Body plethysmography. *Respiratory Care*, *39*, 830–836.

American Association for Respiratory Care. (1996). AARC clinical practice guideline. Spirometry, 1996 update. *Respiratory Care*, *41*, 629–636.

American Thoracic Society. (1991). Lung function testing: Selection of reference values and interpretative strategies. *American Review of Respiratory Disease*, *144*, 1202–1218.

American Thoracic Society. (1995). Standardization of Spirometry 1994 Update. Official statement of the ATS. *American Journal of Respiratory and Critical Care Medicine*, *152*, 1107–1136.

American Thoracic Society. (1999). Dyspnea. Mechanisms, assessment, and management: A consensus statement. *American Journal of Respiratory and Critical Care Medicine*, *159*, 321–340.

Bachofen, H. (1966). Lung tissue resistance in normal and asthmatic subjects. *Helvetica Medica Acta*, *33*, 108–121.

Barnas, G. M., Campbell, D. N., Mackenzie, C. F., Mendham, J. E., Fahy, B. G., Runcie, C. J., & Mendham, G. E. (1992). Lung, chest wall, and total respiratory system resistances and elastances in the normal range of breathing. *American Review of Respiratory Disease*, *145*, 110–113.

Barnes, P. J., & Thomson, N. C. (1992). Neural and humoral control. In P. J. Barnes, I. Rodger, & N. C. Thompson (Eds.), *Asthma* (pp. 343–359). London: Academic Press.

Bates, J. H. T., Daroczy, B., & Hantos, Z. (1992). A comparison of interrupter and forced oscillation measurements of respiratory resistance in dogs. *Journal of Applied Physiology*, *72*, 46–52.

Bates, J. H. T., Ludwig, M. S., Sly, P. D., Brown, K., Martin, J. G., & Fredberg, J. J. (1988). Interrupter resistance elucidated by alveolar pressure measurement in open-chest normal dogs. *Journal of Applied Physiology*, *65*, 408–414.

Benson, M. K. (1975). Bronchial hyperreactivity. *British Journal of Diseases of the Chest*, *69*, 227–239.

Bisgaard, H., & Klug, B. (1995). Lung function measurement in awake young children. *European Respiratory Journal*, *8*, 2067–2075.

Boiten, F., Frijda, N. H., & Wientjes, C. J. E. (1994). Emotions and respiratory patterns: Review and critical analysis. *International Journal of Psychophysiology*, *17*, 103–128.

Borg, G. (1970). Perceived exertion as an indicator of somatic stress. *Scandinavian Journal of Rehabilitation Medicine*, *2*, 92–98.

Boulet, L. P., Leblanc, L. P., & Turcotte, H. (1994). Perception scoring of induced bronchoconstriction as an index of awareness of asthma symptoms. *Chest*, *105*, 1430–1433.

Bridge, P. D., Lee, H., & Silverman, M. (1996). A portable device based on the interrupter technique to measure bronchodilator response in schoolchildren. *European Respiratory Journal*, *9*, 1368–1373.

Briscoe, W. A., & DuBois, A. B. (1958). The relationship between airway resistance, airway conductance and lung volume in subjects of different age and body size. *Journal of Clinical Investigation*, *37*, 1279–1285.

British Thoracic Society and the Association of Respiratory Technicians and Physiologists. (1994). Guidelines for measurement of respiratory function. *Respiratory Medicine*, *88*, 165–194.

Busse, W. W., Kiecolt-Glaser, J. K., Coe, C., Martin, R. J., Weiss, S. T., & Parker, S. R. (1995). NHLBI Workshop Summary: Stress and asthma. *American Journal of Respiratory and Critical Care Medicine*, *151*, 249–252.

Butler, J., Caro, C. G., Alcala, R., & DuBois, A. B. (1960). Physiological factors affecting airway resistance in normal subjects and in patients with obstructive respiratory disease. *Journal of Clinical Investigation*, *39*, 584–591.

Carr, R. E., Lehrer, P. M., & Hochron, S. M. (1992). Panic symptoms in asthma and panic disorder: A preliminary test of the dyspnea–fear theory. *Behavioral Research and Therapy*, *30*, 251–261.

Carr, R. E., Lehrer, P. M., Jackson, A., & Hochron, S. M. (1996). Effect of psychological stress on airway impedance in individuals with asthma and panic disorder. *Journal of Abnormal Psychology*, *105*, 137–141.

Carter, E. R., Stecenko, A. A., Pollock, B. H., & Jaeger, M. J. (1994). Evaluation of the interrupter technique for the use of assessing airway obstruction in children. *Pediatric Pulmonology*, *17*, 211–217.

Cauberghs, M., & Van de Woestijne, K. P. (1984). Comparison of two forced oscillation techniques. *Respiration*, *45*, 22–25.

Cauberghs, M., & Van de Woestijne, K. P. (1989). Effect of upper airway shunt and series properties on respiratory impedance measurements. *Journal of Applied Physiology*, *66*, 2274–2279.

Cauberghs, M., & Van de Woestijne, K. P. (1992). Changes of respiratory input impedance during breathing in humans. *Journal of Applied Physiology*, *73*, 2355–2362.

