EMERGENCIES IN GENERAL PRACTICE

CARDIAC ASTHMA

BY

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The term "cardiac asthma" is applicable to a clinical syndrome—paroxysmal dyspnoea due to heart failure—with a range of severity extending from urgent acute pulmonary oedema to transient nightly attacks of cough, wheezing, and breathlessness, though used by some to refer only to the latter. Although such paroxysms sometimes occur in the presence of mitral stenosis, they are most commonly an expression of left ventricular failure in hypertensive, coronary, or aortic valvular disease. A sudden attack of intense dyspnoea is not infrequently caused by acute myocardial infarction, and the occasional absence of the characteristic pain or the overshadowing of pain by the intense dyspnoea may result in the basic lesion being missed.

Clinical Picture

The patient is most commonly in the second half of life and may or may not be known to suffer from hypertension or coronary disease. He is wakened from sleep, or roused from quiet, recumbent rest, by cough and breathlessness which may rapidly reach urgent severity so that he is obliged to sit up gasping for breath and hardly able to speak. Soon some frothy, watery sputum appears, becoming in the most severe attacks profuse and pink through blood-staining. Wheezing may be prominent. The patient's distress is often accentuated by the occurrence of Cheyne-Stokes periodic respiration. During the phases of apnoea he may fall back on his pillows and close his eyes in thankful comfort, only to be roused in a few seconds by terrifying distress as the phase of hyperpnoea develops. The tantalizing repetition of the cycle may be almost unbearable.

In less severe attacks of breathlessness with cough and wheezing the intensity of the symptoms may decline spontaneously after the patient has been sitting up for 20 to 30 minutes and he may return to sleep without medication. While a severe attack is sufficiently alarming for medical help to be summoned, in the case of the lesser attack the patient commonly consults his doctor only after several disturbed nights. He may be free from breathlessness by day or admit only to some effort distress if questioned. The described picture of cough, wheezing, and breathlessness all too often leads to an erroneous diagnosis of bronchitis or bronchial asthma and the prescription of an ineffectual expectorant or linctus, or the administration of isoprenaline or adrenaline.

The patient taken urgently ill in the night is seen to be sweating and cyanosed, with cold extremities. The heart is accelerated, while the rhythm, commonly regular, may be disturbed by extrasystoles or auricular fibrillation. The blood pressure is usually elevated, often grossly, pulsus alternans being often observable, but may have fallen from a previously hypertensive level to a normal or low level either because of the severity of the heart failure or because the attack has been precipitated by the occurrence of acute myocardial infarction. A complaint of gripping, crushing, or burning retrosternal pain, severe or slight, prior to or coincident with dyspnoea may draw attention to this possibility, which is of more than academic interest, even in the middle of the night, for in several respects treatment should be varied according to whether or not infarction has or seems likely to have taken place. Cardiac enlargement is almost invariably to be found and should be looked for by palpation of the apical impulse, for this provides the necessary conclusive evidence of the existence of organic cardiac

disease, and, in the presence of a normal or low blood pressure and the absence of aortic valvular disease, establishes that hypertensive heart disease underlies the condition. Further valuable evidence of heart failure is provided by the finding on auscultation at the apex of triple or gallop rhythm. The neck veins are often somewhat engorged, but oedema of ankles or sacrum is generally absent, since attacks of cardiac asthma usually antedate the appearance of right heart failure and indeed may cease to occur when right ventricular failure supervenes.

Although respiration may be noisy with wheezing rhonchi, crepitations are to be heard over both lungs, in severe cases in all areas to such an extent and in such profusion that the lungs seem waterlogged, in less severe cases predominantly at the lung bases.

In the case of the patient examined between milder attacks similar evidence of heart disease will be found—hypertension, cardiac enlargement, and basal crepitations, although these last may be scanty at such a time.

The need to elicit these signs of heart disease has been stressed, since there is no little chance that hasty consideration of the symptoms of cough, wheezing, and dyspnoea, and the finding of harsh respiration with rhonchi and crepitations, may focus attention on the respiratory tract and lead to a diagnosis of bronchitis.

Paroxysmal dyspnoea of this type—cardiac asthma—except in the instances in which it is actually precipitated by myocardial infarction or paroxysmal tachycardia, occurs for the most part at night before or during sleep. It may be the first clear manifestation of heart failure in the hypertensive subject, though it often comes after a decline in effort tolerance has set in. Cardiac asthma, however, seldom develops for the first time in patients already under adequate treatment for hypertensive heart failure unless it is due to the occurrence of some complication such as infarction or a paroxysmal arrhythmia. Severe nocturnal attacks may follow unduly vigorous exertion during the preceding day, or an injudiciously large meal in the evening, or high excitement, including intercourse.

