this case is that the walls of the oesophagus were tissue-paper thin, and at the original operation seemed to consist of mucous membrane only, whereas well-formed and apparently normal oesophagus had developed in the three-year interval before excision of the stricture. The mass of scar tissue at the stricture was incredibly hard, forming a block about 2.5 by 2 by 2 cm., and it was not surprising that it did not respond to frequent dilatation.

Summary and Conclusions

The experience of one team with 17 cases of atresia of the oesophagus is described.

Special points in surgical technique and care of these babies are stressed. Results in relation to the findings are given.

It is felt that the knowledge of the simple diagnostic procedures, the possibilities of operative repair, and the location of the nearest unit willing to undertake these cases should be widely disseminated among general practitioners and midwives.

Experience with these cases is invaluable, from the medical and nursing points of view, in reducing the mortality and morbidity rates. Therefore cases should if possible be sent to an experienced team, and the extra journey with the baby is far outweighed by the advantages of getting the case to such a unit.

Great care in performing the anastomosis pays handsome dividends and speed is of no importance.

Special nursing round the clock is essential for these babies in the first few post-operative days.

We thank Mr. Kent Harrison for allowing us to include two of his Cambridge cases in this series. We are grateful to Dr. Douglas Gairdner and his paediatric team and the anaesthetists at Addenbrooke's Hospital for their encouragement and help, without which these patients would not have survived. We thank Miss Sylvia Treadgold for the drawings.

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An "emergency" identification card for the protection of patients on long-term anticoagulant therapy is now available to physicians from the American Heart Association and its affiliates. The card was developed as a result of requests from physicians seeking to protect their patients on anticoagulants in case of accident, dental surgery, or other treatment that may induce bleeding. In case of emergency the card advises that a doctor be called, since the patient may require an antidote. The card contains space for the name, address, and telephone number of the patient's physician, the type of anticoagulant prescribed, and the patient's blood group. Samples of the card are obtainable from the American Heart Association, 44, East 23rd Street, New York City.

PRIMARY ATYPICAL PNEUMONIA AND HIGH SEDIMENTATION RATES

BY

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The diagnosis of atypical pneumonia originated at the time when, following the introduction of antipneumococcal sera and sulphonamides, a group of cases of pneumonia was distinguished from the majority by a failure to respond to these forms of therapy (Finland and Dingle, 1942). Since then further microbiological, serological, and radiological investigations have resulted in the recognition of several subdivisions of this group which are aetiologically, if not clinically, distinguishable. These include the aspiration pneumonias and the pneumonias associated with rickettsial Q fever, ornithosis, the acute specific fevers, and infections by the influenza viruses and the adenoviruses.

The serum of many of the cases of atypical pneumonia which remain unaccounted for contains antibodies which agglutinate homologous red cells in the cold (4° C.). The constitutional upset in these latter cases is often greater, and the radiological changes are often more extensive, than would be expected from the pulmonary symptoms and signs, which are often slight. The white blood count is normal, or only slightly increased, in the early stages of the illness, and the serum may contain agglutinins against the streptococcus M.G. The dramatic response to penicillin therapy usually seen in pneumonia does not occur, but, in some epidemics at least, treatment with one of the tetracycline antibiotics has been found to be effective. The condition has been transmitted by bacteria-free filtrates of respiratory tract secretions to volunteers, and from them to a second series of experimental subjects (Commission on Acute Respiratory Diseases, 1946), but no virus has yet been generally agreed to be responsible. It is for this condition that many now reserve the description of primary atypical pneumonia.

Raised titres of cold agglutinins are sometimes found in other respiratory diseases such as influenza, ornithosis, and pneumococcal pneumonia. These conditions are unlikely to be misdiagnosed as primary atypical pneumonia if titres of cold agglutinins of 1 in 128 or more are required before the diagnosis of the latter is accepted, and even more unlikely if a fourfold increase in titre is observed in serial specimens. Many of these other pulmonary disorders can also be excluded by further serological or bacteriological tests.

Observations

As part of the routine investigation of nine sporadic cases of primary atypical pneumonia, in which the clinical diagnosis, based on the features described above, was confirmed by the demonstration of significant titres of cold agglutinins, estimations of the erythrocyte sedimentation rate (E.S.R.) were made by the method of Westergren (1921). Readings were made of the fall of the red cells in a 200-mm. tube after one hour. Seven of the nine patients had an E.S.R. of over 90 mm. when first estimated, and the E.S.R. of another of these patients was over 80 mm. on admission, rising later to 98 mm. (Cases 1-9, Table I). Four of the nine

TABLE I.—*Sedimentation Rates and Cold Agglutinin Titres in 16 Cases of Primary Atypical Pneumonia*