Chowienczyk, P. J., Lawson, C. P., Lane, S., Johnson, R., Wilson, N., Silverman, M., & Cochrane, G. M. (1991). A flow interruption device for measurement of airway resistance. *European Respiratory Journal*, *4*, 623–628.

Chung, K. F., Morgan, B., Keyes, S. J., & Snashall, P. D. (1982). Histamine dose–response relationships in normal and asthmatic subjects. *American Review of Respiratory Disease*, *126*, 849–854.

Clark, J. S., Votteri, B., Ariagno, R. L., Cheung, P., Eichhorn, J. H., Fallat, R. J., Lee, S. E., Newth, J. L., Rotman, H., & Sue, D. Y. (1992). Noninvasive assessment of blood gases. *American Review of Respiratory Disease*, *145*, 220–232.

Cockroft, D. W. (1997). Airway responsiveness. In P. J. Barnes, M. M. Grunstein, A. R. Leff, & A. J. Woolcock (Eds.), *Asthma* (Vol. 2, pp. 1253–1266). Philadelphia: Lippincott-Raven.

Committee of the Environmental and Occupational Health Assembly of the ATS. (1996). Health effects of outdoor air pollution. Part 1. *American Journal of Respiratory and Critical Care Medicine*, *153*, 3–50.

Cropp, G. J. A. (1979). The exercise bronchoprovocation test: Standardization of procedures and evaluation of response. *Journal of Allergy and Clinical Immunology*, *64*, 627–633.

Dab, I., & Alexander, F. (1976). A simplified approach to the measurement of specific airway resistance. *Pediatric Research*, *10*, 998–999.

Dahme, B., Richter, R., & Maß, R. (1996). Interoception of respiratory resistance in asthmatic patients. *Biological Psychology*, *42*, 215–229.

Davenport, P. W., & Revelette, W. R. (1996). Perception of respiratory mechanical events. In L. Adams and A. Guz (Eds.), *Respiratory sensation* (pp. 101–123). New York: Marcel Dekker.

Delacourt, C., Lorino, H., Fuhrman, C., Herve-Guillot, M., Reinert, P., Harf, A., & Housset, B. (2001). Comparison of the forced oscillation technique and the interrupter technique for assessing airway obstruction and its reversibility in children. *American Journal of Respiratory and Critical Care Medicine*, *164*, 965–972.

Desager, K. N., Cauberghs, M., & van de Woestijne, K. P. (1997). Two points calibration procedure of the forced oscillation technique. *Medical and Biological Engineering and Computing*, *35*, 752–756.

Douma, W. R., van der Mark, T. W., Folgering, H. T. M., Kort, E., Kerstjens, H. A. M., Koeter, G. H., Postma, D. S., & the Dutch CNSLD Study Group (1997). Mini-Wright peak flow meters are reliable after 5 years use. *European Respiratory Journal*, *10*, 457–459.

DuBois, A. B., Botelho, S. Y., Bedell, G. N., Marshall, R., & Comroe, J. H., Jr. (1956). A rapid plethysmographic method for measuring thoracic gas volume: A comparison with a nitrogen washout method for measuring functional residual capacity in normal subjects. *Journal of Clinical Investigation*, 35, 322–326.

DuBois, A. B., Botelho, S. Y., & Comroe, J. H., Jr. (1956). A new method for measuring airway resistance in man using a body plethysmograph: Values in normal subjects and in patients with respiratory disease. *Journal of Clinical Investigation*, *35*, 327–335.

DuBois, A. B., Brody, A. W., Lewis, D. H., & Burgess, F. (1956). Oscillation mechanics of lungs and chest in man. *Journal of Applied Physiology*, *8*, 587–595.

DuBois, A. B., & Van de Woestijne, K. P. (Eds.) (1969). Body plethysmography. In H. Herzog (Series Ed.), *Progress in respiration research* (Vol. 4). Basel: Karger.

Ducharme, F. M., Davis, G. M., & Ducharme, G. R. (1998). Pediatric reference values for respiratory resistance measured by forced oscillations. *Chest*, *113*, 1322–1328.

Ebi-Kryston, K. L. (1988). Respiratory symptoms and pulmonary function as predictors of 10-year mortality from respiratory disease, cardiovascular disease, and all causes in the Whitehall Study. *Journal of Clinical Epidemiology*, *41*, 251–260.

Eigen, H., Bieler, H., Grant, D., Christoph, K., Terrill, D., Heilman, D. K., Ambrosius, W. T., & Tepper, R. S. (2001). Spirometric pulmonary function in healthy preschool children. *American Journal of Respiratory and Critical Care Medicine*, *163*, 619–623.

European Respiratory Society. (1997). Peak expiratory flow: Conclusions and recommendations of a working party of the European Respiratory Society. *European Respiratory Journal*, *10* (Suppl. 24), 2S–8S.

Farré, R., Rotger, M., Marchal, F., Peslin, R., & Navajas, D. (1999). Assessment of bronchial reactivity by forced oscillation admittance avoids the upper airway artifact. *European Respiratory Journal*, *13*, 761–766.

Ferris, B. G., Mead, J., & Opie, L. H. (1964). Partitioning of respiratory flow resistance in man. *Journal of Applied Physiology*, *19*, 653–658.

