EMPHYSEMA*

BY

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Knowledge and understanding of emphysema has advanced considerably during the last two decades chiefly as the result of the work of Christie (Christie, 1934, 1939, 1944a, 1944b; Meakins and Christie, 1934), the Rochester workers (Hurtado and Boller, 1933; Hurtado and Fray, 1933; Hurtado *et al.*, 1933; Hurtado *et al.*, 1934), McMichael (Aslett *et al.*, 1939; McMichael and Sharpey-Schafer, 1944), and Cournand and his colleagues (Cournand *et al.*, 1939; Cournand and Richards, 1941; Bloomfield *et al.*, 1946; Baldwin *et al.*, 1948, 1949). They have thrown a flood of light on the mechanism of emphysema, but its cause remains largely unknown and we still have no effective treatment to offer its victims. Emphysema is regarded as a natural consequence and an inseparable part of long-standing bronchitis or asthma. The usually accepted explanation of this association is that the emphysema results from chronic cough, but this cannot be the whole cause; thousands of people smoke so excessively that they cough almost incessantly and yet their exercise tolerance remains reasonably good and their lungs objectively normal, while not infrequently we find patients whose emphysema has developed either quite suddenly after an attack of pneumonia or insidiously without any antecedent respiratory disorder.

The hypothesis that there is a genetic tendency to emphysema is sometimes utilized to explain such anomalies, but in 103 patients (76 males, 27 females) in whom hereditary factors have been assessed, apart from a few whose emphysema was primarily due to allergic asthma, only three gave any family history of respiratory disease. These 103 patients presenting clinically as cases of emphysema were observed in routine hospital practice over the past five years, and have been further investigated and followed in an endeavour to augment knowledge of the aetiology and natural history of the disease. They all had dyspnoea either at rest or on moderate exertion; none had any primary cardiac disease, anaemia, or obesity to which their poor exercise tolerance could be ascribed; and all presented unequivocal physical signs of emphysema.

Aetiological Factors

The various aetiological factors in these 103 cases are summarized in Table I, from which it will be seen that chronic bronchitis was by far the most important, being dominantly responsible in 50 (49%) of the cases studied; 41 (82%) of these 50 were males. Some of these had developed bronchospasm, which no doubt aggravated the emphysematous process, but they were all primarily bronchitics. In one the bronchitic tendency was associated with kyphoscoliosis, in another with chronic nasal sinusitis, and in two others it developed after an attack of whooping-cough; in the remainder no cause was apparent. Nearly every patient smoked heavily, and it seems probable that this was an aggravating influence, because we have shown elsewhere that smoking is associated with a diminution in ventilatory efficiency (Whitfield *et al.*, 1951a). It is remotely possible that emphysema may in such cases be the primary disorder and chronic bronchitis a complication, but the history of cough and sputum so often precedes by many years the advent of dyspnoea that such an explanation is unlikely in the extreme.

TABLE I.—*Apparent Major Aetiological Factor in 103 Cases of Emphysema*

Aetiological Factor	Number			Age	
	Total	Male	Female	Mean	Range
Chronic bronchitis ..	50	41	9	52	35–80
Asthma	19	8	11	39	15–62
Pneumonia	10	5	5	57	38–74
Being "gassed" in first world war	9	9	—	58	52–64
Sulphur dioxide	1	1	—	60	—
Silicosis	3	3	—	54	52–56
Bronchiectasis	7	6	1	44	31–60
Primary emphysema ..	4	3	1	52	41–64

The second largest group comprised 19 (18%) cases of asthma (11 females and 8 males). Of these, six had a superadded bronchitic element. The mean age of the bronchitic group was 52 while that of the asthmatics was 39. Emphysema due to asthma therefore occurs at an earlier age than when it originates from bronchitis, and, while the former is more commonly encountered in females, bronchitic emphysema is largely a disorder of males.

Another group comprised 10 patients (five males and five females) who had no respiratory symptoms until an attack of pneumonia, after which their exercise tolerance was permanently reduced and they developed either a productive cough or a tendency to asthmatic attacks. It might reasonably be supposed that some recognizable permanent change such as bronchiectasis had developed in the lungs as a result of the pneumonia, but the chest radiographs showed no evidence of this. Seven other patients who had suffered from emphysema for some years developed attacks of pneumonia and thereafter their exercise tolerance was very much less. Two other patients suffered a similar deterioration following the occurrence of a spontaneous pneumothorax, though in both cases the lung re-expanded satisfactorily. It seems, therefore, that any condition which temporarily throws added strain on lungs already damaged by emphysema produces a further deterioration in their ventilatory efficiency which persists indefinitely; but why an attack of pneumonia will sometimes produce emphysema in lungs which were previously normal cannot be explained. The pneumonic episodes in these 10 cases occurred at ages varying from 13 to 69, with a mean of 39.

