THE ACUTE CHEST IN GENERAL PRACTICE

BY

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Respiratory disorders comprised 12% of all attendances in a general practice over a period of one year (Fry, 1952). Together with the upper respiratory infections the respiratory tract accounted for over 25% of all work done in this particular practice. Admittedly, a very large proportion of these ailments were of a mild and transient nature producing no detectable changes in the lungs on clinical examination. Excluding these minor cases, there remain two large groups of more serious chest disease—the acute and chronic disorders.

It is with the former that this paper is concerned. "The acute chest" is a useful term to denote sudden severe chest disease producing definite clinical signs of respiratory tract involvement.

This report is on a series of 164 patients with this type of disorder. They were seen over a period of two years (July, 1950, until July, 1952) in a general practice of over 4,000 patients on the outskirts of London. During this same period 15 new cases of pulmonary tuberculosis were diagnosed, 800 patients were treated for short-term coughs with no chest signs, and 55 patients required repeated treatment for their "chronic bronchitis." This area is not regarded as presenting any extra hazards so far as chest diseases are concerned.

A special effort was made to obtain extra information from radiological and pathological investigations in addition to careful clinical records, but anyone who has had close association with general practice will at once understand that this ideal is a practical impossibility in a number of cases. Thus, not all the cases have been fully investigated according to hospital standards.

The cases are best considered under the following accepted clinical groups: acute bronchitis, the pneumonias, acute pleurisy, severe asthma, and spontaneous pneumothorax.

Acute Bronchitis

This term is rather a vague one. Different interpretations of it are given by different practitioners. Here, it is taken to describe a moderately severe chest illness of sudden onset and varying constitutional upset with bilateral and diffuse clinical signs of involvement of the respiratory tract. It should be stressed that it affects more or less the whole of the "middle respiratory tract" of both lungs.

In this series two subgroups were noted—a primary group, in which the illness occurred in persons with no previous history of chest disease, and a secondary group in which the acute attack complicated some active or latent chest condition.

It is doubtful whether acute bronchitis is in fact ever a primary condition in the true sense of the term, as it is invariably a downward extension of a preceding upper-respiratory-tract infection. The reasons why in these instances this downward spread occurs are not obvious, nor is it clear why the condition should stop short and affect only the bronchi and not proceed to cause definite parenchymal involvement. In fact, it has been found most difficult to differentiate clinically between severe acute bronchitis and so-called bronchopneumonia. The usual differentiation seems to be based on the degree of severity of the illness and on whether rales (which are taken to denote parenchymal involvement) are present in addition to rhonchi.

It is probable that acute bronchitis occurs when the defence mechanisms of the respiratory tract have been rendered ineffectual either by a preceding disease such as "chronic bronchitis," or when, for some reason, it follows on an upper-respiratory-tract infection.

The actual organisms which are detected from the sputum are non-specific, and in this series consisted of the normal inhabitants of the upper respiratory tract (*Neisseria catarrhalis* and *Streptococcus viridans*), with the occasional addition of pneumococci and haemolytic streptococci.

Sixty-six patients were treated for one or more attacks of acute bronchitis in the past two years. The group distribution was as follows.

Primary Group.—This consists of 23 patients (10 males, 13 females). The age distribution is as shown in Table I. Nineteen cases occurred in the winter and spring months.

Secondary Group.—This consists of 43 patients (18 males, 25 females), with 51 actual attacks. Again all but five occurred in the winter and spring. The age distribution was different from that of the primary group in that this condition is more frequent in the older age groups. The associated lung disorders were: asthma in 12 patients (7 children), bronchiectasis in 2, "chronic bronchitis" in 23, and measles in 6 (it was considered that the bronchitis in measles was a secondary form).

TABLE I

Group	Age Group in Years								70 and Over
	0-4	5-9	10-19	20-29	30-39	40-49	50-59	60-69	
Primary	8	4	—	—	1	2	1	4	3
Secondary	3	10	1	1	2	4	3	8	11

Clinical Features

The actual clinical features in both groups were alike.