Fisher, A. B., DuBois, A. B., & Hyde, R. W. (1968). Evaluation of the forced oscillation technique for the determination of resistance to breathing. *Journal of Clinical Investigation*, *47*, 2045–2057.

Fishman, A., Geiger, S. R., Macklem, P. T., & Mead, J. (1986). *The respiratory system. Vol. 3: Mechanics of breathing* (2nd ed.). Bethesda, MD: American Physiological Society.

Florin, I., Freudenberg, G., & Hollaender, J. (1985). Facial expressions of emotional and physiologic reactions in children with bronchial asthma. *Psychosomatic Medicine*, *47*, 383–392.

Folgering, H., van den Brink, W., Van Heeswijk, O., & van Herwaarden, C. (1998). Eleven peak flow meters: A clinical evaluation. *European Respiratory Journal*, *11*, 188–193.

Forster, R. E., DuBois, A. B., Briscoe, W. A., & Fisher, A. B. (1986). *The lung. Physiological basis of pulmonary function tests* (3rd ed.). Chicago: Year Book Medical Publishers.

Freedman, S. (1983). Design of respiratory circuits and spirometry. In G. Laszlo & M. F. Sudlow (Eds.), *Measurement in clinical respiratory physiology* (pp. 9–24). London: Academic Press.

Freedman, S. (1992). Exercise as a bronchodilator. *Clinical Science*, *83*, 383–389.

Fritz, G. K., Spirito, A., Yeung, A., Klein, R., & Freedman, E. (1994). A pictorial visual analog scale for rating severity of childhood asthma episodes. *Journal of Asthma*, *31*, 473–478.

Fritz, G. K., & Wamboldt, M. Z. (1998). Pediatric asthma: Psychosomatic interactions and symptom perception. In H. Kotses & A. Harver (Eds.), *Self-management of asthma* (pp. 195–230). New York: Marcel Dekker.

Fritz, G. K., Yeung, A., Wamboldt, M. Z., Spirito, A., McQuaid, E. L., Klein, R., & Seifer, R. (1996). Conceptual and methodologic issues in quantifying perceptual accuracy in childhood asthma. *Pediatric Psychology*, *21*, 153–173.

Gavin, L. A., Wamboldt, M., Brugman, S., Roesler, T. A., & Wamboldt, F. (1998). Psychological and family characteristics of adolescents with vocal cord dysfunction. *Journal of Asthma*, *35*, 409–417.

Gift, A. G. (1989). Validation of a vertical visual analogue scale as a measure of clinical dyspnea. *Rehabilitation Nursing*, *14*, 313–325.

Gimeno, F., van der Weele, T., Koeter, G. H., & van Altena, R. (1992). Forced oscillation technique. Reference values for total respiratory resistance obtained with the Siemens Siregnost FD5. *Annals of Allergy*, *68*, 155–158.

Godfrey, S. (1997). Exercise-induced asthma. In P. J. Barnes, M. M. Grunstein, A. R. Leff, & A. J. Woolcock (Eds.), *Asthma*. (Vol. 2, pp. 1105–1119). Philadelphia: Lippincott-Raven.

Gotshall, R. W., Mickleborough, T. D., & Cordain, L. (2000). Dietary salt restriction improves pulmonary function in exercise-induced asthma. *Medicine and Science in Sports and Exercise*, *32*, 1815–1819.

Grippi, M. A. (1995). *Pulmonary pathophysiology*. Philadelphia: JB Lippincott.

Guenard, H., Vieillefond, H., & Varene, P. (1977). Adaption of body plethysmography to the study of respiratory mechanics during muscular exercise. *Bulletin Europeen de Physiopathologie Respiratoire*, *13*, 399–407.

Han, J. N., Stegen, K., Cauberghs, M., & Van de Woestijne, K. P. (1997). Influence of awareness of the recording of breathing on respiratory pattern in healthy humans. *European Respiratory Journal*, *10*, 161–166.

Hantos, Z., Daróczy, B., Suki, B., Galgoczy, G., & Csendes, T. (1986). Forced oscillatory impedance of the respiratory system at low frequencies. *Journal of Applied Physiology*, *60*, 123–132.

Harm, D. L., Marion, R. J., Creer, T. L., & Kotses, H. (1985). Effects of instructions on pulmonary function values. *Journal of Asthma*, *22*, 289–294.

Harm, D. L., Marion, R. J., Kotses, H., Creer, T. L. (1984). Effects of subject effort on pulmonary function measures: A preliminary investigation. *Journal of Asthma*, *21*, 295–298.

Harver, A. (1994). Effects of feedback on the ability of asthmatic subjects to detect increases in the flow-resistive component to breathing. *Health Psychology*, *13*, 52–62.

Harver, A., & Mahler, D. A. (1990). The symptom of dyspnea. In D. A. Mahler (Ed.), *Dyspnea* (pp. 1–53). Mount Kisco, New York: Futura.

Harver, A., & Mahler, D. A. (1998). Perception of increased resistance to breathing. In H. Kotses and A. Harver (Eds.), *Self-management of asthma* (pp. 147–193). New York: Marcel Dekker.

