

PSYCHOSOMATIC ASPECTS OF BRONCHIAL ASTHMA



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Psychosomatic Aspects of Bronchial Asthma¹

Introduction

Observations bearing on the role of psychological factors in bronchial asthma have a long history. Hippocrates allegedly said “The asthmatic must guard against anger.” Distinguished clinicians in the 18th century contributed anecdotal evidence about the role of emotions in precipitating or aggravating the disorder. As recently as 1971 a critical review of respiratory function in asthma remarked that “many asthmatic persons are somewhat unstable and it is admitted that the course of the disease may be affected by emotional or environmental factors.” In the late 1930s, a group of psychiatric clinicians, most of them psychoanalytically inspired, along with physiologists and other basic scientists, turned their attention to a group of chronic diseases of unknown etiology. Bronchial asthma was an early object of this psychosomatic scrutiny. The monograph on asthma by French and Alexander reported a series of twenty-six cases treated by psychoanalysis, and surveyed the psychosomatic evidence then extant. Since that time the continuously expanding number of reports has been reviewed by Leigh, Freeman et al., and Kelly.

Various psychosomatic theories have been proposed, the best known of which is the thesis that asthma represents a “suppressed cry for the mother,”

originally stated by E. Weiss and elaborated by French and Alexander. Such views have seldom been rigorously tested, and still less solidly confirmed, although a thorough experiment in blind diagnosis from edited protocols, designed by Alexander and his colleagues to put his theories to test, did yield support for his original hypothesis about asthma. Most of the reported studies have involved clinical and predominantly psychotherapeutic approaches, the ultimate effects of which could only be ascertained by painstaking investigation over a long time. They have also frequently failed to explain their conceptual basis, and to specify by just what means and to what extent events in the psychological-social sphere are conceived as interacting with pulmonary processes. Finally they have often neglected to encompass knowledge about the complex pathophysiological aspects of asthma itself, which has undergone remarkable growth in the past decade.

This chapter will survey relatively recent work in four areas: (1) *Biological observations* which have thrown light on the pathogenesis of bronchial asthma and on pathways leading from brain to peripheral pulmonary tissues, in other words on potential psychosomatic mechanisms; (2) *psychophysiological studies*, offering evidence that short-term psychological influences, possibly utilizing such pathways, may contribute to acute exacerbations and remissions in asthmatic subjects; (3) *psychosocial studies*, suggesting that certain personality constellations, possibly extending short-range influences into chronic states of readiness, may correlate with the

disorder; and (4) studies of *therapy*, examining various psychotherapeutic approaches to asthma, their limitations, and potential future directions.

Biological Observations

The basic pathophysiological defect in bronchial asthma is reversible obstruction of the small airways. Presumably it results from smooth muscle spasm, edema of bronchiolar mucosa, hypersecretion, or possibly all three. A consequence of such obstruction is heightened resistance to the flow of air. Increase in airway resistance—or decrease in its reciprocal, airway conductance—is an essential index of asthmatic dysfunction. Without evidence about air flow, which many clinical reports lack, it is difficult to make meaningful statements about physical changes in an asthmatic patient.

Methods of assessing respiratory function have advanced rapidly in the past two decades. Relatively simple and easily applied techniques for estimating air flow, i.e. the timed vital capacity tracing and the peak-flow meter, suffer from some inaccuracy and are to a large extent dependent on voluntary effort. Since 1956 they have gradually been replaced by the whole-body plethysmograph which yields a rapid accurate sampling of airway conductance, corrected for lung volume, largely independent of effort. This was first applied to the study of psychiatric patients by Ottenberg and Stein, later by Heim et al., Luparello, and others. The instrument is cumbersome,

expensive and requires some skilled cooperation. Additional techniques provide greater flexibility, i.e., flow-volume measurement, and the recently introduced respiratory resistance unit, which allows breath-by-breath assessment of total respiratory resistance.

Studies of gas exchange and of total, as well as regional ventilation have also thrown light on asthma. Dudley and Martin and their colleagues, working with normal subjects, have demonstrated marked sensitivity of numerous respiratory indices to hypnotically induced emotion and pain.

Their techniques and the previously mentioned measures of airway resistance all interfere with free bodily movement and occlude the mouth. As yet there is no satisfactory noninvasive way of monitoring the respiratory functions crucial to asthma. Useful approximations can be obtained by pneumograph tracings of external chest movement, calibrated against other respiratory indices. Heim et al. have applied this approach to speaking subjects, and a number of groups are working with still greater refinements, integrating thoracic and abdominal tracings and using a sensitive magnetic pneumograph.