Onset.—This was more sudden in the primary group, with its preceding upper-respiratory-tract infection. Rapid progression with increasing severity of the disease is particularly likely to occur in young infants, as was seen in two patients in this series, both of whom were under 6 months old.

Symptoms varied in degree. There was a dry, irritating, and distressing cough which became productive after a few days. No chest pain occurred, apart from some retrosternal discomfort. Dyspnoea was often present.

Signs.—The pulse rate was always raised; the fever varied from 99 to 104° F. (37.2 to 40° C.), being least marked in the aged, although there was often pronounced tachycardia. The respiratory rate was usually increased. Examination of the chest revealed diminished chest movement, normal resonance on percussion and on auscultation, numerous bilateral and widespread rhonchi, with an especially prolonged expiratory phase in the secondary group. The presence of associated cardiac failure in the aged was a common finding.

X-ray examination was possible in four patients in the acute attack, increased pulmonary striations with some possible consolidation being noted at both bases in one case. No significant changes were present in the others.

Sputum was examined in 40 patients, and the common organisms found were *N. catarrhalis*, *Str. viridans*, pneumococci, haemolytic streptococci, and occasionally *Staphylococcus aureus*.

Treatment consisted of: (1) Treating the infective process with parenteral procaine penicillin in the more severe type of case, the adult dosage being 700,000 units daily in single doses. The response was satisfactory in most cases, particularly in the primary group. Those in the secondary group which did not respond to penicillin were given chloramphenicol, 500 mg. six-hourly (adult dose), with no further beneficial effects. (2) The use of sedatives to ensure adequate rest was regarded as important. (3) Where bronchospasm was marked, the use of antispasmodics such as adrenaline, ephedrine, and isoprenaline sulphate was of some value.

Course.—All but one patient recovered; the time for recovery was from 6 to 28 days, being most rapid in the primary group. Three patients were admitted to hospital: (1) An infant aged 3 months who was very ill and whose home facilities were inadequate. (2) A woman aged 36 with recurrent asthmatic attacks, who developed a tension pneumothorax during an attack of acute bronchitis. (3) A man aged 57 with 30 years' history of progressively more severe chronic bronchitis; he developed severe cardiac failure during an acute attack, was admitted to hospital, and died six weeks later.

The Pneumonias

Pneumonia is here taken to mean an acute illness with clinical signs of involvement of the lung tissues—it is in fact an inflammation of the lung with alveolar exudate as shown by clinical, and in some cases additional radiological, findings. As with other types of acute respiratory disorders, the aetiology and classification of this condition are far from satisfactory—we have a number of terms purporting to describe different syndromes. These terms may be based on aetiological concepts: we have specific—for example, pneumococcal, streptococcal, etc.—and aspiration pneumonias; on morbid anatomical grounds we have lobar and lobular pneumonias; and topographically there are such subdivisions as massive, central, apical, and creeping varieties. In more recent times we have had the addition of "primary atypical" and "virus" pneumonias. All this is rather confusing, especially so to the general practitioner, who relies largely on clinical findings and who now treats the majority of pneumonias without reference to hospital (in this series only one out of the 77 cases under consideration was sent to hospital).

It is not the purpose of this paper to attempt a reclassification, but a plea is made that appropriate investigations be undertaken to clarify the whole field of acute lung infections as well as to study further the functions and defence mechanisms of the respiratory tract as a whole. A step in this direction has been made by Scadding (1951), who suggests an aetiological basis for classification of pneumonias.

A relatively simple method has been used in this instance, and grouping has been on clinical and possible aetiological grounds (admittedly this is not entirely satisfactory).