Harver, A., Mahler, D. A., Schwartzstein, R. M., & Baird, J. C. (2000). Descriptors of breathlessness in healthy individuals: Distinct and separable constructs. *Chest*, *118*, 679–690.

Hellinckx, J., Cauberghs, M., De Boek, K., & Demedts, M. (2001). Evaluation of impulse oscillation system: Comparison with forced oscillation technique and body plethysmography. *European Respiratory Journal*, *18*, 564–570.

Hyatt, R. E., & Wilcox, R. E. (1961). Extrathoracic airway resistance in man. *Journal of Applied Physiology*, *16*, 326–330.

Hyland, M. E. (1990). The mood–peak flow relationship in adult asthmatics: A pilot study of individual differences and direction of causality. *British Journal of Medical Psychology*, *63*, 379–384.

Isenberg, S. A., Lehrer, P. M., & Hochron, S. (1992). The effects of suggestion and emotional arousal on pulmonary function in asthma: A review. *Psychosomatic Medicine*, *54*, 192–216.

Jackson, A. C., Milhorn, H. T., & Norman, J. R. (1974). A reevaluation of the interrupter technique for airway resistance measurements. *Journal of Applied Physiology*, *36*, 264–268.

Jaeger, M. J., & Otis, A. B. (1964). Measurement of airway resistance with a volume displacement body plethysmograph. *Journal of Applied Physiology*, *19*, 813–820.

Kerstjens, H. A. M., Rijcken, B., Schouten, J. P., & Postma, D. S. (1997). Decline of *FEV*1 by age and smoking status: Facts, figures, and fallacies. *Thorax*, *52*, 820–827.

Kessler, V., Mols, G., Bernhard, H., Haberthür, C., & Guttmann, J. (1999). Interrupter airway and tissue resistance: Errors caused by valve properties and respiratory system compliance. *Journal of Applied Physiology*, *89*, 1546–1554.

Kinsman, R. A., Luparello, T., O'Banion, K., & Spector, S. (1973). Multidimensional analysis of the subjective symptomatology of asthma. *Psychosomatic Medicine*, *35*, 250–267.

Klein, B. E., Moss, S. E., Klein, R., & Cruickshanks, K. J. (2001). Peak expiratory flow rate: Relationship to risk variables and mortality: The Wisconsin Epidemiologic Study of diabetic retinopathy. *Diabetes Care*, *24*, 1967–1971.

Klug, B., & Bisgaard, H. (1996). Measurement of lung function in awake 2–4-year-old asthmatic children during methacholine challenge and acute asthma. *Pediatric Pulmonology*, *21*, 290–300.