Taken as a whole, these physiological methods have clarified the enormous range in severity of pulmonary dysfunction in asthma, which may or may not correlate with a patient's subjective impressions. They have

shown complex impairments of elasticity and regional ventilation, the clinical significance of which is not entirely clear. Exercise also has a puzzling relationship to asthma, at times alleviating and at times aggravating the disorder. Of great importance is the remarkably protracted and persistent impairment of pulmonary function following a severe attack. Equally important is the well-established phenomenon of impaired gas exchange in asthma of more than mild degree. Decrease in arterial oxygen concentration appears early in severe attacks and requires careful monitoring; the concentration of CO₂ rises as a late manifestation when attacks continue and worsen. It can increase alarmingly, adding a marked respiratory acidosis to the effects of hypoxia, dehydration, and exhaustion, with which the patient is struggling; it may require correction by administration of bicarbonate. Despite recent advances in pharmacotherapy, such severe episodes of *status asthmaticus* remain a frequent, life-threatening complication of the disease, calling for competent and vigilant therapeutic management.

The factors initiating all of these pulmonary changes are still obscure. Two pathogenetic views exist, each supported by a body of evidence and often zealous, if not myopic, proponents: the allergic-immunologic and the neurogenic.

The view of asthma as basically an allergic disorder has been paramount during most of this century. Clinically it is well known that

asthmatics tend to have a family history of allergic sensitivity. Careful epidemiological studies support the concept of an hereditary predisposition, although a recent study of 7000 twin pairs in Sweden by Edfors, indicated that concordance for asthma in identical twins is only 18 percent, a surprisingly low figure which suggests a far greater role for environmental factors than had been thought previously.

In so-called “extrinsic” asthma, comprising possibly 40-60 percent of cases, a specific antibody, reagin, is demonstrable in the blood serum and is also present in the bronchial tissue of the predisposed individual. Binding of antigen to this specific antibody (immediate hypersensitivity, type I) results in the release of several substances, or mediators. These are still not fully understood. Histamine, serotonin, and bradykinin have been implicated, as has a less clearly identified, slow reacting substance probably released from polymorphonuclear leucocytes. So, more recently, have prostaglandins. Mathe and his colleagues, measuring airway conductance, found that ten asthmatic subjects were almost 8000 times more sensitive to prostaglandin F_{2a} than were ten matched control subjects.

Factors governing the release and activity of such substances have also not been completely elucidated, particularly in the substantial proportion of cases where no extrinsic allergens can be demonstrated. Pulmonary infections are frequently associated with asthma, but it is not always known

whether these constitute specific or nonspecific precipitants, or complications developing after the asthmatic process is already underway.

A crucial question for our purposes is whether the immunologic-allergic responses of the body are independent of influences from the brain. There is evidence to indicate a connection. Hypothalamic lesions or electrical stimulation of hypothalamic areas can induce changes not only in the autonomic nervous system but in immunologic reactivity. Tubercular lesions have proved capable of protecting the rat against fatal anaphylactic shock. Anterior, but not posterior, hypothalamic lesions have had similar protective effects in the guinea pig. In that species they have led to a significant decrease in circulating antibodies. It may thus, in Szentivanyi's words, "be possible to profoundly alter the anaphylactic reactivity" of various animal species.

Immunological and allergic factors may thus interact with neurophysiological and neurochemical influences on the pulmonary tree. The latter are complex in their own right. The role of the parasympathetic nervous system is a case in point. Vagal stimulation and parasympathomimetic agents produce an asthmalike response of the bronchial tree. Vachon et al. have elicited a short-lived and mild but definite decrease in airway conductance in normal subjects by sudden immersion of their heads in water, apparently eliciting a vagal response via a central link, the so-called "diving reflex."

These more or less pure parasympathetic nervous effects tend to be transient and relatively mild. Airway-conductance deficits of from 15 to 20 percent are the rule. (We shall see that this holds also for effects of simple suggestion, which may also be vagally mediated.) Moreover, pharmacological blockade of the parasympathetic nervous system, or even vagotomy, have not proved effective in treating asthma. The theory that asthma represents a “vagotonic” disorder, prevalent at the turn of the century, was largely eclipsed in succeeding years by immunological advance.

However, careful experiments by Gold and his colleagues have forced a reconsideration of the role of the vagus. Using dogs allergic to known antigens, they demonstrated that vagal blockade, both afferent and efferent, could eliminate the experimentally induced sharp rise in airway resistance. They showed furthermore, that unilateral challenge of one lung with antigen resulted in bilateral bronchoconstriction, also inhibited by vagal blockade on the challenged side. They concluded that “vagally mediated reflex bronchoconstriction, possibly arising from epithelial irritant receptors, is a major component of acute, antigen-induced canine asthma.” Only further investigation can tell whether these striking findings will be applicable in human asthmatic disease.

Advancing knowledge about the sympathetic nervous system adds to this picture of intricately balanced adaptive regulation of airway caliber.

Ahlquist's formulation of alpha- and beta-adrenergic receptors, as two systems which have both overlapping and antagonistic functions, has drawn attention to the role of these receptors in asthma. Some evidence has been offered that the human lung has alpha adrenergic receptors, stimulation of which, contrary to usual thinking about adrenergic activity, can induce mild broncho constriction, although other evidence is contradictory on this point. What is unequivocal is the fact that beta-adrenergic agents have maximal bronchodilating action, tending to override parasympathetic activity. The most widely used, potent, short-range agent, having predominantly beta-adrenergic activity, is isoproterenol.