Nine of the male patients studied were gassed in the first world war and thereafter had a productive cough and exertional dyspnoea, though previously they had had no respiratory symptoms. It is thought that this is a possible aetiological factor in males who at the present time are becoming seriously disabled by emphysema. In one other male the aetiology was somewhat similar, respiratory disability dating from exposure to a high concentration of sulphur dioxide fumes.

In three other males who appeared clinically to be cases of emphysema due to chronic bronchitis, chest *x*-ray films revealed some silicotic mottling as well as gross emphysema, and inquiry into their past occupations showed that they had been engaged in work which exposed them to pneumoconiotic hazard.

Seven of the patients (six male and one female) were found to have a considerable quantity of purulent sputum, and bronchography revealed bronchiectasis, which was probably an important cause of their emphysema.

Four of the patients were of particular interest, as their emphysema had developed without any apparent cause. Three of them were males and one was a female, and in each case symptoms had begun in early middle life. Two of the patients developed pulmonary heart failure from which one died whilst under observation.

Patients suffering from chronic pulmonary tuberculosis commonly have some degree of emphysematous change in

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their lungs. No such patients were included in this series, as it was desired to avoid any possibility of getting tubercle bacilli in the spirometers.

A further aetiological factor that must be considered is age. It has been shown (Kaltreider *et al.*, 1938; Whitfield, Waterhouse, and Arnott, 1950) that in normal subjects there is a progressive diminution in ventilatory efficiency with advancing years, and we are all familiar with the patient who first develops recurrent attacks of winter bronchitis together with exertional dyspnoea and the physical signs of emphysema late in life.

Symptoms and Signs

Emphysema itself has but two symptoms—dyspnoea and dry cough. These are the sole complaints of the victims of primary uncomplicated emphysema. At first the dyspnoea is noticeable only on exertion, but as the disease progresses it becomes evident at rest also.

In most cases the symptoms of the causal disorder are also present—for example, the sputum of bronchitis and bronchiectasis, or the paroxysmal dyspnoea of asthma. In some cases complications such as pneumonia, spontaneous pneumothorax, or pulmonary heart failure may render the symptomatology more complex.

The physical signs of emphysema are well known. The deep poorly expansile chest, kyphosis, hyperresonance, diminution or absence of cardiac and liver dullness, impalpable apex impulse, and the faint breath sounds with prolonged expiration are all evident in severe cases. Of all these the diminution of cardiac and liver dullness is perhaps the most reliable evidence of emphysema, but if cardiac failure supervenes the right ventricle enlarges, the cardiac dullness returns, and the apex impulse once more becomes palpable, usually well outside the mid-clavicular line. In many cases rhonchi indicate either an asthmatic aetiology or the presence of secondary bronchospasm. The presence of finger-clubbing suggests that bronchiectasis is the basic lesion. Many severely emphysematous patients are cyanosed, and this is particularly evident in those progressing to pulmonary heart failure in whom the chronic arterial oxygen unsaturation has resulted in polycythaemia of greater or less degree. The cyanosis in emphysema is relieved by oxygen.

Radiology

In hospital practice examination of the chest tends to rely more and more on the radiograph. It is therefore important that the radiological appearances in emphysema and their correlation with the clinical state should be widely known and clearly defined.

To this end 52 patients suffering from varying degrees of emphysema were assessed clinically, spirometrically, and radiologically (Whitfield, Smith *et al.*, 1951). All showed some radiological abnormalities, and there is no doubt that the presence of emphysema can be readily detected radiologically. Moreover, radiography is often the only means by which the primary pathology is revealed—for instance, in cases of silicosis.

Nevertheless the degree of emphysema can be determined more readily and more accurately by ordinary clinical assessment and by spirometry and the other ancillary investigations mentioned below than it can radiologically.

The most reliable radiological sign of emphysema is depression, flattening, and restricted respiratory excursion of the diaphragm. In this connexion it must, however, be added that bronchospasm limits diaphragmatic movement, and an immobile diaphragm has not the same significance if the patient is asthmatic at the time of screening.