Kohlhäufl, M., Brand, P., Scheuch, G., Schuiz, H., Haussinger, K., &

Heyder, J. (2001). Impulse oscillometry in healthy nonsmokers and asymptomatic smokers: Effects of bronchial challenge with methacholine. *Journal of Aerosol Medicine*, *14*, 1–12.
Korn, V., Franetzki, M., & Prestele, K. (1979). A simplified approach to the measurement of respiratory impedance. *Progress in Respiration Research*, *11*, 144–161.
Kotses, H. (1998). Emotional precipitants of asthma. In H. Kotses & A. Harver (Eds.), *Self-management of asthma* (pp. 35–62). New York: Marcel Dekker.
Kotses, H., Rawson, J. C., Wigal, J. K., & Creer, T. L. (1987). Respiratory airway changes in response to suggestion in normal individuals. *Psychosomatic Medicine*, *49*, 536–541.
Kotses, H., Westlund, R., & Creer, T. L. (1987). Performing mental arithmetic increases total respiratory resistance in individuals with normal respiration. *Psychophysiology*, *24*, 678–682.
Lándsèr, F. J., Nagels, J., Demedts, M., Billiet, L., & Van de Woestijne, K. P. (1976). A new method to determine the frequency characteristics of the respiratory system. *Journal of Applied Physiology*, *41*, 101–106.
Lebecque, P., Desmond, K., Swartebroeckx, Y., Dubois, P., Lulling, J., & Coates, A. (1991). Measurement of respiratory system resistance by forced oscillation in normal children: A comparison with spirometric values. *Pediatric Pulmonology*, *10*, 117–122.
Lebowitz, M. D., Krzyzanowski, M., Quackenboss, J. J., & O'Rourke, M. K. (1997). Diurnal variation of *PEF* and its use in epidemiological studies. *European Respiratory Journal*, *10* (Suppl. 24), 49s–56s.
Lehrer, P. M., Hochron, S., Carr, R., Edelberg, R., Hamer, R., Jackson, A., & Porges, S. (1996). Behavioral task-induced bronchodilation in asthma during active and passive tasks: A possible cholinergic link to psychologically induced airway changes. *Psychosomatic Medicine*, *58*, 413–422.
Lehrer, P. M., Hochron, S. M., Mayne, T., Isenberg, S., Carlson, V., Lasoski, A. M., Gilchrist, J., Morales, D., & Rausch, L. (1994). Relaxation and music therapies for asthma among patients prestabilized on asthma medication. *Journal of Behavioral Medicine*, *17*, 1–24.
Lehrer, P. M., Isenberg, S., & Hochron, S. M. (1993). Asthma and emotion: A review. *Journal of Asthma*, *30*, 5–21.
Levenson, R. W. (1979). Effects of thematically relevant and general stressors on specificity of responding in asthmatic and nonasthmatic subjects. *Psychosomatic Medicine*, *41*, 28–39.
Lindemann, H. (1979). Body plethysmographic measurements in children with an accompanying adult. *Respiration*, *37*, 278–281.
Loveridge, B., West, P., Anthonisen, N. R., & Kryger, M. K. (1983). Single-position calibration of the respiratory inductance plethysmograph. *Journal of Applied Physiology: Respiration and Environmental. Exercise Physiology*, *55*, 1031–1034.
Lynch, P., Bakal, D. A., Whitelaw, W., & Fung, T. (1991). Chest muscle activity and panic anxiety: A preliminary investigation. *Psychosomatic Medicine*, *53*, 80–89.
Mahler, D. A., & Loke, J. (1989). The pulmonary function laboratory. *Clinics in Chest Medicine*, *10*, 129–134.
Manning, H. L., & Schwartzstein, R. M. (2001). Respiratory sensations in asthma: physiological and clinical implications. *Journal of Asthma*, *38*, 447–460.
Maß, R., Dahme, B., & Richter, R. (1993). Clinical evaluation of a respiratory resistance biofeedback training. *Biofeedback & Self-Regulation*, *18*, 211–223.
Maß, R., Harden, H., Leplow, B., Wessel, M., Richter, R., & Dahme, B. (1991). A device for functional residual capacity controlled biofeedback of respiratory resistance. *Biomedizinische Technik—Biomedical Engineering*, *36*, 79–85.
Mathé, A. A., & Knapp, P. H. (1971). Emotional and adrenal reactions to stress in bronchial asthma. *Psychosomatic Medicine*, *33*, 323–340.
McFadden, E. R., Jr. (1992). Heat and water exchange in human airways. *American Review of Respiratory Disease*, *146*, S8–S10.
McFadden, E. R., Luparello, T., Lyons, H. A., & Bleeker, E. (1969). The mechanism of action of suggestion in the induction of acute asthma attacks. *Psychosomatic Medicine*, *31*, 134–143.
McGrath, P. A. (1987). An assessment of children's pain: A review of behavioral, physiological and direct scaling techniques. *Pain*, *31*, 147–176.
McIlroy, M. B., Mead, J., Selverstone, N. J., & Radford, E. P. (1955). Measurement of lung tissue viscous resistance using gases of equal kinematic viscosity. *Journal of Applied Physiology*, *7*, 485–490.