These facts have led to another line of investigation. Szentivanyi, working with mice, which usually show little allergic reactivity, exposed them to *Bordetella pertussis* and found that they then developed marked sensitivity to histamine and to various antigens. He suggested that this altered state was an animal model of "beta-adrenergic blockade" and proposed a sweeping hypothesis: namely, that the key feature in human asthma was such a "blockade" of beta receptors, leaving the lungs prey to bronchoconstrictive reaction upon exposure to allergens, as well as to other noxious stimuli, including cold, infection, and emotional arousal.

Human studies yield some support for the clinical importance of this concept. It is well known that asthmatics are dangerously sensitive to the

beta-adrenergic blocking agent, propranolol. Although earlier investigations using relatively insensitive methods failed to show a pulmonary effect in normal subjects, McNeil and Ingram, using the body plethysmograph to measure five normal subjects, produced beta blockade by propranolol and found marked increase in airway resistance, varying from 40 to over 100 percent. These findings in normal subjects of responses which approached the asthmatic range are unusual. They support the possibility, which is though by no means a certainty, that deficient beta- adrenergic responsivity could play a part in asthma. If so, we should note, it would not necessarily represent a fixed receptor lesion, but might equally well result from a reversible deficiency of circulating catecholamines (as suggested by Mathe.) Conceivably this could play a role in the nocturnal incidence of many asthmatic attacks, although evidence is unclear on this point.

Understanding is still incomplete of the additional steps mediating cellular responses following the action of catecholamines at receptor sites. The second messenger system, adenosine monophosphate (cyclic AMP), is probably involved; and other messenger systems may play various roles (as indicated in the earlier reference to prostaglandins). For present purposes it is important to indicate the existing pathways that link brain processes with those occurring at the peripheral cellular level.

Obviously all these facts have important pharmacological implications.

As noted, atropine and antihistamine drugs have only limited influence on the asthmatic process, in contrast to the powerful short-term effects of beta-adrenergic agents, notably isoproterenol. In severe asthma, one encounters limits posed by excitatory adrenergic side effects, particularly cardiovascular ones, as well as by the still puzzling phenomenon of “fastness” to adrenergic agents. Other drugs, such as aminophylline and especially corticoid substances, have important bronchodilatory action. The latter, as we shall see later, have their own complications, although so far no satisfactory substitute has been found for them.

An intriguing speculation is that tricyclic antidepressant drugs, thought to mobilize catecholamine stores in the brain, might have some beneficial central and also peripheral effect in asthma; preliminary evidence supports such a possibility, though the putative mode of action is obscure.

To summarize: Physiological regulation of lung function represents a complex adaptive balance; its accurate study is necessary to provide precise knowledge about the wide range of pathophysiological respiratory function encountered in asthma.

Immunologic-allergic reactions can lead to a broncho obstructive asthmatic response. However, it seems unlikely that they are the sole cause of asthma, particularly in the substantial number of chronic asthmatics without

clear evidence of extrinsic allergy. There is reason to believe that immunological systems may be integrated with the parasympathetic nervous system centrally or peripherally or both. Nervous influences may augment or decrease the final broncho obstructive process.

The sympathetic nervous system may also be important in the etiology of asthma. Relative deficiency of beta-adrenergic function may aggravate the disorder because of failure to counteract persistence of basic immunologic and/or parasympathetic nervous activity. A second, radical hypothesis is that some sort of “beta-adrenergic deficit” is fundamental in asthma, allowing allergic as well as other noxious influences to produce the disorder. A third, more general possibility is that there is a reciprocal balance between bronchodilating (beta-adrenergic) and bronchoconstrictive (immunologic and parasympathetic) influences, both having links to the brain; and that asthma may represent an acute or chronic imbalance between the two.

Clearly mechanisms are available permitting psychological factors to play a role in asthma, either in conjunction with allergy or possibly at times by themselves. We next must ask what is the psychophysiological and psychosocial evidence for their activity.

Psychophysiological Observations

Acute Exacerbation and Remission

The physiological considerations just mentioned may throw light on a paradox that has puzzled students of asthma. Acute attacks often occur in a setting of turmoil and anxiety, both in adults and children. Hahn found elevations in both heart rate and skin temperature in asthmatic, as compared with normal, children and concluded that there was sustained activation of some parts of the sympathetic nervous system. One is faced with the troublesome question of why the asthmatic patient, in a state of turmoil and arousal, does not "cure himself."

