Is Asthma a Psychosomatic Illness?

In asthma the bronchial muscle is hyperirritable, making asthmatics susceptible to a wide variety of external and endogenous trigger factors normally experienced by us all. The level of hyperirritability determines the risk of developing bronchospasm on exposure to the trigger factors.

Bronchial muscle tone is in part maintained by vagal nerves. Emotional factors can act as triggers for bronchospasm through these nerves, particularly in patients with the greatest hyperirritability. Asthma itself may arouse hostilities in parents, peers, and therapists. By definition asthma can therefore be considered psychosomatic, but because of current usage, this term does not help good medical management. The complex interplay between the organic abnormality in asthma and the psychosocial environment should always be carefully considered.

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This article is based on a paper presented at the Australian Physiotherapy Association National Congress, Adelaide, May, 1983

All who have suffered even partial obstruction of any portion of the respiratory tract know the atavistic fears of suffocation that arise. Perhaps this is related to primitive superstition and religious beliefs, clearly stated in no lesser authority than the Old Testament: 'Then the Lord God formed man of the dust of the ground, and breathed into his nostrils the breath of life and man become a living soul' (Genesis 2:7). Small wonder that physiological disturbances of the 'breath of life' call forth dramatic symptoms and signs.

Is asthma a psychosomatic disease? Perhaps we should carefully explore the current state of knowledge before giving an over-hasty answer.

Psychosomatic Disease

The Oxford English Dictionary states that a psychosomatic disorder is one involving or depending on both the mind and the body as mutually dependent entities. Psychosomatic medicine has developed the reputation of considering somatic disease as being psychogenic, since Descartes separated the mind from the machine-like body (Wilson 1978). Alexander further developed the theory of psychogenesis for many physical disorders (Alexander 1950). Indeed, Engel (1962) proposed that individuals born with a genetically-determined predisposition to a disease will have their psychological development influenced by this genetic 'abnormality' and will thereby develop a set of personality characteristics. If these characteristics do not allow the individual to cope with a given stress, the disease will become overt. Though the disease is demonstrably a physical disorder, psychological factors appear to play an important aetiological role.

As an extension of this concept, Halliday (1943) proposed a psychosomatic formula which included: (i) specific personality type, (ii) emotion as a precipitating factor, (iii) disproportionate sex ratio, (iv) association with other psychosomatic affections, (v) family history of the same or associated disorder, and (vi) phasic manifestations.

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Specific Personality Type in Asthma

Neuhaus (1958) found no specific personality type occurring among asthmatic children, but chronically disabled children in general were 'over-dependent'. Wittkower and White (1959) found from a study of ten patients that there were strong feelings of ambivalence towards the mother who was portrayed as rigid and domineering yet insecure, depriving the child of maternal love. In a larger study, Rees (1963) found a high incidence of parental over-protectiveness which antedated the asthma. An over-protected child may become over-dependent and introspective, and so be unable to cope adequately with bronchial narrowing. High dependency levels have been shown to be associated causally with high rates of re-hospitalization for asthma (Dirks *et al* 1977a and 1977b, Staudenmayer *et al* 1979). However, it has always been difficult to prove that parental attitudes or the patient's personality type are true aetiological factors rather than consequences of the disease.

Emotions as Precipitating Factors

Medical opinion generally holds that emotional factors are not aetiologically important. However, there is considerable evidence for a significant trigger role by the emotions in a pre-existing asthmatic state. Suggestion to an asthmatic patient that either bronchodilation or bronchoconstriction will be produced by a dose of saline or other placebo, often results in the suggested change (McFadden *et al* 1969, Luparello *et al* 1970, Spector *et al* 1976, Horton *et al* 1978). Rather than accept the term Emotional Asthma it is better to consider that there is an abnormally sensitive state of bronchial smooth muscle susceptible to any trigger, including the emotions.

Sex Ratio

In children, asthma is more common in males (male:female ratio = 2:1), but the ratio is closer to unity in adult asthmatics (Norman 1974). There appears to be no evidence for a sex ratio of emotionally-triggered asthma that differs from the normal sex ratio in asthma (Maher-Loughnan *et al* 1962).

Psychosomatic Disease Association

Hypertension, coronary artery disease, urticaria, psoriasis, hyperventilation syndrome, and migraine are relatively common. They would therefore be expected to occur quite frequently in asthmatics. This cannot be taken to imply a specific association on the basis of a common psychosomatic aetiology. There has been no reported increase in prevalence of any of these disorders in asthmatics, except when asthma is caused by therapy (*eg* beta-adrenergic antagonists used in hypertension and coronary artery disease), or by hyperventilation syndrome, or is associated with other allergic disorders (*eg* eczema and urticaria).

Family History and Phasic Manifestations

These other criteria in Halliday's formula are important features in asthma. However, as will be described, they seem to be related more to the physical than to any psychological disturbances.