McKerrow, C. B., & Rossiter, C. E. (1968). An annual cycle in the ventilatory capacity of men with pneumoconiosis and of normal subjects. *Thorax*, *23*, 340–349.
McQuaid, E. L., Fritz, G. K., Yeung, A., & Mansell, A. (1996). Resistive load detection in healthy school-aged children. *Pediatric Pulmonology*, *22*, 357–363.
Meessen, N. E. L, van den Grinten, C., Luijendijk, S., & Folgering, H. (1997). Breathing pattern during bronchial challenge in humans. *European Respiratory Journal*, *10*, 1058–1063.
Merkus, P. J., Mijnsbergen, J. Y., Hop, W. C., & de Jongste, J. C. (2001). Interrupter resistance in preschool children: Measurement characteristics and reference values. *American Journal of Respiratory and Critical Care Medicine*, *163*, 1350–1355.
Meyer, R., Kröner-Herwig, B., & Spörkel, H. (1990). The effect of exercise and induced expectations on visceral perception in asthmatic patients. *Journal of Psychosomatic Research*, *34*, 455–460.
Michaelson, E. D., Grassman, E. D., & Peters, W. R. (1975). Pulmonary mechanics by spectral analysis of forced random noise. *Journal of Clinical Investigation*, 56, 1210–1230.
Michels, A., Decoster, K., Derde, L., Vleurinck, C., & Van de Woestijne, K. P. (1991). Influence of posture on lung volumes and impedance of respiratory system in healthy smokers and nonsmokers. *Journal of Applied Physiology*, *71*, 294–299.
Mullen, J. B. M., Wiggs, B. R., Wright, J. L., Hogg, J. C., & Paré, P. D. (1986). Nonspecific airway reactivity in cigarette smokers. *American Review of Respiratory Disease*, *133*, 120–125.
Nadel, J. A., & Tierney, D. F. (1961). Effect of a previous deep inspiration on airway resistance in man. *Journal of Applied Physiology*, 16, 717–719.
National Heart, Lung, and Blood Institute. (1997). *Expert panel report 2: Guidelines for the diagnosis and management of asthma*. National Asthma Education and Prevention Program. Washington, DC: U.S. Department of Health and Human Services.
Newman, K. B., Mason, U. G., III, & Schmaling, K. B. (1995). Clinical features of vocal cord dysfunction. *American Journal of Respiratory and Critical Care Medicine*, *152*, 1382–1386.
Nicholson, K. G., Kent, J., & Ireland, D. C. (1993). Respiratory viruses and exacerbation of asthma in adults. *British Medical Journal*, *307*, 982–986.
Orehek, J., Gayrard, P., Grimaud, C., & Charpin, J. (1975). Effect of maximal respiratory manoeuvres on bronchial sensitivity of asthmatic patients as compared to normal people. *British Medical Journal*, *1*(5950), 123–125.
Oswald-Mammosser, M., Llerena, C., Speich, J. P., Donata, L., & Lonsdorfer, J. (1997). Measurements of respiratory system resistance by the interrupter technique in healthy and asthmatic children. *Pediatric Pulmonology*, *24*, 78–85.
Pearlman, D. S., & Lemanske, R. F. (1996). Asthma (bronchial asthma): Principles of diagnosis and treatment. In C. W. Bierman, D. S. Pearlman, G. G. Shapiro, & W. W. Busse (Eds.), *Allergy, asthma and immunology from infancy to adulthood* (3rd ed., pp. 484–497). Philadelphia: Saunders.
Pengelly, L. D., Rebuck, A. S., & Campbell, E. J. M. (Eds.). (1974). *Loaded breathing*. Edinburgh: Churchill Livingstone.
Peslin, R., Duvivier, C., Didelon, J., & Gallina, C. (1985). Respiratory impedance measured with head generator to minimize upper airway shunt. *Journal of Applied Physiology*, *59*, 1790–1795.
Phagoo, S. B., Watson, R. A., Pride, N. B., & Silverman, M. (1993). Accuracy and sensitivity of the interrupter technique for measuring the response to bronchial challenge in normal subjects. *European Respiratory Journal*, *6*, 996–1003.
Phagoo, S. B., Watson, R. A., Silverman, M., & Pride, N. B. (1995). Comparison of four methods of assessing airflow resistance before and after induced airway narrowing in normal subjects. *Journal of Applied Physiology*, *79*, 518–525.
Quanjer, P. H., Stocks, J., Polgar, G., Wise, M., Karlberg, J., & Borsboom, G. (1989). Compilation of reference values for lung function measurements in children. *European Respiratory Journal*, *2*(Suppl. 4), 184s–261s.
Quanjer, P. H., Tammeling, G. J., Cotes, J. E., Pedersen, O. F., Peslin, R., & Yernauld, J.-C. (1993). Lung volumes and forced ventilatory flows. Report working party. Standardization of lung function tests. European Community for Steel and Coal. *European Respiratory Journal*, *6* (Suppl. 16), 5–40.
Richardson, J. B. (1988). Innervation of the lung. In M. A. Kaliner & P. J. Barnes (Eds.), *The airways, neural control in health and disease*. New York: Marcel Dekker.