Hypertranslucency of the lung fields is valuable evidence of emphysema and is very pronounced in those with an asthmatic aetiology. There are, however, many factors affecting the reliability of this observation. In an over-penetrated film normal lungs appear to be hypertranslucent, and in a "soft" film emphysematous lungs appear to have a normal translucency, while the presence of some degree of fibrosis or silicosis, or of congestion of the lung fields due to the supervention of heart failure, may obscure translucency. Increased brilliance of the lung bases as compared with the upper zones, and hypertranslucency and increase in the area of the retrocardiac and anterocardiac windows on the lateral film, provide more definite evidence of emphysema than does generalized hypertranslucency.

A dragging down of the hila on inspiration is good evidence of emphysema, but in advanced cases in which the diaphragm has become immobile this is no longer evident. Bullae and cysts are occasionally seen, but they are no indication of the severity of the disease, and usually tend to obscure the diagnostic issue by suggesting the possibility of a tuberculous infection or some other pathological condition. Kyphosis and apical rib crowding is often seen, particularly in males whose emphysema has resulted from chronic bronchitis, and a high maximum transverse diameter of the chest is present in some patients, most of whom are female asthmatics. Premature calcification of costal cartilage is not a feature of emphysema.

On the antero-posterior radiograph the heart shadow is characteristically small and is often described as "drop-like" or ptotic. The cardio/thoracic ratio is reduced and air is often visible between the diaphragm and the cardiac apex. All these changes probably result from the low diaphragm and the consequent more vertical position of the heart; they are particularly evident in asthmatic subjects. About two-thirds of the group studied showed enlargement of the pulmonary conus and one-fifth had enlargement of the right ventricle, but this radiological evidence of cor pulmonale was unrelated to the severity of the emphysema. With the development of pulmonary heart failure the heart becomes generally enlarged.

Other Ancillary Investigations

(a) Spirometry

Spirometry is valuable in the investigation of obscure respiratory disorders. In emphysema, however, the diagnosis is usually clear from the history, physical signs, and chest radiograph, but the alterations in lung volume give valuable information on the degree of damage to the lung tissue. Spirometry is carried out on a Benedict–Knipping spirometer, the vital capacity, inspiratory capacity, and expiratory reserve volume being measured in the usual way, and the functional residual capacity (and from it the residual volume) is determined by the constant-volume hydrogen dilution method described by McMichael (1939–42). Helium has now largely superseded hydrogen as the indicator gas because of the explosive properties of the latter.

The lung-volume changes which characterize emphysema are a diminution in vital capacity and an increase in the residual volume and in the ratio of the residual volume to the total capacity (Meakins and Christie, 1929–30; Hurtado *et al.*, 1934; Kaltreider *et al.*, 1938; Aslett *et al.*, 1939;

TABLE II.—*The Total Capacity and its Subdivisions in Normal Subjects Compared with that in Emphysematous Patients (Mean Absolute Values in Litres)*

Division of Lung Volume	Males			Females		
	Normal (64)	Mod. Emphysema (18)	Severe Emphysema (14)	Normal (32)	Mod. Emphysema (11)	Severe Emphysema (1)
Total capacity	5·74	6·17	5·74	4·28	4·87	4·35
Vital capacity	4·00	3·31	2·22	2·87	2·37	1·54
Inspiratory capacity	2·73	2·26	1·52	1·99	1·64	0·85
Expiratory reserve volume	1·27	1·05	0·70	0·88	0·73	0·69
Functional residual capacity	3·02	3·91	4·21	2·29	3·23	3·50
Residual volume	1·75	2·86	3·51	1·41	2·50	2·81

TABLE III.—*Mean Percentage Values of Lung Volume of Normal Subjects Compared with those in Emphysematous Patients*

Division of Lung Volume	Males			Females		
	Normal (64)	Mod. Emphysema (18)	Severe Emphysema (14)	Normal (32)	Mod. Emphysema (11)	Severe Emphysema (1)
Vital capacity / Total capacity	69·8	53·9	38·5	67·8	48·6	36·0
Inspiratory capacity / Total capacity	47·8	36·6	26·3	47·4	33·5	20·0
Expiratory reserve volume / Total capacity	22·0	17·3	12·2	20·4	15·1	16·0
Functional residual capacity / Total capacity	52·2	63·4	73·7	52·6	66·5	80·0
Residual volume / Total capacity	30·2	46·1	61·5	32·2	51·3	64·0