Experimental stress should throw some light on this matter. Mathe and Knapp used as stressful stimuli a film and a mathematical task carried out under negative criticism. No effort was made to differentiate the "first-day" stress from these experimental stimuli. Their subjects were eight mild asthmatics free of symptoms and not requiring medicine, and eight comparison subjects matched for age and socioeconomic status. All dietary and activity factors relevant to catecholamine excretion were controlled. Asthmatics and normals alike responded with increase in heart rate, blood pressure, and circulating corticoids under stress as compared to the control day. As expected, asthmatics differed significantly in their respiratory responses, showing a decrease in airway conductance, as well as slowing of respiration. In addition they showed a highly significant difference in free fatty acid response and epinephrine excretion. Both of these measures were elevated in the control subjects under stress but remained strikingly constant

in the asthmatic subjects. Subjectively the asthmatics also reported increases in anxiety and in overall affect but differed on one emotional dimension: they reported significantly less anger in the provoking experimental circumstances. A partial repetition of this work, confirming the faulty mobilization of epinephrine excretion, has been recorded by Bernstein and Greenland. These findings are consistent with the view that some kind of adrenergic defect does play a role in acute asthma.

Physiological considerations are also important in evaluating studies of *learning* in bronchial asthma. Classical conditioning of asthma was reported by Noelpp-Eschenhagen and Noelpp who exposed guinea pigs to an allergic stimulus, provoking a dyspneic asthmalike response; later this was obtained merely by introducing the animals to the experimental chamber. More recently Justesen, Braun, et al. described similar conditioned “asthma” in guinea pigs and used a variety of pharmacological influences to affect this response. Both of these studies must be questioned, however, in the light of the careful work of Ottenberg and Stein and of Stein and Schiavi. These authors showed that the preponderant effect observed in attempts at conditioning is hyperventilation, presumably as part of a diffuse stress reaction. By careful measurement of airway resistance they were able to identify an apparently true asthmatic response and to obtain this in a small number of animals as a true learned response. However, it readily extinguished, and extrapolation to the human disorder is difficult.

In humans, Dekker, Pelsler, and Groen attempted to show conditioning. However, their results were inconstant and their measurement technique, the timed vital capacity, was relatively crude. Effects of suggestion or pseudo-conditioning could not be excluded in those cases who did show some increase in timed vital capacity. Sloanaker and Luminet used a classical paradigm, exposing subjects in a closed-breathing circuit to a parasympathomimetic stimulus (mecholy) preceded by a tone. In a small number of instances they obtained a possible conditional asthmalike response, but, again, chance or suggestive effect could not be excluded. We must conclude that there is no convincing evidence for classical conditioning in the strict sense as a cause of bronchial asthma in the human. The possibility remains that classical sequences, such as strong emotions associated with prior asthma, may serve as conditional triggering stimuli for subsequent attacks.

An alternative approach is found in the operant paradigm. Vachon used the respiratory resistance unit, which gives a breath-by-breath feedback of information; this was computer processed and fed back to subjects. He worked with two groups of mild asymptomatic asthmatics, fifteen in one and thirteen in another. They were instructed to keep a red light on, programmed to flash when their resistance fell below a critical level. They were also given a monetary reward. The result was a "learned" drop in airway resistance in both groups of experimental subjects. They differed significantly from control subjects exposed to the same situation but given purely random

reinforcement. The decrease in resistance was modest, about 15 percent. How reproducible and lasting, in short, how clinically significant the change was, remains to be determined, as does the question of whether its extent can be increased.

Suggestion (which perhaps should be regarded as a variant of learning, capitalizing on either acquired associations, or implied reinforcement, or both) has also intrigued students of asthma ever since Sir James McKenzie's vivid description in 1886 of "rose asthma" (that is, acute coryza and respiratory congestion accompanied by wheezing) induced in a young woman by the sight of a paper rose. Dekker and Groen reported a number of attacks in subjects, which followed exposure to pictorial or verbal suggestions of objects or substances to which they were sensitive. Their results were not uniform; the extent and mechanism of the asthmalike response remain unclear. Luparello et al. and McFadden, Luparello, and Lyons carried out body plethysmographic studies of forty subjects exposed to saline aerosol, suggesting that it was either an allergenic precipitant to which they had been found sensitive, or a bronchodilator. In approximately half of their subjects they found clear-cut changes in airway conductance in the suggested direction. The effect was blocked by atropine, pointing to an acute vagal influence. Weiss was unable to confirm this suggestive effect in children, possibly because he used less sensitive measures. White also, using the less sensitive timed vital capacity, attempted by hypnosis to influence asthmatics

suffering from clinical disease. As a group they reported subjective relief, but gave no objective evidence of improved pulmonary function.

Environmental change has played a time-honored role with asthmatics, particularly children. Many clinical observations have indicated that when a child is sent away from his family, whether to a hospital, school, or camp, his asthma improves, at least initially. The suspicion followed that one might be removing the child from noxious interaction with family members, especially his mother. Abramson and Peshkin have even talked of the beneficial effects of “parentectomy.”