Rigau, J., Farre, R., Roca, J., Marco, S., Herms, A., & Navajas, D. (2002). A portable forced oscillation device for respiratory home monitoring. *European Respiratory Journal*, *19*, 146–150.
Ritz, T., Dahme, B., & Wagner, C. (1998). Effects of static forehead and forearm muscle tension on total respiratory resistance in healthy and asthmatic subjects. *Psychophysiology*, *35*, 549–562.
Ritz, T., George, C., & Dahme, B. (2000). Respiratory resistance during emotional stimulation: Evidence for a nonspecific effect of experienced arousal? *Biological Psychology*, *52*, 143–160.
Ritz, T., & Steptoe, A. (2000). Emotion and pulmonary function in asthma: Reactivity in the field and relationship with laboratory induction of emotion. *Psychosomatic Medicine*, *62*, 808–815.
Ritz, T., Steptoe, A., De Wilde, S., & Costa, M. (2000). Emotions and stress increase respiratory resistance in asthma. *Psychosomatic Medicine*, *62*, 401–412.
Ritz, T., Wiens, S., & Dahme, B. (1998). Stability of total respiratory resistance under multiple baseline conditions, isometric arm exercise and voluntary deep breathing. *Biological Psychology*, *49*, 187–213.
Roth, W. T., Wilhelm, F. H., & Trabert, W. (1998). Voluntary breath holding in panic and generalized anxiety disorders. *Psychosomatic Medicine*, *60*, 617–679.
Rubinfeld, A. F., & Pain, M. C. F. (1976). Perception of asthma. *Lancet*, *1*, 882–884.
Saisch, S. G. N. (1994). An introduction to capnography. *Biofeedback and Self-Regulation*, *19*, 115–134.
Selner, J. C., Staudenmeyer, H., Koepke, J. W., Harvey, R., & Christopher, K. (1987). Vocal cord dysfunction: The importance of psychological factors and provocation challenging testing. *Journal of Allergy and Clinical Immunology*, *79*, 726–733.
Simon, B. A., Zanaboni, P. B., & Nyhan, D. P. (1997). Effect of hypoxia on respiratory system impedance in dogs. *Journal of Applied Physiology*, *83*, 451–458.
Sly, P. D., & Bates, J. H. T. (1988). Computer analysis of physical factors affecting the use of the interrupter technique in infants. *Pediatric Pulmonology*, *4*, 219–224.
Stein, M. (1962). Etiology and mechanisms in the development of asthma. In J. H. Nodine & J. H. Moyer (Eds.), *Psychosomatic medicine. The first Hahnemann symposium* (pp. 149–156). London: Henry Kimpton.
Sterk, P. J., Fabbri, L. M., Quanier, P. H., Cockroft, D. W., O'Byrne, P. M., Anderson, S. D., Juniper, E. F., & Malo, J.-L. (1993). Airway responsiveness. Standardized challenge testing with pharmacological, physical and sensitizing stimuli in adults. *European Respiratory Journal*, *6*(Suppl. 16), 53–83.
Stocks, P. D., Sly, R. S., Tepper, W. J., & Morgan, W. J. (1996). *Infant respiratory function testing*. New York: Wiley.
Stout, C., Kotses, H., & Creer, T. L. (1997). Improving perception of air flow obstruction in asthma patients. *Psychosomatic Medicine*, *59*, 201–206.
Tomalak, W., Peslin, R., & Duvivier, C. (1998). Variations in airways impedance during respiratory cycle derived from combined measurements of input and transfer impedances. *European Respiratory Journal*, *12*, 1436–1441.
Truelsen, T., Prescott, E., Lange, P., Schnohr, P., & Boysen, G. (2001). Lung function and risk of fatal and non-fatal stroke. The Copenhagen City Heart Study. *International Journal of Epidemiology*, *30*, 145–151.
van Aalderen, W. M. C., Postma, D. S., Koeter, G. H., & Knol, K. (1989). Circadian change in bronchial responsiveness and airflow obstruction in asthmatic children. *Thorax*, *44*, 803–807.
van Altena, R., & Gimeno, F. (1994). Respiratory resistance measured by flow-interruption in a normal population. *Respiration*, *61*, 249–254.
Van de Woestijne, K. P., Desager, K. N., Duiverman, E. J., & Marchal, F. (1994). Recommendations for measurements of respiratory input impedance by means of the forced oscillations method. *European Respiratory Review*, *4*, 235–237.
van den Elshout, F. J. J., van Herwaarden, C. L. A., & Folgering, H. T. M. (1991). Effects of hypercapnia and hypocapnia on respiratory resistance in normal and asthmatic subjects. *Thorax*, *46*, 28–32.
van Noord, J. A., Clément, J., Van de Woestijne, K. P., & Demedts, M. (1989). Total respiratory resistance and reactance as a measurement of response to bronchial challenge with histamin. *American Review of Respiratory Disease*, *139*, 921–926.
van Noord, J. A., Clément, J., Van de Woestijne, K. P., & Demedts, M. (1991). Total respiratory resistance and reactance in patients with asthma, chronic bronchitis and emphysema. *American Review of Respiratory Disease*, *143*, 922–927.
van Noord, J. A., Smeets, J., Clément, J., Van de Woestijne, K. P., & Demedts, M. (1994). Assessment of reversibility of airflow obstruction. *American Journal of Respiratory and Critical Care Medicine*, *150*, 551–554.
von Leupoldt, A., & Dahme, B. (2000). Induced emotions and airway resistance—Measuring the respiratory impact of emotion laden movie clips by whole body plethysmography. (Abstract) *Biological Psychology*, *53*, 88.
von Neergaard, J., & Wirz, K. (1927). Die Messung der Strömungswiderstände in den Atemwegen des Menschen, insbesondere bei Asthma und Emphysem. *Zeitschrift für Klinische Medizin*, *105*, 51–82.
Warren, J. B., Jennings, S. J., & Clark, T. J. H. (1984). Effect of adrenergic and vagal blockade on normal human airway response to exercise. *Clinical Science*, *66*, 79–85.
West, J. B. (1990). *Respiratory physiology*. Baltimore: Williams & Wilkins.
Wientjes, C. J. E. (1992). Respiration in psychophysiology: Measurement issues and applications. *Biological Psychology*, *34*, 179–203.
Wilhelm, F. H., & Roth, W. T. (1996). Ambulatory assessment of clinical anxiety. In J. Fahrenberg & M. Myrtek (Eds.), *Ambulatory assessment. Computer-assisted psychological and psychophysiological methods in monitoring and field studies* (pp. 317–355). Seattle: Hogrefe & Huber.
Woolcock, A. J., Macklem, P. T., Hogg, J. C., & Wilson, N. J. (1969). Influence of autonomic nervous system on airway resistance and elastic recoil. *Journal of Applied Physiology*, *6*, 814–818.
Worth, H., Jacobi, N., & Muenster, R. (1987). Messung der Atemwegsobstruktion bei unspezifischer inhalativer Provokation mittels Spirometrie, Bodyplethysmographie, Oszillometrie und Verschlussdruckmethode. [Measurement of airway obstruction in unspecific inhalatory provocation via spirometry, body plethysmography, oscillometry and the occlusion pressure method.] *Praxis und Klinik der Pneumologie*, *41*, 512–513.
Zapletal, A., Samanek, M., & Paul, T. (1987). Lung function in children and adolescents. Methods, reference values. In H. Herzog (series Ed.), *Progress in respiration research* (Vol. 22). Basel: Karger.
Zechman, F. W., Jr., & Wiley R. L. (1986). Afferent inputs to breathing: Respiratory sensation. In N. S. Cherniack & J. G. Widdicombe (Eds.), *Handbook of physiology: Section 3. The respiratory system* (Vol. II, pp. 449–474). Bethesda, MD: American Physiological Society.
Zureik, M., Kauffmann, F., Touboul, P. J., Courbon, D., & Ducimetiere, P. (2001). Association between peak expiratory flow and development of carotid atherosclerotic plaques. *Archives of Internal Medicine*, *161*, 1669–1676.