Group 1. Cases with Lung Consolidation (7 Cases)

In this group are those cases of what is now termed "classical pneumonia." It is an illness of sudden onset with severe constitutional disturbance and clinical signs of lobar consolidation. In the experience of this practice it is now a relatively rare disease. Only seven cases were seen over the period in question (10% of all pneumonias), and four of these were complications of influenza. It is striking that all patients were over 55 years of age, three being over 70. All were very ill, with sudden onset, high fever, tachycardia, and dyspnoea. Locally, evidence of consolidation was found in all cases. Response to penicillin (700,000 units daily) was rapid, and complete recovery occurred in two to four weeks. Sputum contained pneumococci in two, haemolytic streptococci in two, and *Staph. aureus* in one case. One patient was admitted to hospital on account of unsatisfactory home arrangements. There were no deaths.

Bronchopneumonia is a common and favourite diagnosis, and, as has already been mentioned, it is differentiated from bronchitis by the severity of the illness and the presence of signs denoting involvement of the lung parenchyma. Great difficulty was experienced in making this differentiation, and rather than attempt this distinction all such cases were diagnosed as acute bronchitis and dealt with in that section.

Group 2. Localized Pneumonia with no Clinical Signs of Consolidation (70 Cases)

The most common form of pneumonia encountered was a relatively mild condition producing identical signs in all cases, but in which there was a possibility of more than one aetiological process. This syndrome presented as a

fairly sudden illness in a fit person, with a distressing cough, little real constitutional upset, and characteristic signs on auscultation of the chest consisting of diminished air entry and a shower of rales at the end of inspiration. An attempt was made to group these cases in three possible aetiological sections.

A. Probable Viral Influence (35 Cases).

Thirty-three such cases were seen in the influenza epidemic of 1950–51 (Fry, 1951). Although no conclusive tests were carried out to prove the viral aetiology, these cases occurred during a nation-wide epidemic which other observers had shown to be due to influenza A virus. The onset of lung involvement in the majority occurred a few days after the beginning of influenzal symptoms, the general picture being as already described. One or other base was involved in 30 instances, and the right upper zone in three. A pleural rub was heard in three patients. Sputum was mucopurulent, blood-staining occurred in two cases, and bacteriological examination showed the presence of *Str. viridans*, *N. catarrhalis*, and haemolytic streptococci in the six cases so investigated. There were no abnormal radiological findings in the four patients x-rayed. There was no definite response to antibiotics, but recovery was eventually complete in all cases, taking from 2 to 28 days for signs to clear. Since the end of 1951 all such cases were investigated by virus complement-fixation tests, but there has been no epidemic, and only one case of influenza B with identical clinical findings was seen in March, 1952. In June, 1952, a case of probable psittacosis was seen. This is described because of its relatively mild nature.

A normal healthy schoolgirl aged 11 was seen at home on June 23 with a history of malaise, fever, and cough for one week. She did not appear ill or distressed apart from an irritating dry cough. The pulse rate was 70 and temperature 99° F. (37.2° C.). Examination of the chest revealed diminished movement and dullness at the left base, with inspiratory rales and diminished air entry. A diagnosis of pneumonia was made and she was given 500,000 units of procaine penicillin intramuscularly daily for the next five days. As she was not really ill no dramatic improvement occurred and the signs cleared slowly. By July 7 the chest was free of abnormal signs. The results of investigations were as follows. On June 23 the white cell count totalled 6,500 and the E.S.R. was 40 mm. in one hour. A radiograph taken at the patient's home showed "extensive consolidation of basal segments of the left lower lobe with some associated collapse" (Dr. P. S. G. Campbell). On July 17 her chest x-ray film was clear. Blood was sent to the Virus Reference Laboratory (on the ninth day of illness), and the report was that the serum reacted to a titre of 1/160 against the psittacosis group of viruses. This was thought to indicate a specific infection. Serum on the eighteenth day of disease reacted against a titre of 1/80. Frei's test on July 4 was negative. Questioning revealed that the patient had played with a bird's nest one week before the onset of illness. No other contact with birds was elicited.