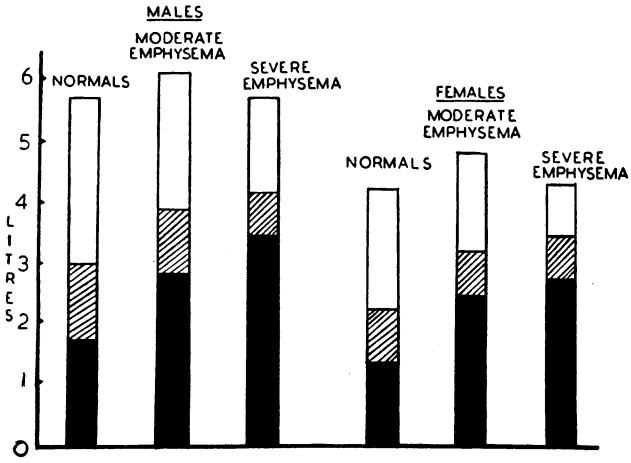

FIG. 1.—Lung volume of normal subjects compared with that of emphysematous patients. Residual volume—black. Expiratory reserve volume—hatched. Inspiratory capacity—white.

Christie, 1944a ; Baldwin *et al.*, 1949). They are well shown in Tables II and III and Fig. 1, which summarize the results of the determination of the total lung volume and its subdivisions in a series of normal subjects and a group of patients suffering from emphysema. The latter were divided into two groups—moderately emphysematous and severely emphysematous. The severely emphysematous were dyspnoeic at rest, while the moderately emphysematous were dyspnoeic only on exertion. It will be seen from the Tables that the lung-volume changes in emphysema increase with the severity of the disease.

Other spirometric features of emphysema are, first, a slowing of expiration (Fig. 2), and, secondly, an increased mixing time in functional residual-capacity determinations. In normal subjects in our apparatus the indicator gas is fully mixed throughout the lung–spirometer system within three minutes or less, but in emphysematous subjects seven or more minutes may elapse before mixing is complete. Normal mixing-times have to be ascertained for each design of apparatus, as small differences have a profound influence on mixing-time.

The mechanism of these changes is discussed below.

(b) Maximum Breathing Capacity

The maximum breathing capacity is the maximum amount of air that a subject can respire by voluntary hyperpnoea expressed in litres per minute. It is determined on a Benedict–Roth type spirometer from which the carbon-dioxide-absorbing canister and the one-way valves have been removed and smooth-bore 1¼-in. (3-cm.) tubing

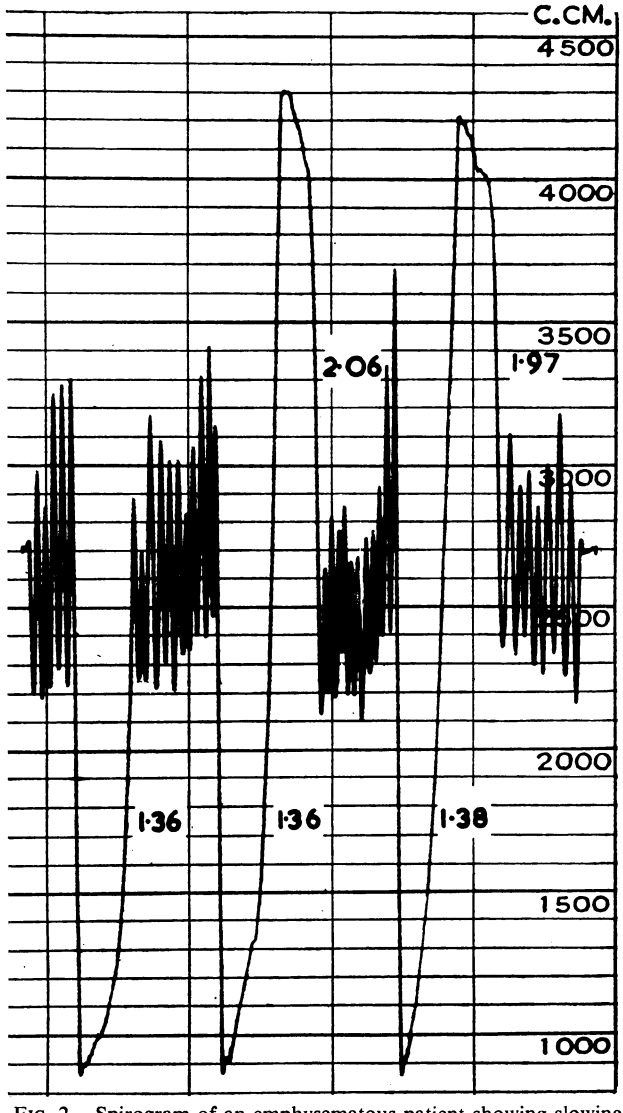

FIG. 2.—Spirogram of an emphysematous patient showing slowing of expiration.

substituted in order to diminish resistance. It is usually measured over a period of 15 seconds, as it is difficult to maintain a maximum state for a longer period. In emphysema the maximum breathing capacity is always reduced, and the degree of reduction is probably a more valuable guide to the severity of the emphysema than are the changes in lung volume (Gilson and Hugh-Jones, 1949 ; Cournand *et al.*, 1939). Another feature of the maximum breathing capacity in emphysematous patients is that it is carried out at or near the position of full inspiration and not at the resting respiratory level as in normal subjects.