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APPENDIX

Appendix A1 shows abbreviations, symbols and units for lung-function testing.

Appendix A1. *Abbreviations, Symbols and Units for Lung-Function Testing*

Abbreviation, symbol	Unit[a,b]	Description
BTPS		Body temperature, barometric pressure and saturated with water vapor
C	l/kPa	Compliance
C_{rs}	l/kPa	Respiratory system compliance
E	kPa/l	Elastance

Appendix A1. *Continued*

Abbreviation, symbol	Unit[a,b]	Description
$FEF_{25–75\%}$	l/s	Mean expiratory flow during the middle half of the forced expiratory vital capacity (see also *MMEF*)
FEV_1	l	Forced expiratory volume in the 1st second
FEV_1/VC	%l	FEV_1 as a percentage of the vital capacity (coefficient of Tiffeneau)
FOT		Forced oscillation technique
FRC	l	Functional residual capacity
FVC	l	Forced expiratory vital capacity
G	$l \cdot s^{-1} \cdot kPa^{-1}$	Conductance
G_{aw}	$l \cdot s^{-1} \cdot kPa^{-1}$	Airway conductance
sG_{aw} (G_{aw}/TGV)	$s^{-1} \cdot kPa^{-1}$	Specific airway conductance
G_{int}	$l \cdot s^{-1} \cdot kPa^{-1}$	Conductance measured by the interrupter technique
I	$kPa \cdot l^{-1} \cdot s^2$	Inertance
I_{rs}	$kPa \cdot l^{-1} \cdot s^2$	Respiratory system inertance
MEF	l/s	Maximal expiratory flow (see also V'_{max})
$MEF_{x\%FVC}$	l/s	Maximal expiratory flow when x% of *FVC* remains to be exhaled
MIF	l/s	Maximal inspiratory flow
MMEF	l/s	Maximal midexpiratory flow (see also $FEF_{25–75\%}$).
PEF	l/min (or l/s)	Peak expiratory flow
P	kPa	Pressure
P_{alv}	kPa	Alveolar pressure (also P_A)
P_B	kPa	Barometric pressure
PC_{20}	mg/ml	Provocative concentration of bronchoconstrictor causing FEV_1 to fall by 20%
P_{box}	kPa	Box pressure, cabin pressure of the body plethysmograph
P_{dyn}	kPa	Dynamic pressure, pressure used to generate flow or volume acceleration
P_{H2O}	kPa	Pressure of water vapor
P_L	kPa	Transpulmonary pressure
P_{mo}	kPa	Mouth pressure
P_{rs}	kPa	Respiratory system pressure
P_{stat}	kPa	Static pressure, pressure used to displace volume
P_w	kPa	Transthoracic pressure
P_{tot}	kPa	Total lung inflation pressure ($P_{stat} + P_{dyn}$)
PRN		Pseudo-random noise, method relevant to FOT
R	$kPa \cdot l^{-1} \cdot s$	Resistance
R_{aw}	$kPa \cdot l^{-1} \cdot s$	Airway resistance
R_{int}	$kPa \cdot l^{-1} \cdot s$	Resistance measured by the interrupter technique
R_{os}	$kPa \cdot l^{-1} \cdot s$	Impedance measured by single frequency FOT at 10 Hz (P_{mo} calibrated in resistance units)
R_{rs}	$kPa \cdot l^{-1} \cdot s$	Respiratory resistance, resistance of the total respiratory system[c]
RV	l	Residual volume
T_E	s	Expiratory time; duration of expiration
T_I	s	Inspiratory time; duration of inspiration
T_{TOT}	s	Total respiratory cycle time; duration of the total breathing cycle
TGV	l	Thoracic gas volume (also V_{tg})
V	l (or ml)	Gas volume
V'	l/s	Flow[d]
V''		Volume acceleration[d]
V_L	l (or ml)	Lung gas volume
VAS		Visual analog scale, for ratings of dyspnea
VC	l	Vital capacity
V'_{max}	l/s	Maximal expiratory flow (see also MEF)
V_T	l (or ml)	Tidal volume
X		Reactance
X_{rs}		Reactance of the respiratory system
Z	$kPa \cdot l^{-1} \cdot s$	Impedance
Z_{rs}	$kPa \cdot l^{-1} \cdot s$	Impedance of the respiratory system

[a]Divisions are sometimes used in complex expressions of physical units, for example, kPa/l/s for resistance, but this is discouraged in recent guidelines. One division is acceptable, that is, l/s for flow, alternatively $l \cdot s^{-1}$.
[b]In general, SI units are recommended. For pressure, millimeters of mercury (mmHg) or centimeters of water (cm H_2O) are sometimes used, with 1 mmHg = 133.322 Pa and 1 cm H_2O = 98.066 Pa.
[c]R_{tot} or TRR were used in previous psychophysiological texts for total respiratory resistance, but are not recommended here.
[d]In American recommendations, $\dot{V}$ and $\ddot{V}$ are used for V' and V''.