Case No.	Age and Sex	E.S.R. (in italics) in mm. in First Hour (Westergren) and Cold Agglutinin Titre						
		Weeks of Illness						
		1	2	3	4	5	6	7
1	52 F		<i>117</i> 1/512	<i>116</i> ,384	<i>30</i> 1/128			
2	27 F		<i>91</i> 1/512		<i>16</i> 1/32			
3	25 F	83	<i>85</i> 1/512	98	<i>80</i> 1/64			
4	33 F		<i>120</i> 1/128	<i>12</i> 1/256				
5	64 F			<i>126</i> 1/4,096		<i>75</i> 1/4,096		<i>58</i> 1/32
6	36 F				91		<i>62</i> 1/128	<i>106</i> 1/32
7	34 F		<i>138</i> 1/1,024	<i>40</i> 1/1,024	<i>7</i> 1/256			
8	58 M		10	<i>40</i> 1/640	<i>1</i> /250	<i>40</i> 1/320	<i>33</i> 1/160	(8th week) <i>25</i> 1/64
9	78 M		<i>105</i> 1/80		<i>95</i> 1/128	<i>122</i> 1/8		
10	61 F	<i>126</i> 1/1,280		<i>92</i> 1/640	<i>97</i> 1/160	<i>92</i> 1/320		
11	78 M		<i>100</i> 1/16		<i>90</i> 1/128	<i>1</i> /64		
12	67 M		<i>102</i> 1/512	63	<i>49</i> 1/256	<i>31</i> 1/128		
13	72 M	<i>115</i> 1/64			<i>80</i> 1/128	<i>60</i> 1/128	<i>43</i> 1/32	
14	60 M	<i>98</i> 1/256		<i>1</i> /128	<i>79</i> 1/32		<i>59</i> 1/64	<i>61</i> 1/32
15	65 M			<i>54</i> 1/1,024	<i>1</i> /1,024	<i>5</i> 1/256		
16	81 M		<i>101</i> 1/128	93	<i>104</i> 1/64	<i>120</i> 1/64		

patients showed readings of 120 mm. or more. A further patient (Case 10) with hypertensive heart disease, who was admitted with a recurrence of left ventricular failure apparently precipitated by a respiratory infection, had an E.S.R. of 126 mm., which prompted the examination of the serum for cold agglutinins. These were present in a titre of 1 in 1,280.

Such high sedimentation rates are not found with great frequency in acute pneumonia in present-day hospital practice. Retrospective examination of the notes of 160 patients with acute pneumonia uncomplicated by non-cardiorespiratory disorders (group II), showed that 22 (14%)

FIG. 1.—Distribution of the Westergren sedimentation rates in mm. in the first hour of 160 patients with acute pneumonia (Group II).

had an E.S.R. of over 90 mm. at the time of admission (Fig. 1). A rate of over 120 mm. in one hour was exceedingly rare. Four of these 22 patients were thought on clinical grounds to have a virus pneumonia.

A further 77 consecutive patients with acute pneumonia (group III) had estimations of the E.S.R. and of the cold agglutinin titre made soon after admission to hospital. The cold agglutinin titre was measured by a method similar to that described by Dacie (1956). Any patient with an E.S.R. of over 80 mm. or whose serum contained cold agglutinins was re-examined on one or more occasions. Six patients (8%) showed significantly raised and varying titres of cold agglutinins (Cases 11–16, Table I), and five of these had an E.S.R. of over 90 mm. (Fig. 2). These patients with high

FIG. 2.—Distribution of the Westergren sedimentation rates in mm. in the first hour of 77 patients with acute pneumonia (group III). Shading shows cases with high cold agglutinin titres.

cold agglutinin titres were often not clinically distinguishable from the other cases of pneumonia in this third group, and are not therefore completely comparable to the sporadic cases of atypical pneumonia (Cases 1–9). Over one-third of the cases in groups II and III with very high sedimentation rates but without raised cold agglutinin titres had some complicating condition, such as pleural effusion or bronchiectasis, which might have explained the finding of higher rates than are usually seen in pneumonia.

Discussion

It must be admitted that many writers in the years 1930–9, including Motzfeldt (1932) and Douglas-Wilson (1939), often found Westergren sedimentation rates above 90 mm. in cases of lobar pneumonia during the first week of illness. This frequency of high sedimentation rates is at variance with present experience. Douglas-Wilson (1939) found, however, that such high levels were not reached until the third day of the illness, at which stage nowadays antibiotic therapy usually will have controlled the infection. Two other possible reasons for the discrepancy are that diagnostic criteria have broadened and that the type of case admitted to hospital has changed.

In some of our cases of atypical pneumonia minor symptoms had been present for many days before the condition had become serious enough to lead to their admission to hospital for treatment. This gradual onset, and therefore the later stage in the illness at which the E.S.R. was first estimated, might be one of the factors responsible for higher rates being found in this group, but it would not explain why such very high levels were found in almost every case.