Physiological Abnormalities in Asthma

The cause of asthma is unknown. However, most asthmatics display a bronchial hyperirritability. They may therefore develop episodic wheeze, cough, or dyspnoea on contacting certain atmospheric conditions or inhaled particles to which non-asthmatics react mildly or not at all. Allergens are substances, like grass pollen, animal danders, or housedust mites, to which the patient is allergic or sensitized and which can act as triggers. Non-allergic trigger factors include dusts, smoke, cold air, strong smells, exertion, industrial chemicals, and chemicals which may normally exist in the body. An allergic person develops an asthma attack only if the bronchi have an underlying hyper-responsiveness, or if the allergen load is overwhelming (Cockcroft *et al* 1979). The risk of developing allergen-induced bronchospasm, exercise-induced bronchospasm, or bronchospasm related to any of the other non-specific agents, is highest in those asthmatics with the greatest degree of bronchial hyper-responsiveness (Hargreave *et al* 1981, Dolovich and Hargreave 1981). Patients with high levels of responsiveness tend to have most symptoms (Makino 1966) and to need more treatment (Cockcroft *et al* 1977, Juniper *et al* 1981).

These findings are based on laboratory tests in which asthma is deliberately provoked by exercise, by hyperventilation, by allergen inhalation, or by inhalation of chemicals like histamine or methacholine (Cockcroft *et al* 1977, Eggleston 1979, Cockcroft *et al* 1979, Salome *et al* 1980, Deal *et al* 1980, Hargreave *et al* 1981). Other tests which may be useful are the response to bronchodilators and the variability in lung function tests, such as thrice daily domiciliary peak flow measurements. All of these are interrelated (Hargreave *et al* 1981, Frith *et al* 1982), but the histamine or methacholine inhalation tests appear to be the most sensitive (Frith *et al* 1982).

The reasons why asthmatics have bronchi that are hyper-responsive to normal everyday stimuli are unknown. Using evidence from individual cell studies, smooth muscle preparations, animal models, and inhalation tests, a number of theories have been evolved.

In asthma the factors contributing most to airways obstruction are mucosal oedema, mucus hypersecretion with bronchial plugging, and smooth muscle spasm with or without hypertrophy (Dunnill *et al* 1969). These three abnormalities have been demonstrated mainly in post mortem studies of patients dying with severe asthma. In well-controlled asthmatic patients not exposed to allergen or

infections it has not been possible to determine the main factor determining the spontaneously-variable airway calibre. However, because the onset of airways obstruction in inhalation provocation studies is so rapid, it seems most reasonable to attribute the tendency to airways obstruction to smooth muscle spasm.

The mechanisms involved in the control of bronchial smooth muscle appear to require the interactions of nerves, secretory cells, circulating chemicals, various antibodies, and smooth muscle cells

Nerves

There are irritant nerve endings within the epithelial lining of the bronchi with connections to the vagus nerve (Widdicombe 1977). From pulmonary ganglia, afferent fibres pass up the vagi to the brain stem, and from thence back to the ganglia, then to bronchial smooth muscle and mucosal glands. There appear to be two main types of innervation of the smooth muscle, namely a non-adrenergic inhibitory system, stimulating relaxation, and a cholinergic system, stimulating contraction (Richardson *et al* 1976). It should therefore be possible to block bronchospasm induced by irritant substances or irritating conditions with anti-cholinergic agents, as has been shown with hyper-responsiveness developing with viral infections (Empey *et al* 1976). It is conceivable that excessive vagal discharge in a normal patient, or normal degrees of vagal discharge in the presence of primary muscle over-sensitivity could produce bronchospasm. Any effects of the emotions could be mediated in this way. Support for this idea has come from a study showing that asthmatics suffering exacerbations as a consequence of emotional triggers can achieve significant relief with an anticholinergic agent (Rebuck and Marcus 1979).

While there is considerable evidence for the role of the parasympathetic vagus in bronchomotor tone, the neuromediator for the inhibitory system has not been isolated, so no blocking studies have been performed. It is possible, though, that in Rapid Eye Movement (R.E.M.) sleep, when dreaming occurs, excessive activity of the parasympathetic system, or underactivity of the inhibitory system could result in bronchospasm. It is a well-recognized feature of poorly controlled asthma that nocturnal wheezing episodes occur. These are related more to sleep than to time of day, posture, cold air, or allergens, and may be linked to R.E.M. sleep (Clark and Hetzel 1977).

Secretory Cells and Chemicals

Bronchial epithelial cells are linked by impermeable Tight Junctions. These can become abnormally permeable after smoke, allergen, histamine, or methacholine inhalation (Boucher *et al* 1979, Boucher *et al* 1978, Johnson *et al* 1978). Following this increased permeability, irritant or directly bronchoconstrictor substances would have improved access to both irritant nerve endings and bronchial smooth muscle leading to asthmatic bronchospasm.