Cause of the Dyspnoea

The symptoms of cardiac asthma are immediately due to pulmonary engorgement. Quiet rest in bed with its decline in physical effort and reduction in metabolism is associated with reabsorption of tissue oedema fluid, even where this has not been manifest, and therefore with an increase in circulating blood volume. The recumbent posture results, owing to the removal of the influence of gravity, in an increased venous return to the right side of the heart which, through increased venous filling pressure, is a stimulus to increased ventricular output. In the presence of hypertension or aortic valvular disease or coronary sclerosis, whose main detrimental influence is upon the left ventricle, this chamber, beginning to reach the stage of dilatation and failure, may be less able to respond to such a demand for greater effort and output than the less directly burdened right ventricle. The vascular bed between these chambers—that is, the pulmonary capillaries and venules—is congested therefore to accommodate the excess of the right ventricle's output over that of the left, tiny though that excess may be at each stroke.

Pulmonary congestion and the resulting transudation of oedema fluid into the alveolar walls and cavities progressively reduce the vital capacity, while the emergence of this fluid on to the sensitive bronchial epithelium touches off

the cough reflex. Either the dyspnoea of reduced vital capacity and stiff congested lungs or the cough weakens the patient. The intense effort of the dyspnoea and cough constitutes very vigorous exercise which itself aggravates the cardiac embarrassment. An additional factor operating in some cases is general peripheral arteriolar constriction. The increase in arterial pressure which this causes throws a sudden additional load on a left ventricle already prejudiced by established hypertensive disease, coronary ischaemia, or both, and precipitates left ventricular failure. In such cases the blood pressure is found to be elevated well beyond what is known to be that patient's customary level.

In the mild or moderate case the assumption of the erect posture when the patient sits up in bed, or better still in a chair with his feet on the floor, may break the vicious cycle of pulmonary engorgement→dyspnoea and cough→exercise→further pulmonary engorgement→worse dyspnoea, by allowing a reduction in venous return so that the labouring left ventricle can gradually overtake its debt and clear the pulmonary congestion below the critical level. Then the attack may end spontaneously. In the worst cases the vicious cycle progresses and the patient dies, drowned by his own pulmonary transudate.

Partial ischaemia and anoxia of the respiratory centre resulting from diminished arterial output from the overloaded left ventricle, combined probably with a measure of cerebral vascular stenosis, especially in the elderly, is the cause of Cheyne-Stokes respiration in cardiac asthma. It is not to be attributed to uraemia in the circumstances under discussion.

Immediate Treatment

Treatment of cardiac asthma resolves itself into the handling of the emergency and the long-term management of the underlying disorder with the object of rendering less likely the repetition of such disturbing and dangerous crises which are almost certain to recur if the crisis is taken on its own and sustained treatment neglected.

In the emergency morphine should be administered immediately the diagnosis of *cardiac* asthma has been made. The object here is twofold: first, to reduce the sensitivity of the cough reflex and of those respiratory reflexes having their origins in the stiffened congested lungs, whereby the intensity of the effort of coughing and breathing may be diminished, and, secondly, to calm the apprehension, even panic, and the restlessness which a severe attack of cardiac asthma causes in even the most phlegmatic person. With these effects achieved venous filling pressure on the right heart and therefore right ventricular output are reduced and the left ventricle given an opportunity of partially clearing the congested pulmonary vascular bed. The effects must be achieved with the maximum certainty and without delay, since any severe attack of this condition must be regarded as critical and immediately endangering life. For a patient of average or over-average weight morphine sulphate, $\frac{1}{4}$ gr. (15 mg.), should be injected intravenously and the same quantity subcutaneously. In the presence of acute heart failure absorption of the whole dose from a subcutaneous injection is undesirably slow, and if intravenous injection is for any reason impracticable, half of the total dose of $\frac{1}{2}$ gr. (30 mg.) should be given by intramuscular injection. In the case of patients of under-average weight the total dose can be reduced to $\frac{1}{4}$ gr. (20 mg.), but again half of this dose should be given intravenously. When the attack is of only moderate severity the ordinary subcutaneous injection of morphine, $\frac{1}{4}$ gr. (15 mg.), may suffice.