B. Probable Aspiration Pneumonia (21 Cases).

In these cases infected mucus from the upper respiratory tract, acting as a bronchial embolus, producing an area of collapse and infection, was considered the probable cause. In 18 cases there was a definite preceding history of an acute upper-respiratory-tract infection, and in three there was a recurring catarrhal condition causing a persistent cough, but

TABLE II

Age:	0-4	5-9	10-19	20-29	30-39	40-49	50-59	60-69	70 and Over
No. of cases..	3	8	2	—	3	4	—	1	—

with no organic changes in the lungs. The age distribution is shown in Table II. It will be seen that most cases occurred in the under 10 group, and those were inclined to be "catarrhal children." Twelve cases occurred in the winter months, 8 in the summer, and one in the autumn. There were 10 males and 11 females. The areas of lung affected were right base in 9, left base in 10, right upper zone in 1, and left mid-zone in 1.

The condition presented with a gradual onset (although on four occasions it was sudden) following an upper-respiratory-tract infection. The chief complaint was the irritating cough and some degree of general malaise. No patient was considered to be very ill. Temperature varied from normal to 102° F. (38.9° C.) and there was a relative bradycardia. The signs were identical with those described in the preceding group. These signs cleared in from four days to two months, the average being twelve days.

Treatment did not appear to influence the course of the disease except where there was more pronounced constitutional upset; then penicillin was given and the toxæmia was rapidly controlled. The white-cell counts varied from 25,000 to 5,000 (the average of eight cases being 8,000), and the average E.S.R. figure (for six cases) was 10 mm. in one hour. Only *N. catarrhalis* and *Str. viridans* were grown from six sputa. All virus complement-fixation tests were negative (five cases), cold agglutinins were present in one case out of eight tested. Radiographs were taken in 17 cases either at home or at hospital. In 12 of these some abnormality was detected. This abnormality consisted of small dense patches in the affected areas. Associated collapse was reported in four children. Hilar glands were enlarged in three instances.

C. Possible Specific Cause (at present unidentified) (14 Cases)

The clinical picture was identical with that of the previous group with the exception of a really sudden onset, no preceding catarrhal condition of the upper respiratory tract, and more severe constitutional reaction. There were seven males and seven females. The age incidence is shown in Table III.

TABLE III

Age:	0-4	5-9	10-19	20-29	30-39	40-49	50-59	60-69	70 and Over
No. of cases..	—	5	3	1	1	2	—	1	1

The signs cleared in 4 to 21 days, with an average of 7.5 days. In 10 cases the right base was affected, in four the left base, and in one the left mid-zone. X-ray findings showed abnormalities in three out of seven cases examined. A more diffuse opacity was observed. Sputa from seven cases were found to contain the usual organisms of the upper respiratory tract, but in three cases *Staph. aureus* was found and in one haemolytic streptococci. The white-cell counts varied from 5,000 to 13,000 (average 9,000). Virus complement-fixation tests were negative in six cases. Cold agglutinins were also absent. Three cases were treated with aureomycin, 500 mg. six-hourly, but in only one was a dramatic change noted. Penicillin in six cases was again found to control the systemic upset, but signs persisted. The remainder were treated symptomatically. There was no mortality and no patient was admitted to hospital.

Differential Diagnosis

The question of whether these conditions might be due to any other specific state must be considered. The possibilities which arise are carcinoma of the bronchus causing bronchial obstruction with collapse and inflammation of the affected lung segment. Follow-up in this series has excluded this possibility. Pulmonary tuberculosis of the progressive type was also excluded by follow-up. Tuberculin jelly skin tests were carried out on all the children and were negative (two positive reactors with similar signs were excluded from this report). Loeffler's syndrome has also been considered, but in the 11 patients who had a differential count there was no evidence of any eosinophilia.