(c) Oxygen Saturation and Carbon Dioxide Tension of the Arterial Blood

As the outstanding functional defect in emphysema is inadequate alveolar ventilation, alveolar tensions of oxygen become reduced and of carbon dioxide raised. Baldwin *et al.* (1949) have evolved an excellent functional classification of severity based on the oxygen content and carbon dioxide tension of the arterial blood. It comprises four grades:

(1) Arterial oxygen saturation above 92% and carbon dioxide tension below 48 mm. Hg following standard exercise. Despite ventilatory inefficiency, adequate alveolar ventilation is maintained by means of compensatory hyperventilation, consequently the arterial blood shows neither oxygen unsaturation nor carbon dioxide retention.

(2) Arterial oxygen saturation falls below 92% after standard exercise, but the carbon dioxide tension remains at the normal level of below 48 mm. Hg.

(3) Arterial oxygen saturation below 92% and carbon dioxide tension above 48 mm. Hg following standard exercise.

(4) The same as grade 3, with the addition of congestive cardiac failure.

Arterial puncture is with practice as easy and as safe as venepuncture. While the estimation of the oxygen content of blood is an easy procedure the measurement of carbon dioxide tension (Lilienthal *et al.*, 1946) is more exacting; however, oxygen content alone is of value in assessing severity.

Mechanism of Emphysema

Christie (1934) and Christie and McIntosh (1934) proved the loss of elasticity in emphysematous lungs by measuring the intrapleural pressure in three cases, in all of which it was either atmospheric or positive and unaltered by the induction of pneumothorax. Sections stained with elastic-tissue stains serve to confirm that one of the essential features of emphysema is an atrophy of the elastic tissue in the lung. The other important abnormalities revealed by histology are the breaking down of alveolar septa, with the formation of large spaces and the thinning and avascularity of the alveolar walls. Such changes are evident to the naked eye in the bullae that are so often to be seen at necropsy. The increase in the amount of air in the lungs at the end of a normal expiration (functional residual capacity) and at the end of a maximal expiration (residual volume) results partly from the loss of elastic recoil in the lungs and partly from the increase in alveolar space which is such a striking histological feature. This residual-volume increase inevitably reduces the vital capacity. The altered mechanics of breathing are an additional factor. Diaphragmatic and lower costal activity is reduced with increased activity of the upper costal and accessory muscles of respiration. In severe cases the entire thoracic cage may be seen to be elevated during inspiration as a rigid unit by the strong contractions of the accessory muscles. The diminished chest expansion and the reduction in maximum breathing capacity are further expressions of the abnormalities leading to limitation of the vital capacity. When the maximum breathing capacity becomes reduced to a level at which the ventilation at rest is equivalent to 30 or 40% of its volume (that is to say, the breathing reserve is only 60 or 70%) dyspnoea becomes evident.

Inspired air is slowly and inadequately mixed in the large saccular alveolar spaces and bullae. This is clearly shown in the prolonged mixing-times found in functional residual-capacity estimations. As a result the alveolar oxygen content becomes too low to saturate the haemoglobin, especially when oxygen demand is raised by exercise, and alveolar carbon dioxide retention occurs, with consequent raised blood carbon dioxide tension.

Although much remains to be discovered concerning the development of congestive cardiac failure in emphysema, some aspects of its physiological basis are known. Pulmonary hypertension seems to be an important factor in its causation. Cardiac catheterization has enabled studies of pulmonary artery pressure to be made in cases of emphysema both at rest and during exercise (Harvey *et al.*, 1951). Cases fall naturally into three groups: (*a*) those in which pulmonary artery pressure does not rise during standard exercise; (*b*) those with normal resting pressures which become elevated on exercise; and (*c*) those in which the pressures are high even at rest.

The reason for this pulmonary hypertension is to some extent the mechanical reduction of the pulmonary vascular bed and also the vasoconstriction which follows alveolar anoxia. This latter mechanism was uncovered by Motley *et al.* (1947) and is reversible; its operation may explain the precipitation of crises of cardiac failure by acute respiratory infection or prolonged asthmatic attacks.