An obvious question is whether social or allergenic factors were changed. Lamont and his collaborators hospitalized children allegedly sensitive to house dust in their homes. The investigators then secured house dust from each home and distributed it copiously in each child’s hospital room. In nineteen of twenty cases no asthma ensued. Purcell et al. carried out an even more rigorous experiment with thirty-five children. They paid their parents to take a vacation away from home, and brought in an experienced nurse to make daily measurements of medication, respiratory symptoms, and peak air flow. Though some children seemed to have mild anticipatory anxiety, at times accompanied by symptoms, the main effect was, as predicted, improvement in their asthmatic status in the absence of their parents. Again this was modest in scope and occurred in about half of their

subjects. It was of further interest that, on the basis of a brief specially designed diagnostic interview, the authors were able to predict with a high degree of success which children would show improvement and which would not.

Such observations on separation from the home environment fit with earlier reports of Purcell, Bernstein, and Bukantz, indicating that two types of children appeared to be admitted to the Children's Asthma Research Hospital in Denver. They labelled these rapid remitters and steroid dependent, respectively. The former cleared up rapidly; the latter had persistent intractable symptoms and required continued steroid medication. The authors found more obvious neurotic difficulties, both in the children and in their families, in the rapid remitters. They postulated two distinct types of asthma, one more psychosocial in origin, the other more biological. However, one cannot be sure that they ruled out more subtle emotional conflicts, perhaps deep-seated and masked by denial, nor that they excluded a possible physiological chain of events which kept children bound to maintenance steroids, partly on an iatrogenic basis. We will see a similar debate about long-term personality trends in asthmatics. It is worth noting that Kinsman, Luparello, and their colleagues, studying acute symptom patterns in adults, rather than finding simple dichotomous distribution, noted a complex patterning of subjective symptomatology, based around five clusters of symptoms: panic-fear, irritability, hyperventilation-hypercapnia,

bronchoconstriction, and fatigue.

To *summarize*: Acute changes in airway conductance can occur in response to psychosocial stimuli in certain subjects. Presumably these are mediated by the parasympathetic nervous system, that is, vagal influences on the upper airways.

Somewhat more sustained changes in airway conductance, probably involving altered neuroendocrine balance, may be related to specific impairment of epinephrine mobilization. Conceivably this may be a function of altered patterns of arousal, in particular partial mobilization of aggressive impulses along with inhibition of their full expression.

These findings point to avenues whereby learning may influence moment-to-moment airway patency. Classical conditioning has yet to be demonstrated to play a significant role in human asthma, although its participation in triggering attacks cannot be excluded. Possibly more important is the role of operant learning, in which subjects achieve change to gain reinforcement. Preliminary evidence suggests that this can influence airway resistance, though the extent and lasting nature of the effect remains to be shown.

Regardless of the exact nature of mediating pathways, there is strong evidence that remission of asthma may be brought about in certain subjects

by interruption of on-going pathogenic interaction, especially with parental figures. The assertion remains open, but not proved, that these subjects represent a distinct subgroup, comprising about 50 percent of severe perennial asthmatics; they may have prominent “neurotic” elements in their exacerbations, in contrast to other patients with a more permanent “organic” basis.

Psychosocial Observations

Long-term Factors in Bronchial Asthma

It seems wise to return to the assumption stated earlier that asthma as a long-term disorder almost invariably requires some biological vulnerability, hereditary or (conceivably in some instances) acquired in early years. The classical life history is that of a child with a positive family history who develops allergic manifestations, usually eczema, in his first year of life, followed by respiratory difficulty, coming on gradually in the second or third year, often associated with infection, which then develops into typical periodic obstructive disease running its complex fluctuating course.

Although the predisposition is probably always lifelong, many major variations occur in its course. Approximately half of the children with infantile eczema do not develop asthma. A crucial study of family differences

among those who do and those who do not has been undertaken, but not yet completed, by Meijer. Asthmatics change with chronological age. There is allegedly a preponderance of males among asthmatic children, and of females among adults, though recent accurate studies bearing on this distribution are not available. We do know that some children “grow out” of asthma in adolescence. While they are growing out, others are “growing in.” Asthma in midlife seems to have special features. It may run an acute, even fatal course; often it seems to resemble a midlife depression, as illustrated in cases described by Knapp and Nemetz. Recovery, when it takes place, may seem relatively complete. Often in such cases a strong family history is absent. As with many chronic diseases, a sudden onset relatively late in life may carry an improved prognosis, providing the patient weathers the acute phase successfully.

Other specific features have been sought in populations of asthmatic patients. Studies purporting to show EEG abnormality have been reviewed by Leigh and Pond, who conclude that asthmatics are not different from other “psychiatric” patients; the authors explicitly leave open the rather tenuous possibility that all such patients may differ from “normals.”

The incidence among different cultural groups is unclear. Anecdotal reports have suggested a higher frequency of asthma among Puerto Rican immigrants to large inner cities, such as New York and Boston. However, valid

epidemiologic evidence has not been forthcoming; and the relative role of sociocultural stress, as against air pollution, inadequate heating, infection, dietary deficiency, and other factors, cannot be ascertained.