Comment on the Pneumonias

Some would place these pneumonias in the so-called "primary atypical" group. This term, however, is a most unfortunate one, as it implies some standard condition which must be regarded as a typical pneumonia and also that the

condition is due to a specific agent. A much more reasonable explanation is that this type of condition can be brought about in a number of different ways. Is it in fact a new disease, or was it recognized in the past and known as "congestion of the lung" or "bronchial catarrh"? It is being seen more frequently in hospital practice. Reimann (1943) stated that these conditions account for 70% of all pneumonias; Scadding (1951) showed how the incidence of pneumococcal pneumonia had dropped from 70 to 40% of all pneumonias, and that in 1949-50, at the Hammersmith Hospital, 20% of pneumonias were due to viruses, and in 23% the aetiology was inconclusive. In my series this group accounted for 90% of cases.

As has already been stated, there are probably multiple causes for this syndrome. It has already been proved that viruses—for example, psittacosis and rickettsia—can cause this type of condition. Influenza presents a more difficult problem. In fatal cases of influenza with lung involvement secondary infection with bacteria is usual, and in "influenzal pneumonias" sputum culture usually grows organisms which might be pathogenic. In addition, influenza often begins as a catarrhal condition of the nose and throat; and it is possible that this catarrh might produce bronchial embolism. Therefore it is doubtful whether the influenza virus is the only factor concerned. A most important possibility is that of *bronchial embolism* by mucus from catarrhal conditions of the upper respiratory tract. Animal experiments have shown that the introduction of viscid mucus into the trachea is followed by collapse and infection of areas of the lungs. Quinn and Meyer (1929) showed that if radio-opaque oil is placed in the nose of a normal sleeping subject this oil will be found in the lungs the following morning.

It was Brock (1946) who stressed the great importance of bronchial embolism and gravitation in the causation of lung abscess. Nicholson (1950) put forward the view that bronchial embolism accounted for a whole range of conditions, from benign aspiration pneumonia (similar to cases described above) through an intermediate non-specific suppurative form to acute lung abscess. Lees (1950) described pulmonary lesions resulting from probable blockage of the bronchi by tenacious mucus in whooping-cough.

Ramsay and Scadding (1939) described a series of 21 cases with radiological changes which were seen at a chest clinic, and they ascribed these to a similar cause. Scadding (1948, 1951) and Robertson and Morle (1951) have attempted to show that the "primary atypical pneumonia" syndrome is in fact an aspiration pneumonia. This particular series includes 35 cases with a probable viral influence, 21 which were probably aspiration pneumonias, and 14 which may have had a specific cause, either viral or bacterial, that at present is unidentifiable.

Acute Pleurisy

In the period under discussion six cases (three male and three female) of apparent primary pleurisy were seen in which, apart from a pleural rub at the corresponding painful region, there were no other abnormal chest signs. The patients' ages ranged from 20 to 75, but four were under 40. Five were x-rayed in the acute phase, and in all a small patch of consolidation was found in the affected area. On follow-up this cleared within two weeks, with the exception of one case in which it persisted for two months. Follow-up did not reveal any tuberculous process in any of these patients. The question arises whether pleurisy is ever a primary condition and whether it is always secondary to some lung lesion. In this same period three cases of acute tuberculous pleurisy were seen in young adults.

Severe Asthma

This accounted for 10 cases of "acute chest." It produced marked distress in the patient and gave great anxiety to both relatives and medical adviser. The sex distribution was equal and the age incidence varied from 10 to 65. Two patients required treatment in hospital.

Spontaneous Pneumothorax

This is included as it accounted for five cases. It had a sudden onset with pain and a variable degree of dyspnoea. In four there was no previous or subsequent chest trouble. The fifth patient, an elderly lady of 68, had a history of recurring coughs and developed a valvular pneumothorax. She died in hospital.

Conclusion

It will be seen that these numerous cases from a single general practice present a different picture from those seen in hospital work. This is, of course, due to the fact that only the cases which cannot be managed at home are sent into the hospital wards. This series was relatively benign—thus only two patients died in this group and nine required admission to hospital. This type of case has received almost no attention in the past, and it is felt that there is a need for further study of this group. A simpler and more precise method of classifying acute chest diseases, which should be based on aetiological grounds, is also necessary.