Arterial unsaturation produces several changes which throw strain on the heart. Blood volume increases, a feature of this being an increase in the haematocrit value out of proportion to the haemoglobin increase. In other words, the circulation is flooded with hypochromic red cells. It is thus a characteristic of the polycythaemia of severe emphysema that it should result in a reduced colour index. This hypervolaemia is probably an important factor in producing the increased cardiac output, which, although to some extent compensatory, aggravates pulmonary hypertension and burdens the right ventricle.

The summation of these stresses increases the volume of blood remaining in the right ventricle at the end of systole, the right ventricular diastolic pressure rises, and in quick succession follow right auricular and systemic venous hypertension. Congestive failure thus results. It is of the utmost importance to realize that by correcting the acute attack of respiratory infection and the bronchospasm that set in train this vicious sequence the whole process can be reversed and the patient may make a remarkable, albeit temporary, recovery from acute cardiac failure.

Natural History

The degree of emphysema seems largely dependent on the severity and duration of the primary causal disease. Many whose bronchitis, asthma, bronchiectasis, or silicosis is moderate in degree, although their exercise tolerance is considerably reduced, live their natural span without any serious restriction of their activities and ultimately die from some quite unconnected condition. On the other hand, those whose primary disease is severe may become totally incapacitated by dyspnoea in the course of a few years and die soon from pulmonary heart failure or pulmonary insufficiency. A more common story, however, is of productive cough over a period of 20 to 30 years or more. At first the cough is present only after a cold, later it persists throughout the winter months, and as time goes by there is a progressive shrinkage in exercise tolerance, the cough and sputum are present all the year round, and the winters bring febrile respiratory episodes with increasing periods off work. Ultimately dyspnoea becomes so severe that the patient has to abandon his occupation, and in the course of the next few years he succumbs either in a pneumonic episode or from pulmonary heart failure or dyspnoea and anoxia—the syndrome of pulmonary insufficiency. Reference has already been made to the serious aggravating effect of pneumonic episodes and of spontaneous pneumothorax due to rupture of emphysematous bullae.

Such patients are particularly prone to attacks of pneumonia, and spontaneous pneumothorax may occur repeatedly, recurrences often being provoked by effort. Sometimes after a spontaneous pneumothorax the lung fails to re-expand. Such cases of recurrent or chronic pneumothorax are best treated by producing a chemical irritation of the pleura by the injection of iodized talc (poudrage) or silver nitrate. Pleurodesis is thereby effected and there is usually no further trouble (Brock, 1948). Alternatively, thoracotomy and suture of the affected area of lung is carried out.

Pneumonia may bring life to an end in two or three days either from dyspnoea and anoxia or by precipitating acute pulmonary heart failure. It is remarkable, however, how such patients can, with penicillin and oxygen, be rescued from the brink of the grave and survive another two or three winters. An attack of status asthmaticus is often the factor which terminates life in the emphysematous asthmatic, but even in the most desperate cases cortisone therapy may produce miraculous improvement.

Occasionally mental confusion develops in severe cases. This is sometimes associated with the presence of papilloedema, and such patients are often described as suffering from emphysematous encephalopathy. Although in the majority of patients showing mental symptoms there is some degree of pulmonary heart failure it is not believed that the confusion of mind and the papilloedema result from circulatory failure. The current belief is that they

are due to the anoxia and high carbon dioxide tension in the arterial blood producing dilatation of the cerebral vessels, a high cerebral blood flow, and a high cerebrospinal fluid pressure (Simpson, 1948; Donald, 1949; McMichael and Lennox, 1949; Davies and Mackinnon, 1949).

Treatment

While important aetiological factors such as bronchitis may prove susceptible to control, there is no curative treatment for emphysema itself, as the disease results from changes which are essentially degenerative in nature. Much can, however, be done to improve the lot of these unfortunate respiratory invalids with measures designed to control infection and relieve bronchospasm.

If sputum is profuse, and particularly if it is purulent, the amount of expectoration and the degree of dyspnoea and respiratory distress can be greatly reduced by postural drainage and by appropriate antibiotic therapy. The usual practice is to prescribe penicillin systemically and by inhalation without prior bacteriological examination of the sputum. Such practice is in most cases satisfactory and leads to marked reduction in the volume of sputum, but the inhalations sometimes give rise to a troublesome glossitis and soreness of the nasal and buccal mucosae which takes some weeks to clear up. Where bacteriological facilities are available it is better to withhold antibiotic therapy until the predominant organism in the sputum has been identified and its sensitivities have been ascertained. "Estopen" may be preferable to penicillin.