The family structure of asthmatics has been a focus of much attention. A number of studies have dealt with mother and child. The early notion of a “rejecting” mother yielded to that of an “engulfing” one. An investigation by Block et al. suggests that clinicians working with asthma are not in complete agreement about maternal characteristics, raising the possibility of more than one subgroup of mothers and children. Freeman and her colleagues bring evidence that there may be a reciprocal relationship between the allergic potential of a child and psychopathology in the mother. The authors suggest two types of disease, one primarily biogenic, the other sociogenic. This and other efforts to dichotomize the population of asthmatic mothers and children—as in the work of Purcell, Bernstein and Buchantz already mentioned—risk overlooking the role of denial. Some mothers may have a powerful need to overemphasize putative biological contributions to the children’s illness and to minimize psychosocial conflict. Jacobs and his colleagues in two studies tested young adult males with hay fever and mild asthma, using selected indices of biological reactivity, along with a battery of projective tests administered on a “blind basis.” The subjects perceived their mothers retrospectively as controlling or rejecting or both; their feelings in this respect differentiated them from the healthy comparison group. It was

possible, on a basis of both “allergic potential” alone and “psychologic potential” alone, for blind judges successfully to select individuals who showed actual manifestations of allergic disease. Jacobs’ hypothesis was additive: that both psychological and biological factors are widely distributed in the allergic population and that their combined strength determines the severity of the disorder.

We are entering an area which requires complex estimates of family constellations and inner psychological processes. Surface factors may directly contradict and mask hidden elements. Objective methods to measure such balanced forces are not available, and we must rely on more subjective clinical judgments. These have come chiefly from psycho-analytically oriented investigations of a small number of cases. Some of these are summarized below, recognizing their limitations but feeling that their insight cannot be ignored. It remains to be seen whether future large-scale, methodologically more refined, studies will verify them.

Examining a small number of children in psychoanalytically oriented psychotherapy and in psychoanalysis, Jessner and her associates found evidence of continuing oscillation between attempts on the part of the asthmatic patient to separate from the mother and to achieve intense erotized closeness. Sperling elaborated this pattern, underlining the existence of faulty differentiation between mother and child. She felt that mothers of asthmatic

children, like those of young patients with other psychosomatic diseases, could tolerate their child only when ill. This viewpoint, phrased in other terms, suggests that the mother may inadvertently provide powerful sustained reinforcement of the asthmatic process.

Fathers may play an important auxiliary role. Jacobs' retrospective studies with allergic subjects suggest that fathers of asthmatics tend to be absent in one or another way, either physically out of the home, aloof, or ineffective, so that they fail to correct the imbalance between mother and child.

These findings might lead one to expect consistent *characterological* features in patients suffering from asthma, and there is clinical evidence that these do exist. Some of them center around conflictual concerns which are relatively specific and perhaps have a somatopsychic basis: sensitivity to odors, concern about water, sleep, crying, and use of the voice. Stein and Ottenberg and Herbert offer evidence that asthmatics as a group have a heightened sensitivity to odors, which at times antedates the onset of asthmatic symptoms. McDermott and Cobb reported that fears of water or of drowning were more common and intense in asthmatics than in comparison subjects, thus partially confirming what a number of clinicians, for example Deutsch reported. Excessive secretion in the respiratory tract can lead to a state in which the individual feels close to drowning in his own fluids, and such experiences could well mold the anxieties of the asthmatic.

Asthmatic attacks tend to develop at night, often waking the sufferer from sleep, frequently being interwoven with bizarre sleep patterns. It has not been possible to identify asthma clearly with the REM phase of sleep, nor with variations of the diurnal cycle of catecholamine or cortisol secretion, although all of these features may play some role.

Early studies of asthmatics suggested that weeping, crying, and related vocalization were often stirred up, yet were conflictual and not easily expressed. Such “suppressed crying,” as French and Alexander put it in their familiar formulation, might be part of the acute process of asthma, though obviously not specific for that disorder alone. Concern with the voice and the process of speech itself seems at times to be closely related to asthmatic symptoms, possibly connected with inhibition of powerful primitive aggressive impulses, as Bacon speculates. Knapp and Nemetz reported a number of cases in which episodes of aphonia were strikingly interwoven with asthmatic symptomatology. They discussed the possibility, raised by earlier clinicians, that sensitizing life experiences, including close interpersonal experiences with an individual suffering from respiratory disease, might contribute to the later development of asthma as a form of primitive or “pregenital” conversion symptom. Occasional reports keep this possibility alive; for example, a striking case mentioned by Lofgren, in which a patient had been nearly strangled in childhood, and an example of Coolidge’s of asthma in three successive mother-daughter generations.

These reported conflicts influence other habitual character traits. Alternation of asthmatic symptoms with overt psychotic manifestations may occur, although this appears to be the exception, not the rule. Chessick et al. reported that the most frequent chronic disease among narcotic addicts at the U. S. Public Health Hospital, Lexington, Kentucky was asthma.