Summary

A clinical report is presented on 164 cases of acute chest disease as seen in a general practice over a period of two years.

Acute bronchitis accounted for 66 cases. Subdivision into primary and secondary groups is made.

Seventy-seven cases of pneumonia were seen. Only three of these were the primary "classical" pneumonias. The common type was a localized area of inflammation with no clinical evidence of consolidation. On the aetiology of this subgroup it is difficult to dogmatize, but three possibilities are put forward: (1) infection due to an identifiable virus; (2) an infection due to a specific unidentifiable agent; or (3) the condition might in fact be an aspiration pneumonia.

The other conditions which produced the acute chest were pleurisy, severe asthma, and spontaneous pneumothorax.

A plea is made for a simpler and more logical classification of acute chest diseases, and the need for further research on these conditions is stressed.

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A radical change in the footwear of this year's British Everest expedition is announced. Instead of the familiar heavily nailed leather climbing-boots, the assault parties will wear a much lighter boot with soles of artificial rubber and glacé kid uppers. The boots are insulated inside with a layer of kapok nearly an inch thick. It is pointed out that lightness and warmth are far more important qualities than durability and water-resistance for the Everest assault. The boots, which have already been tried in the Alps, will be required only for a few days' use, and at the temperatures high on Everest water is unlikely to be encountered (*The Times*, January 6).

DISSECTING AORTIC ANEURYSM: A VARIED CLINICAL PICTURE

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Dissecting aneurysm was first described by Morgagni in 1761. In 1934 Shennan published his exhaustive monograph on the subject, having been able to collect over 300 cases from the literature. A significant point arising from this report was that only six cases were diagnosed before death.

The correct ante-mortem diagnosis may present much difficulty for two reasons. (1) Bizarre symptoms and signs may occur, and disease of other systems may be simulated. (2) It is a somewhat uncommon condition in hospital practice. Many cases die suddenly and therefore fall into the hands of the coroner. In San Francisco, Mote and Carr (1942) compared the incidence at coroners' post-mortem examinations with that at hospital post-mortem examinations. They found three times as many in the former as in the latter, and a similar state of affairs may well hold in this country.

The clinical picture which has come to be recognized as typical of aortic dissection is that of the sudden onset of severe tearing thoracic pain accompanied by shock and radiating down both legs. This view of dissecting aortic aneurysm may have obscured the correct diagnosis in many instances in the past, and patients may have been subjected to operative intervention when this condition was not borne in mind. It is the purpose of this note to suggest that aortic dissection may often be a silent process and may progress with only minor symptoms for months or years.

Five cases are described, illustrating the paucity of symptoms and the tendency to simulate other diseases.

Case 1

A 53-year-old woman was admitted to hospital with 30 years' history of epigastric pain relieved by food and alkaline powders. For two to three years previously the pain had radiated through to the back. Two weeks before admission she developed a sudden weakness in the left arm and left leg. Twelve hours before admission she had a severe attack of epigastric pain radiating through to the back. She persisted that the sudden pain which occurred before admission was of the same nature as that which she attributed to her dyspepsia.

On examination her general condition was reasonably good. The pulse was regular and the blood pressure 230/130. The apex beat was not displaced. A systolic murmur was present over the praecordium. There was definite muscular weakness of the left arm and leg, with exaggerated reflexes and a doubtful plantar response. There was no abnormal finding in any other system.

Investigation.—Haemoglobin, 56%; white cells, 15,000 (polymorph leucocytosis). Urine: albumin +, granular and hyaline casts, red blood cells present. The blood urea was 70 mg. per 100 ml. The Wassermann reaction was negative. An electrocardiogram showed inversion of T₁, T₂, and T₃ R 4.

The epigastric pain persisted, resisting all the usual methods of treatment. Strong sedation had to be employed. Five days after admission a small effusion was detected clinically at the left lung base, and this extended up to the spine of the scapula during the next 48 hours. X-ray examination of the chest revealed almost complete opacity of the left hemithorax. Repeated attempts