Many practitioners combine a dose of atropine sulphate with the subcutaneous injection of morphine or even give atropine alone. This habit is based upon a misconception that the excessive fluid in the respiratory passages is due to excessive bronchial secretion. However, the fluid is in reality transuded oedema fluid whose production cannot be controlled by paralysing by atropine the bronchial secretory glands. If atropine has any action in cardiac asthma it is by depressing vagus-mediated reflexes involved in the pro-

duction of dyspnoea. Atropine is better omitted altogether, since the dryness of the mouth which it induces makes the patient demand fluid to drink which he would be better without.

Although Cheyne-Stokes respiration, if present, may be expected to be aggravated by the injection of a large dose of morphine, this must not deter the physician from giving morphine in an adequate dose. The distressing dyspnoea (which is what immediately matters) of the hyperpnoeic phases will be suppressed. The periodicity of the respiration, however, can usually be rapidly relieved by an intravenous injection of aminophylline, 0.5 g. Such an injection should be given in all severe or moderate cases, even in the absence of Cheyne-Stokes breathing, for its vasodilator and bronchodilator action.

The patient should be propped securely on a high bank of pillows or made comfortable in an easy chair.

Other Measures

The question of the administration of digitalis or digitalis glucosides requires rather more careful consideration in each case. When the patient is known not to have been taking digitalis or digoxin for at least a week before the attack, and when there is no reason to suppose that acute myocardial infarction has occurred, digoxin, 1 mg., should be injected intravenously directly after the administration of morphine. It may be combined with the injection of aminophylline. When the patient is known to be fully under the influence of an adequate maintenance dose of digitalis or digoxin already, it is wise to assume at the time of the acute episode that the attack is due to the occurrence of myocardial infarction and withhold the intravenous injection of digoxin. Such should also be avoided, even in the undigitalized patient, when the features of the case strongly suggest that an infarction has precipitated the crisis. The risk of inducing paroxysmal ventricular tachycardia or even fatal ventricular fibrillation is not negligible. Reliance should then be placed on intravenous morphine and aminophylline for the emergency and digoxin, 0.5 mg., given by mouth as the starting point of a more gradual process of digitalization.

Where there is a strong clinical indication that acute myocardial infarction has taken place, early and rapid anticoagulant therapy with heparin may prevent an immediate extension of the infarct, with serious and perhaps fatal results. Heparin, 10,000–15,000 units (100–150 mg.) according to body weight, should be given intravenously and repeated six hours later, after which it can be decided whether a full course of anticoagulant therapy is to be undertaken.

If oxygen is readily obtainable it should be used. It is desirable that a flow-meter should be employed and a flow of 6–8 litres a minute administered by double nasal catheter or by a light plastic nose-and-mouth mask. The patient should be allowed to go to sleep with this in place, but naked lights should not be permitted near the oxygen cylinder or the mask.

If in a severe case dyspnoea does not subside within 10 to 30 minutes of the injection of morphine, *venous* tourniquets should be applied on the proximal parts of all four limbs to reduce venous return. They should be tight enough to hamper the venous outflow from the limbs but not tight enough to impede the arterial inflow. After about an hour the tourniquets should be *slowly released one at a time* to avoid a sudden augmentation of venous return which would again overload the recovering heart.

Rapid venesection of one pint (570 ml.) through a wide-bore needle, or by simply slashing a vein, may be usefully employed instead of the above "bloodless phlebotomy," especially in plethoric patients.

When a striking rise in blood pressure is observed, suggesting a hypertensive crisis, the use by intravenous injection of powerful vasodilators and hypotensive agents such as tolazoline and the methonium compounds has much to

commend it on theoretical grounds. In patients suffering from severe acute left ventricular failure due to hypertension the rapid reduction of an extravagantly high blood pressure may so reduce the load on the failed left ventricle as to contribute importantly to the interruption of the vicious cycle. Beneficial effect has been obtained in many such cases, but the use of methonium compounds by intravenous injection is fraught with risks and is better avoided except by those with experience of their use in this and other fields.

Long-term Management

On the morning after the severe attack the long-term measures should begin. Arrangements should be made for the patient to be confined to bed, well propped up at night, or to bed and a chair in his room according to the severity of the condition, in the "uncomplicated" case for about two weeks, but of course for longer when myocardial infarction has been diagnosed. Mersalyl, 2 ml., should be given by intramuscular injection, to be repeated two days later, twice weekly for two to three weeks, and weekly thereafter, even though manifest peripheral oedema be absent. A more cautious approach to the use of full doses of a mercurial diuretic—for example, mersalyl, 0.5 ml. for the first dose and 1 ml. for the second—is indicated where previous symptoms of prostatic hypertrophy suggest the possibility of acute retention being precipitated by the sudden massive diuresis of the full dose. The process of thorough digitalization should be continued by ordering, for example, digoxin, 0.25 mg. thrice daily by mouth. Later, when a full digitalis effect has been achieved, as indicated by the appearance of anorexia and squeamishness, the dose should be adjusted, after 24 to 48 hours' "desaturation-holiday," to one suitable to maintain an optimum digitalis effect for the rest of the patient's life. The occurrence of cardiac asthma should be the signal in the untreated patient for the institution of the "digitalis life."