Other clinical observations have suggested that asthmatics as a group have unusually strong passive and dependent personality traits, reflecting needs to maintain gratification and support from key persons in their environment. This form of personality organization is obviously not unique to asthmatics. At times, furthermore, it appears to be subjected to a kind of personality “counterrevolution” in which a surface picture of marked assertiveness becomes dominant, perhaps assisting the individual in some way to overcome his illness. Frequent anecdotal reports describe children hospitalized for asthma, recovering symptomatically from their disorder and becoming aggressive behavior problems. The more frequent finding is that hints of hidden aggressive impulses, appearing only briefly, are followed by intensification of asthma, often with evidence of guilty depressive feeling. In a study of psychoanalytic material from one patient, two psychoanalysts using a model of primitive impulse-arousal plus failure of psychological defenses, studied notes of sessions immediately before twenty-five exacerbations of asthma and twenty-five comparison sessions from the same period of the treatment. Notes were edited to remove medical cues; as a further control

they were given to two allergist-internists who attempted the same task. The psychoanalysts were able to select “asthmatic” contexts from neutral comparison contexts with significant success, whereas the allergists’ results were only at a chance level.

A clinical hypothesis to account for these observations is that many asthmatics have a deep and early developmental personality defect, which leaves them both attached to a parental figure and subject to powerful aggressive impulses of an early infantile type. Some of these may have a particularly sadistic flavor. The hidden sadistic components have been striking in a series of our patients studied psychoanalytically. Full emergence of such impulses would threaten their “partial symbiotic” attachment, so that when aggression is aroused it is in some way “switched off” and results in asthma. (One is tempted to speculate about connections with a possible parallel defect in adrenergic catecholamine mobilization mentioned earlier.) It is interesting that detailed biographical material of a famous asthmatic, Marcel Proust, supports this picture of a passive loving son attached to and deeply identified with his mother. He showed unmistakable evidence of hidden sadistic perverse impulses.

To *summarize*: Long-term psychological or social patterns associated with asthma can be defined only tentatively. There appears to be an interaction between hereditary vulnerability and the early environment. The

exact nature of their respective roles is uncertain; so is the question of whether we are dealing with a simple dichotomy of allergic versus sociopsychologic cases, as advocated by some, or with a more variable spectrum.

Concerns with odors and water, and conflicts over crying and over use of the voice may be partly related to heightened somatic responsivity in a somatic subject. Other characterological features, although colored by somatic illness, often antedate it and appear related to early experience with the human environment. These pertain to fears of separation from the mother, and to primitive impulses often of a sadistic destructive nature, deeply hidden and defended against by passive, dependent, and masochistic personality organization. Such traits are not unique for asthmatics but may interact with other factors localizing a somatic process in the pulmonary system, and thus contribute to the overall picture of the disease.

Therapeutic Considerations

If, indeed, we are dealing with a lifelong, often life-threatening disorder, having multiple etiologic factors and running a fluctuating course, the problem of evaluating therapy is inevitably difficult.

These considerations apply to all therapy in bronchial asthma, including reports of medical measures. As Bates, Macklem, and Christie remark: "In no other common disorder have so many different therapeutic approaches been

adopted and it is suspicious that many of these are credited with improving the condition.” They add that “spontaneous improvement and remission are common, often occurring independently of any change in treatment,” though one must add that a careful search for any change in life situation must be conducted before one can be sure change is entirely “spontaneous.” If there is any truth to the assertion that asthmatics are sensitive to personal relationships, the influence of these must be considered in reports of success with allergic or other measures. In all assessments of therapeutic results the severity of cases treated must be carefully specified, as well as the amount and kind of all modalities of treatment. Therapy which produces amelioration must be distinguished from therapy which approaches long-range cure. As a rule, ethical considerations demand, at least in patients with asthma of any severity, that some medical allergic management go along with psychotherapy.

A particular problem is posed by steroid medications. In the short run these have changed the picture of the management. Terrifying episodes of *status asthmaticus* can be brought under control and immediate dangers to life greatly diminished. However, the longterm effects are far from clear. Schnerer cites evidence that mortality in children with asthma is as great a decade after the introduction of steroids as it was before their advent. Certainly the persistent complications and side effects, i.e., electrolyte imbalance, hypertension, or tendency to peptic ulcer, present hazards. They

are compounded by the states of dependence which patients develop. These appear to have a clearly physiological basis. Removal of steroids leads to a characteristic state of lassitude and weakness, and in asthmatics, a proneness to explosive and drastic exacerbation, sometimes itself fatal. Yet occasionally in our experience, possibly aided by a variety of therapeutic modalities, including careful medical weaning, also with psychotherapeutic support, patients who have been receiving large amounts of steroid medication do recover and function without the need for medication.

