LEADING ARTICLE

Pitfalls in the diagnosis of asthma

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Asthma is a common disease which affects at least 2% of the British population (Gregg, 1977). The last two decades have seen major advances in safe treatment with regular inhalation of sodium cromoglycate, selective beta₂ stimulant drugs (salbutamol, terbutaline etc.) or beclomethasone. For the majority of asthmatics, these drugs can achieve excellent control and allow the patient to lead a normal life even if treatment with oral corticosteroids is also required. It is therefore depressing to note that there is, as yet, little evidence of a significant improvement in asthma mortality. A recent survey (British Thoracic Association, 1982) reported a mortality rate of 1.84 per 100,000, which indicates no real improvement on rates of 0.46-1.34 per 100,000 reported over the last century by Speizer and Doll (1968). Several recent studies of asthma mortality (Cochrane and Clark, 1975; Ormerod and Stableforth, 1980; British Thoracic Association, 1982) have highlighted two consistent problems; failure to diagnose and assess the severity of asthma by using simple respiratory function tests and, as a result, failure to implement adequate treatment. In this article, I would like to remind the reader of some of the more important pitfalls in the diagnosis of asthma.

Asthma can mimic other respiratory or even cardiac diseases and a common failing is to omit it from the differential diagnosis in patients who do not have the classical and commonest presentation with childhood onset associated with wheezing on exposure to common inhaled allergens such as house dust and grass pollen. Many children are therefore misdiagnosed as having repeated 'bronchitis' and are treated ineffectively with repeated courses of antibiotics when the problem is asthma provoked by viral infections of the bronchial tree. Children, in particular, may appear only to cough, rather than wheeze, and this most commonly occurs at night. The diagnosis may be confirmed by observing wheezing repeatedly when the child is examined or, ideally, by demonstrating variable airflow obstruction by moni-

toring peak expiratory flow rate (PEFR). If the diagnosis remains in doubt, however, a trial of regular bronchodilator therapy is mandatory. This is a safe investigation, particularly if aerosol drugs are used, but the undiagnosed, untreated asthmatic child is always at risk. While 'wheezy bronchitis' may be much commoner than true asthma, most children with recurrent attacks do turn out to have true asthma and this should be suspected sooner rather than later.

Similarly, in the older age groups, late onset intrinsic asthma may be missed. Particularly if patients have smoked (and smoking is much commoner in asthmatics than one might expect), a diagnosis of chronic bronchitis will be made and treatment limited to advice to stop smoking and courses of antibiotics. These patients will then be sentenced to years of misery from their airways obstruction which, at least in the earlier stages, may be quite amenable to treatment. Sputum production can be quite profuse in asthma and may satisfy the criteria for chronic bronchitis. It is therefore a good policy to give serious consideration to a careful trial of regular treatment with aerosol beta₂ stimulants, anticholinergic drugs and subsequently oral steroids in any patient with chronic airways obstruction if there is no documented evidence that they have previously been shown to have no reversibility after such treatment. In patients with asthma, or an asthmatic component to their airways obstruction, a response to corticosteroids is usually seen after about 8 days (Webb, Clark and Chilver, 1981).

There has recently been a considerable interest in the phenomenon of nocturnal or early morning asthma (Editorial, 1981a). This apparently relates to a normal circadian rhythm in airway calibre which is exaggerated by the asthmatic's labile airways. Nocturnal cough and wheezing should alert the clinician to the diagnosis of asthma and it has long been known that asthma attacks can occur almost exclusively at night in some patients (Floyer, 1698). It is

important to realise that in these patients, physical examination and respiratory function tests can be normal during the day when seen in the out-patients or surgery, but every night they may display a deterioration in respiratory function tests compatible with an acute asthma attack (Hetzel, Clark and Houston, 1977). These nocturnal asthma attacks may be attributed to chronic bronchitis; particularly if cough is prominent, if sputum is produced during the attack and if the patient has smoked. Alternatively, these attacks may be misdiagnosed as paroxysmal nocturnal dyspnoea of left ventricular failure. Asthma can present as attacks of cough, breathlessness and wheeze at night as the first sign of late onset intrinsic asthma in the older age groups where chronic bronchitis or left ventricular failure are common conditions.