Moderation of salt intake should be arranged by the prohibition of salty foods and added salt, the limitation of salt in cooking, and the avoidance of sodium-containing medicines. If severe sodium restriction can be achieved and if a mercurial diuretic is regularly given the patient can be permitted a normal fluid intake.

For three or four nights after the acute attack of cardiac asthma morphine, $\frac{1}{4}$ gr. (15 mg.), should be given subcutaneously and for a week thereafter an opium-containing hypnotic given by mouth, such as:

Chloral hydrate	gr. 20 (1.3 g.)
Tincture of opium	min. 20 (1.2 ml.)
Liquid extract of liquorice	min. 10 (0.6 ml.)
Water	to fl. dr. 2 (7 ml.)

It is important that until the full effect of digoxin, mersalyl, and rest has been obtained the patient be protected by opium against a recurrence of the nocturnal crisis.

In the case of the patient complaining of recurrent milder attacks of cardiac asthma at night an opium-containing hypnotic is indicated for seven to ten nights. For this purpose a mixture like the above, but with 30 gr. (2 g.) of chloral hydrate and 30 min. (1.8 ml.) of tincture of opium, is suitable, half the dose being given in the last few nights of the period or for a few further nights if required. He should be advised to use extra pillows and to avoid slipping down to a low position if he can. While the patient is thus protected against the nocturnal attacks he should be brought fully under the influence of digoxin in the usual way and a permanent maintenance dose established. Again, even in the absence of manifest oedema, intramuscular injections of mersalyl, 2 ml., should be given in the morning twice a week for two or three weeks, and weekly thereafter.

The other measures called for in the treatment of cardiac failure—salt limitation and modification of habits in respect of physical and mental effort—will if necessary be inaugurated at the same time. In the milder cases under consideration, unless there are manifest features of chronic heart failure or frank evidence of serious coronary disease, it is seldom necessary to confine the patient to bed for more

than perhaps a day or two, though a week or two off work with modified rest about the house is desirable while the full effect of treatment is being attained.

Conclusion

There must be few acute disorders in the non-surgical realms of adult medicine in which treatment is so immediately rewarding as in cardiac asthma. The utter relief of the acutely dyspnoeic patient, or the achievement of nights blessed by sound sleep undisturbed by attacks of wheezing breathlessness in the less severely ill patient, does, for me at any rate, amply reward the night-call, the bloody mess of the venesection, or the early morning injection of mersalyl. However, it cannot be too often or too forcibly emphasized that the foundation of the emergency treatment of this serious and dangerous disorder is its recognition as heart failure and not bronchitis, and the use of *morphine*, *aminophylline*, and *digoxin*, and of its prevention the permanent treatment of hypertensive and coronary heart disease with *digoxin*, *mersalyl*, and salt restriction.

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THE SWEEP-FREQUENCY METHOD OF MAKING SCREENING TESTS OF THE HEARING OF SCHOOLCHILDREN

STATEMENT PREPARED BY PROFESSOR A. W. G.
EWING AT THE REQUEST OF THE M.R.C.
COMMITTEE ON THE EDUCATIONAL
TREATMENT OF DEAFNESS*

In a recent investigation it was found that approximately 70% of pupils in English schools for the deaf and partially deaf have suffered from their handicap since before the age of 2 years. From the Ministry of Education's returns and from the results of recent surveys it seems that nearly all deaf children are admitted to special schools by the age of 7 years, but that the ascertainment of partial deafness as a cause of educational retardation is at present less effective among children under 7 than among those who are older.

The sweep-frequency technique has been shown to be a reliable and efficient method of making screening tests of the hearing of 5- and 6-year-old schoolchildren. A pure-tone audiometer is used, and, when the range of frequencies that are used is extended from 250 to 6,000 cycles per second, early symptoms both of conductive and of perceptive deafness can be detected. It has been established that a sweep check over this range of frequencies at 15 decibels above normal threshold of detectability is more thorough and reliable as a screening test than the use of word material (as in the test by gramophone audiometer), the loudness of which cannot be monitored satisfactorily for this purpose. The fading-intensities, called-number test has been shown to be sometimes ineffective for the detection of high-tone deafness.

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