Antidepressive Measures

These have been tried in a sporadic fashion with asthmatics. In sudden late-life asthma, electric shock has been occasionally used, although there are no systematic evaluations of its effect. Experimental evidence of possible bronchodilatory effects of antidepressant medication has led to clinical reports of their use, though these are scattered and inconclusive.

Suggestive Measures, Hypnosis

Although hypnosis may be unable to affect the fully established pathophysiological process, it is possible that hypnotherapy may have long-term benefits. Falliers studied 120 asthmatic patients, using 115 control subjects, also suffering from asthma. The experimental group was treated

with brief rapid-induction hypnosis at weekly intervals. Control subjects were treated with body relaxation. Both groups showed improvement, the females significantly more with hypnosis. By the end of a year in the hypnotized group 59 percent were better and 8 percent worse; the control-group figures were 43 percent better and 17 percent worse; the difference between the groups was significant at the 5-percent level. One patient in each group was dead. One cannot exclude a general “physician-interest” effect, and it would be interesting to know more about the exact characteristics of the patients involved. Nevertheless, a beginning in the study of an important treatment modality is represented here.

Relaxation

This form of quasisuggestive therapy has also been tried, mostly with children. A study by Alexander et al. shows some effects, mostly in mild cases, of relaxation instructions, relayed to the subjects in different ways. The approach has a certain logic. Clinically many asthmatics state that if they can only “relax,” their tightness, wheezing, and congestion seem to improve. It will be necessary to await further results before we can fully assess the clinical importance of this approach.

Behavior Modification

There are only a few systematic studies of this form of treatment. Walton used Wolpe's method of "reciprocal inhibition" successfully with one case. Moore extended his observations in a controlled study, comparing systematic "desensitization" with two other treatment modalities, simple suggestion and a relaxation therapy. She studied twelve subjects, half of them children, in a balanced incomplete block design so that two forms of treatment were given to every patient, and each of the three treatments could be compared in eight subjects. All three forms of treatment led to some subjectively reported improvement. Significantly more improvement in peak air flow, the physiological measure used, was found in the group which had reciprocal inhibition. The strength of this study lies in the fact that the patients were their own controls. However, the numbers were small. A major share of the variance was contributed by two subjects who received reciprocal inhibition as their first treatment, and had a rather marked effect from it. It is possible that individual differences in such a small group of subjects still played a major role. Few details were given about the initial status of the patients and the severity of their illness. The study needs replication, but is nevertheless a model for systematically controlled investigation of therapeutic approaches in this area.

Operant Conditioning

This method, using the biofeedback model, is technically available after

the initial studies of Vachon, but it has not yet been systematically applied as a therapy.

Group Therapy

Groen reports an intensive group-therapy experience in the Netherlands. It involved weekly meetings with patients and an extensive supportive medical and milieu regime. Results were positive, but the large number of variables involved makes definitive assessment difficult.

Long-term Psychoanalytically Oriented Psychotherapy

This method was applied to the original series of twenty-six adults and children reported by French and Alexander. They described substantial improvement in their series but did not give detailed physiological or other follow-up data. The approach has also been applied to severely incapacitated patients by Knapp, Sperlring, and others. One can argue logically that such a long-term approach is indicated if one accepts the evidence of early disturbance in mother-child relationships and deep, primitive personality disorganization in many asthmatic patients. The classical analytic approach must be modified, most observers state, as many individuals suffer from severe personality disturbances. It is necessary to think of severe asthmatics as suffering from "borderline" or "narcissistic" disturbance, though this may be

masked by many effective areas of functioning. Different strategies are possible within this general psychoanalytic framework, such as the more ego-nurturant empathic approach advocated by some, or a more confrontative and active attack on the defensive and gratifying “use” of symptoms by a subject, as advocated by Spertling. No real evidence can decide between these, considering the lack of definitive long-term assessment of all therapeutic results with asthma.

In conclusion: Given the tentative nature of therapeutic evidence, what should a psychiatrist advise for an asthmatic? He is probably wisest if he approaches patients with this “psychosomatic” disorder on the basis of their obvious psychopathology. As with all forms of psychiatric treatment at this time, he must be guided by his own beliefs and experience, and must try to carry out therapy systematically with the hope that time and the accumulation of clinical knowledge will make it possible to sort the wheat of results from the chaff of claims.

Two contrasting cautions are important: the psychiatrist should respect the potential seriousness of the biological process and utilize sophisticated medical knowledge, which in most cases means a sophisticated medical colleague, as part of the total treatment plan. Yet he should respect the remarkable capacity of psychological conflict to lurk behind a screen of “real” physiological symptoms; he must be prepared to stick to his insight when he

senses such conflict, though the patient, the family, and even the attending physician may rationalize it away as an unfortunate by-product of physical suffering. Time-limited and controlled approaches are valuable for purposes of comparative study, particularly of mild cases; but in severe asthma one faces a complicated problem of long-term management, and a long-term relationship with an individual whose somatic and psychic difficulties are extraordinarily intertwined.

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