While most cases of extrinsic asthma provoked by common inhaled allergens, such as house dust and pollens, are easily recognised from the history, an increasingly important problem is now being posed by occupational asthma. Apparent late onset intrinsic asthma, or recurrence in adult life after childhood asthma, may be due to agents inhaled at work and a careful occupational history is very important. This is becoming increasingly difficult for the clinician since the range of products and processes which cause occupational asthma is increasing. Moreover, the asthma may be caused, not by the patient's work, but by aerosols generated by his workmates or even exhaust ventilation from another factory across the street which just happens to blow into the patient's work area. Some agents, notably toluene di-isocyanate which is produced in manufacture of polyurethanes and plastics, can cause a severe progressive asthma in non-atopic subjects without a previous history of asthma. Patients may become extremely sensitive to very low concentrations and irreversible airways obstruction may result if the diagnosis is not made early enough and exposure is allowed to continue. Occupational asthma is now a prescribed disease under the Social Security Act (1975) in Britain and compensation can be given, through the Pneumoconiosis Panel, for asthma due to platinum salts, isocyanates, epoxy resins, colophony fumes (in solder fluxes), proteolytic enzymes, laboratory animals and grain or flour dust (Editorial, 1981b). Diagnosis tends to become more difficult the longer that the patient's occupational asthma has been present. In the early stages it may be clear that attacks occur while the patient is at work, or on coming home from work. As more persistent airways obstruction develops, however, improvement may only be noticeable after a long period away from work on holiday and insufficient recovery may be seen, just over a weekend, for the relationship to work to be apparent. Bronchial challenge to a

suspected agent is not required for compensation to be awarded as this is usually established by the history of asthma developing after a variable latent period of exposure to a known sensitizing agent. Moreover, bronchial challenge is often impossible because patient's airways obstruction is too severe for challenge to be safe. Many workers therefore find the collection of a 'work record' of peak flow readings at frequent intervals while the patient is at work at home is a much more valuable screening test for occupational asthma (Burge, O'Brien and Harries, 1979).

In assessing patients by clinical examination, it is also important to remember that auscultation of the lungs gives no reliable indication of the severity of asthma and the absence of wheezing does not exclude asthma or other types of airways obstruction. The 'silent chest' may indicate very severe airways obstruction. Clinical signs related to the secondary effects of airways obstruction (pulse rate, arterial paradox, impaired mental function) are of more value, but these must be combined with simple physiological measurements of peak expiratory flow rate (PEFR) or forced expiratory volume in 1 sec (FEV₁) (Stark, 1972).

Measurement of PEFR is invaluable in management of asthma and will avoid most of the common pitfalls in diagnosis. It is also vital in obtaining a reliable assessment of the severity of the asthma. Failure to take such measurements is a major, correctable, factor in asthma mortality (Cochrane and Clark, 1975) since doctors cannot judge this effectively by clinical examination alone. Patients are also poor judges of their asthma and studies comparing patients' impressions with results of respiratory function tests show very poor correlations (Rubinfeld and Pain, 1976). With the introduction of the cheap mini-peak flow meter (Wright, 1978) regular records of PEFR, ideally at 4-hourly intervals throughout the waking day are easily compiled by nursing staff on the wards or by patients themselves at home. Patients can keep accurate records with a minimum of instruction (Hetzel, Williams and Shakespeare, 1979).

Different patterns of PEFR response can be identified from these continued records (Turner Warwick, 1977) which can be used to substantiate the diagnosis and select more appropriate treatment. The 'morning dipper' shows much lower readings in the early morning, due to excessive circadian variation in airways tone. Morning dips of greater than 20% of the highest daily reading suggest a circadian variation in clear excess of the normal rhythm in PEFR, which has a lower amplitude, and are therefore of some value as a screening test for asthma (Hetzel and Clark, 1980). Severe morning dips may be an indicator of a dangerous degree of bronchial lability.

and risk of sudden death (Hetzel, Clark and Branthwaite, 1977; Bateman and Clark, 1979).