If the patient is conscious of wheezing or if rhonchi are audible on auscultation a diminution in dyspnoea and an improvement in exercise tolerance will result from bronchodilator drugs, though no benefit is likely in cases in which bronchospasm is absent (Whitfield, Arnott, and Waterhouse, 1950, 1951b). Ephedrine is the most effective and useful drug of this type, and is usually given in a dose of ½–1 gr. (32–65 mg.).

The larger doses advocated by Herxheimer (1946) have not in our experience widened its range of usefulness or increased its efficacy, and they do produce distressing side-effects. Aminophylline is a valuable alternative, and is particularly useful where hypertension makes one hesitant to employ ephedrine. It is usually given in a dose of 0.2 g. orally. Various bronchodilator sprays, such as adrenaline (1 in 100) or "neo-epinine" can also be used with benefit in this type of case.

In patients with arterial anoxia oxygen will almost always produce marked relief from both cyanosis and dyspnoea. If human ingenuity could devise a portable form of oxygen therapy it would be a godsend to severely emphysematous patients. In advanced cases of emphysema with gross arterial anoxia and carbon dioxide retention the predominant stimulus in the chemical control of respiration appears to be the low oxygen tension and not the high carbon dioxide tension in the blood. In such patients oxygen therapy may, by improving the oxygen saturation of the arterial blood, depress ventilation, with further retention of carbon dioxide. Oxygen therapy should therefore be intermittent—for example, 45 minutes per hour—and a close watch kept for evidence of carbon dioxide poisoning (Davies and Mackinnon, 1949; Donald, 1949; Simpson, 1949; McMichael and Lennox, 1949).

The almost invariable aggravation of symptoms that emphysematous patients suffer during the winter months bears eloquent testimony to the effect of climate on the disease. The warmer and drier the weather is, the less troublesome are the respiratory symptoms. As regards tobacco, the less the patient smokes the better.

Breathing exercises have become a routine measure in a variety of respiratory disorders. It is difficult, indeed impossible, to assess their precise value in the treatment of emphysema. One's impression is that they are most beneficial in the younger age groups and in patients whose emphysema has an asthmatic aetiology. It is probably true to say that breathing exercises are worth while in emphysema if they can be carried out without exposing the patient to inclement weather and without incurring economic hardships. Some of the more intelligent patients learn the exercises and continue them as a daily ritual.

Although expectorants are still widely prescribed (Dunlop *et al.*, 1952) they have or should have virtually disappeared from hospital therapeutic practice, and are probably of no value in the treatment of respiratory disease. Indeed, codeine as a linctus is the only drug of the old guard that is likely to survive.

When polycythaemia and pulmonary heart failure develop, venesection often produces marked temporary relief. McMichael (1948) has suggested that ouabain may be of more value than digitalis in pulmonary heart failure, but, in general, digitalis, combined with mercurial diuretics, fluid and salt restriction, and measures to combat the primary lung disorder, remains current practice.

The dangers of morphine in status asthmaticus, pulmonary heart failure, and kyphoscoliosis (Daley, 1945) are well but not sufficiently well known. Morphine is a powerful respiratory depressant, and in such cases readily precipitates a fatal anoxia and carbon dioxide narcosis. The asthmatic, the kyphoscoliotic, and the patient with cor pulmonale dying from cardio-respiratory failure within a few hours of a dose of morphine is still a commonplace and the dangers of the drug cannot be overstressed.

While hope for the future must lie in the discovery of methods for the prevention or rapid arrest of the common cold—thus striking at the main root of bronchitis—and the control of bronchial asthma, advances in applied physiology have shown the rational therapy for the severe crises of emphysematous congestive cardiac failure. We now know how to use oxygen so as to gain the great advantages it offers without producing carbon dioxide poisoning. Thus seemingly abstruse and academic studies in respiratory physiology have yielded swift and rich benefits to the victims of emphysema.

Summary

Investigation of the aetiological factors in 103 cases of emphysema showed the apparent cause to be most commonly chronic bronchitis or asthma. Less frequently bronchiectasis or silicosis appeared to be the primary lesion. In some cases respiratory disability dated from an attack of pneumonia or from being gassed in the first world war. In four cases emphysema developed without any antecedent respiratory disorder.

Radiologically, depression, flattening, and diminished respiratory excursion of the diaphragm, and hypertranslucency of the lung fields, especially the bases and the anterocardiac and retrocardiac windows, are the most constant features of emphysema. The heart is characteristically small and the cardio/thoracic ratio reduced. A large proportion of cases show enlargement of the pulmonary conus, and in some the right ventricle is also enlarged.