About 30-40 per cent of the population are allergic; that is, they produce specific, highly reactive IgE antibodies to common substances. Most cells can be found in the mucosa and lumen of bronchi, and in allergic (or atopic) individuals they bear specific IgE antibodies to common inhaled substances like grass or housedust mite, so they are well placed to cause a local allergic reaction. The combination of specific IgE and allergen at the surface of circulating basophils or tissue-fixed mast cells results in release of histamine, prostaglandins and leukotrienes.

Histamine causes bronchoconstriction through stimulation of specific histamine receptors on bronchial smooth muscle, as well as mucosal gland secretion. While histamine acts immediately, prostaglandins and leukotrienes produce bronchoconstriction and inflammation more slowly, probably also through specific cell receptors. These time differences may account for the phenomena of early and late asthmatic reactions to allergen. The late reaction occurs 4 to 8 hours after exposure to the allergen, and so may appear at night in the case of grass pollen or industrial chemical allergy. The late reaction may itself induce even greater histamine hyperactivity (Cartier *et al* 1983), which probably explains the worsening and the nocturnal symptoms of asthma occurring in summer in grass pollen-sensitive patients.

Mast cells may also be 'degranulated' by physical trauma, temperature changes, and certain chemicals. There are cholinergic receptors on their surface, so they may release mediators in the presence of acetylcholine. Circulating adrenaline may prevent degranulation via beta-receptors. It now appears that hyperventilation and heat/water exchange across the bronchial mucosa contribute to mast cell release of mediators to produce bronchospasm. Exercise, hyperventilation, temperature change, osmolarity change, or physical trauma may result in bronchospasm. Thus, if the attainment of physical fitness results in less hyperventilation for a given level of exercise, less bronchospasm is likely.

Psychology and Physiology

Like many other diseases, asthma should be considered as a disease of the whole body. 'It is more important to know what sort of person has a disease than to know what sort of disease a person has' (Hippocrates). The position of the patient in his or her environment in both a physical and a psychosocial sense should be considered of great importance in the overall management of the patient with asthma. Just as the external physical environment (cold air, smoke, allergens, infections) can be responsible for triggering the hypersensitive bronchial muscle to constrict and cause asthmatic symptoms, so the internal environment (chemical, hormonal, neurological, or psychological)

may also be responsible for the same symptoms. If it is accepted that the central abnormality in asthma is hypersensitivity of the smooth muscle, it can be easily appreciated that emotional perturbations can precipitate an attack of asthma. The greater the degree of hypersensitivity of the muscle, the smaller the degree of psychological disturbance required to trigger the attack. Similarly, in a highly emotionally labile individual, vast swings of emotion may precipitate asthma even in patients with very mild bronchial hypersensitivity. Whether this is mediated directly through cholinergic nerves stimulating the smooth muscle, through hormonal changes, or through release of mediators from mast cells via a neurological feed-back mechanism is undetermined.

The possibility that an inborn error of smooth muscle can produce, of itself, psychological abnormalities is difficult to prove or disprove. Much work has been done over the years to show the effects of varying emotions on gastric circulation and acid production but it is impossible to be as invasive in the bronchi. However, all who handle patients with asthma recognize the occasional patient who uses his or her already developed bronchial hyperactivity for secondary gain. The patient who since childhood has been over-protected often is preyed upon by the stresses of the outside world, and his reactions to these external stresses may manifest as asthma. The child who was brought up to deny his symptoms is most likely to put off treating his symptoms of developing bronchospasm until they are so severe as to require hospitalization and corticosteroid therapy.

The tensions which exist in every home, and in every job, include both physical and psychological stresses. An asthmatic working in a dusty environment is more likely to develop symptoms. An asthmatic living in unstable family surrounds may also be more likely to have symptomatic asthma. But it is not the physical or the psychological stresses that produce asthma; it is the presence of bronchial smooth muscle hypersensitivity.

While attempts are occasionally made to remove an asthmatic from an allergenic environment, or a physically irritating environment, in practice it is almost impossible to so isolate a person. It is also difficult to isolate the asthmatic from infections. In the same way it may be hard to insulate the asthmatic from all emotional and psychological stresses. However, by adopting a holistic approach we may achieve better control of asthmatic symptoms. We should try not only to protect against allergens, to eradicate bronchial infections, or to treat pharmacologically with bronchial relaxants, but we should also attempt to improve potential psychosocial trigger factors. We should not consider dividing asthma into psychogenic, allergic, exercise-induced, or infective. We should rather consider the patient as an individual with his own individual reactions to the different trigger factors, his own individual exposure to allergic and infective agents, and with his own social and psychological pecularities.

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