The 'brittle' asthmatic shows a chaotic pattern of PEFR with sudden rapid falls which, nevertheless, may respond rapidly to aerosol bronchodilator drugs but cannot easily be prevented by regular treatment with other conventional drug therapy on a regular basis. These patients also appear to be a high risk group. They may be wrongly accused of aerosol abuse when in fact they have a genuine need to take frequent doses of beta, stimulant aerosols and may improve if these are taken regularly every few hours. The 'drifter' shows low PEFR readings which at first do not appear to improve, but, as treatment continues a slowly increasing trend can be identified. These patients will not make a dramatic improvement and PEFR may only improve by 100 litre/min or so, but the symptomatic benefit to the patient may still be considerable. In this situation, identification of the slow improvement by PEFR monitoring avoids the common error of abandoning attempts at aggressive treatment too soon.

Patients are occasionally seen who have obvious symptomatic improvement on treatment but in whom the PEFR does not improve. In these cases, more detailed physiological testing is needed to confirm improvement, when it will usually be found that the vital capacity has improved and the functional residual capacity (FRC) has reduced. To understand this phenomenon, noted in acute asthma as the 'guy rope' effect by Woolcock and Read (1966), it must be remembered that the main driving force in expelling air from the lungs in expiration is the elastic recoil pressure of the lung. To overcome a high airways resistance, patients therefore breathe at a higher FRC so that tidal breathing takes place at a higher point on the compliance curve of the lung; in other words, if the lung interstitium is regarded as a spring, this manoeuvre stretches it so that more force is available when it is released on expiration. Because the compliance curve of the lung is sigmoid in shape, however, greater changes in intrapleural pressure are required for a given change in lung volume so that the work of breathing increases at a high FRC. Thus patients in this category may not achieve a better PEFR when their airways obstruction improves but they can now achieve the same result at a more normal FRC and therefore feel less breathless.

Less commonly, errors are made in diagnosing asthma when this is not the cause of patients' breathlessness. The most serious of these is the misdiagnosis of upper airways obstruction due to tumours, as illustrated in this volume (Parrish, Banks and Fennerty, 1983), strictures or foreign bodies. Careful clinical examination should normally prevent this error by noting that the patient has stridor or a fixed wheeze over a main bronchus and that

breath sounds are normal, or diminished but without wheezes, on auscultation over the lungs. Further suspicion should be aroused by failure to respond to bronchodilator drugs and by lack of any history of variability in the airways obstruction. Respiratory function tests can help establish the diagnosis but more careful interpretation is needed if obstruction is due to a high, extra thoracic airways obstruction in the trachea. In asthma, and other disease causing widespread obstruction of intrapulmonary, intrathoracic airways, airways obstruction is greatest on expiration, since the intrathoracic airways are compressed on expiration as intrathoracic pressure rises. In the extrathoracic part of the trachea, however, airflow limitation is greatest in inspiration, since the intraluminal pressure is then lower than atmospheric pressure on the outside of the trachea, tracheal collapse being prevented by the rigidity of the tracheal cartilages. Since the majority of spirometric tests are routinely carried out in expiratory manoeuvres, this tends to minimise the apparent airflow limitation caused by an upper tracheal lesion. Obstruction of extrathoracic airways tends to reduce the PEFR more than average flow rates so that a reduction in the ratio of PEFR/FEV₁ will be noted (Empey, 1972). It is best detected, however, by spirometric tests in inspiration, as seen in the inspiratory part of the flow volume loop (Bass, 1973). The peak inspiratory flow rate is particularly reduced and the loop assumes a characteristic 'box' shape with impairment of both inspiratory and expiratory flow but with more marked impairment of the inspiratory curve.

In conclusion therefore, pitfalls in the diagnosis of asthma are best avoided by an awareness of the range of presentations that are possible and by the use of simple physiological tests, rather than relying on clinical judgement alone. Asthmatics should test PEFR at home just as diabetics test their urine (Editorial, 1978) and it is regrettable that peak flow meters are not available on NHS prescription. The wider use of peak flow meters would enable patients to anticipate deterioration in their asthma so that earlier remedial treatment, with reduced need for outpatient and inpatient treatment, would be possible.

It is a sobering thought that, in a recent study of asthma deaths outside hospital (MacDonald, Seaton and Williams, 1976), 23% of these fatal attacks lasted less than 30 min so that death frequently occurred before any doctor was available to see patients. For 9 of 21 patients seen by the GP, admission to hospital was not advised, emphasising the difficulty experienced in identifying potentially fatal asthma attacks. This is, of course, the greatest pitfall of all. Peak flow monitoring may not be the complete answer, but it is certainly a significant step in the right direction.

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(Received 31 May 1983)