Spirometry shows a decreased vital capacity and increased residual volume in emphysematous subjects. The degree of change is of some value in assessing the severity of the disease.

The maximum breathing capacity is reduced in emphysema, and this is a more reliable index of severity than are the changes in lung volume.

Emphysema produces a reduced oxygen content and an increased carbon dioxide tension in the arterial blood. These changes are of the greatest value in assessing the degree of lung disease.

The mechanism and natural history of emphysema and pulmonary heart failure are discussed.

Measures to control infection and relieve bronchospasm are the essentials of treatment. The value and dangers of oxygen therapy are pointed out and the lethal effect of morphine is stressed.

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INCIDENCE AND NATURE OF PUERPERAL PSYCHIATRIC ILLNESS

BY

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Many writers have expressed the view that no form of psychiatric illness occurring in pregnancy or the puerperium is a specific clinical entity. The general opinion seems to be that the ordinary stress and strains of pregnancy, operating in predisposed individuals, produce non-specific mental illnesses which are indistinguishable from other mental reactions and are due to the interaction of factors—the pregnancy being of psychological importance only. Brew and Seidenberg (1950) state that these disorders occur only in predisposed individuals, and disregard the possibility that a physiological or endocrine disturbance may be the primary activating factor.

As other surveys have been made only on patients who have been admitted to mental hospitals these conclusions are not justified; and as no psychiatric survey of women confined in maternity hospitals has been undertaken there is no evidence about the importance of alleged psychogenic factors.

The question has been considered afresh by studying the case records of all the women delivered at maternity hospitals in Bristol between January, 1938, and June, 1948, a period of 10½ years, the cases numbering about 37,000. Physical and psychological factors were noted in order to ascertain their importance. The incidence, nature, and outcome of mental reactions were studied, and all cases of mental illness related to the puerperium or pregnancy that were treated in Bristol Mental Hospitals during the same period were examined by me; thus there was a double check and control. The tables relate to these surveys. Reference has also been made to some other cases, which were not included in the surveys as they occurred in other years or outside Bristol.

This appears to be the first double survey made, although a report of the Rotunda Hospital, Dublin (quoted by Noyes, 1948) refers to 81 cases of unspecified psychiatric illness in 54,000 confinements.

In Bristol, about half the total number of pregnant women were confined in Southmead Hospital and Bristol Maternity Hospital, the remainder at home or in nursing-homes. Probably all the cases that required in-patient psychiatric treatment were admitted to the Bristol Mental Hospitals. The survey, therefore, is an almost exact record of the incidence of serious and prolonged puerperal mental illness in Bristol from 1938 to 1948.

In all cases included in this study there was a considerable degree of mental unbalance (such as depression or mania, clouding of consciousness, delirium, or thought disorder). Slight emotionalism and sleeplessness that cleared up in a few days were noted so frequently in the case histories that they cannot be regarded as important or even abnormal, and these were disregarded. Mental complications during pregnancy, occurring before confinement, were very rare, and unless otherwise stated the figures apply to puerperal mental illness only.

Incidence of Puerperal Reactions

During the 10½ years under survey 81,000 women were confined in Bristol, and 116 (1.4 per 1,000) of these were admitted to the Bristol Mental Hospitals. Approximately 37,000 were confined in the two maternity hospitals and 44,000 elsewhere. Mental illness was noted in 64 of the 37,000 (1.7 per 1,000), but in 27 of these it cleared up quickly, and only 37 (1 per 1,000) required mental-hospital treatment. Of the 44,000 women confined at home or in nursing-homes, 79 (1.8 per 1,000) were admitted to the mental hospitals.

Thus the incidence of mental illness was practically the same in both categories; but the incidence of admission to a mental hospital was greater in the case of women confined at home, probably because it is impossible to cope with even slight degrees of mental illness at the puerperium in the average house or flat. The maternity hospital cases that required mental-hospital treatment represent the more nuclear type of mental illness, not subject to quick remission.

Diagnostic Categories and Distribution

The cases were classified as follows: (1) manic-depressive psychosis; (2) puerperal depression; (3) schizophrenia; (4) mixed schizophrenic states; (5) neurosis; (6) confusion and exhaustion; (7) toxic delirium; and (8) other conditions (Tables I and II).

To facilitate evaluation of various factors they have also been reclassified as follows: (1) affective (including manic-depressive cases and puerperal depression); (2) schizophrenic (including mixed schizophrenic cases); and (3) other cases.

In the maternity hospital series nearly all cases in the schizophrenic group and the majority in the affective group required mental-hospital treatment, as these represented true