The high sedimentation rates found in our cases are not due to excessive aggregation of red cells consequent upon the action of the cold agglutinins present, for the following reasons: (1) The association was not invariable. (2) The levels of the E.S.R. did not appear to be directly or constantly related to the titres of cold agglutinins; thus Case 4 showed a fall in the E.S.R. from 120 mm. to 12 mm., while the cold agglutinin titres rose from 1 in 128 to 1 in 256, and Cases 5 and 7 showed no change in their titres of cold agglutinins while their E.S.R. fell by more than 50 mm. (3) Estimations of the sedimentation rate at different temperatures show that the rate remains high even if the test

is carried out at 37° C., at which temperature the agglutinins are inactive, and that the rate bears no consistent relationship to the temperature (Table II). (4) Cold agglutinins are

TABLE II.—Westergren E.S.R. at Different Temperatures

Case No.	E.S.R. in mm. in First Hour at Temp. of:		
	4° C.	Room Temp.	37° C.
3		98	70
7		138	163
9		105	125
10	12	92	80
13	80	80	103
16	55	93	82

adsorbed on to the red cells when separation of plasma and cells is carried out at 4° C. In one case, after the patient's red cells, separated in this way, were mixed with normal plasma, a normal Westergren sedimentation rate was obtained, but when normal cells were mixed with the patient's plasma, after separation at either 4 or 37° C., a high E.S.R. was obtained.

It is not denied, however, that high titres of cold agglutinins can sometimes be the immediate cause of rapid sedimentation at room temperature. Jessen and Bing (1940) have reported such a case in which active agglutinins were present in sufficient concentration at 19° C. to produce an E.S.R. of 106 mm., while the E.S.R. at 40° C. was only 55 mm.

Hardwicke and Squire (1952) have shown that, in the absence of gross changes in red cell shape and size, the E.S.R. depends principally upon alterations in one or more of the plasma fractions: fibrinogen, α_2 -globulin and γ -globulin. A significant elevation of the plasma fibrinogen was found in all nine of our cases in which this was estimated, and in most cases there was an increase in serum globulin and a slight decrease in serum albumin (Table III).

TABLE III.—Blood Proteins in Cases of Primary Atypical Pneumonia

Case No.	Serum Proteins in g./100 ml.			Plasma Fibrinogen in g./100 ml. (Normal: 0.2-0.4)
	Total	Albumin	Globulin	
3	6.7	4.9	1.8	0.56
5	6.7	4.2	2.5	
7	6.3	3.4	2.9	
9	7.0	2.1	4.9	0.74
10				0.52
11	7.1	3.2	3.9	0.76
12	5.1	2.3	2.8	0.6
13	6.1	2.6	3.5	1.03
14	7.4	3.3	4.1	0.82
15	6.2	2.6	3.6	0.63
16	6.8	3.4	3.4	0.69

Electrophoretic examination of the serum of each of Cases 8 to 16 showed an increase in globulins, particularly in the γ and less obviously in the α_2 fractions. Some cases of pneumonia with very high sedimentation rates but without raised cold agglutinin titres showed similar plasma protein abnormalities, except that an increase in α_2 -globulin was seen more frequently. As Cases 8 and 15, in which very high E.S.R.s were not detected, also showed plasma protein changes apparently similar to those of the other cases of atypical pneumonia, the way in which these changes produce the high sedimentation rate must be a subtle one. The situation does not appear to be comparable to that in another virus infection, lymphogranuloma venereum. In this condition the very rapid rates sometimes found are related to the high concentration of γ -globulin which occurs (Waldenström *et al.*, 1951).

MacLean and Parnell (1955) have described a case of pneumonia associated with a high titre of cold agglutinins and with a haemolytic anaemia; a similar case has been reported by Stewart and Friedlander (1957). The Westergren sedimentation rates were 158 and 156 mm. in one hour respectively. The pneumonia in one of our patients (Case 7) was also complicated by haemolytic anaemia; her E.S.R. was 138 mm. at room temperature and 163 mm. at 37° C. The extremely high figures in these three patients are pos-

sible because of the reduction in the packed cell volumes, but the mechanism of the rapid sedimentation would otherwise appear to be the same as in the rest of our cases. Shaw and Fry (1955) reported that the highest sedimentation rate found in 66 cases of acute infections of the chest was 113 mm. in one hour in a case of virus pneumonia with raised cold agglutinin titre; a personal communication confirms that the Westergren method was employed. The great majority of references in the literature to sedimentation rates in atypical pneumonia with high cold agglutinin titres relate, however, to estimations by the Wintrobe or similar methods. The rates recorded were normal or only moderately increased.

Simultaneous estimations of the sedimentation rate by both the Westergren and Wintrobe methods were carried out in each of six of our more recently observed patients (Table IV). The disparity in the results of the two methods

TABLE IV.—Westergren and Wintrobe Sedimentation Rates of Same Blood Samples

Case No.	Sedimentation Rate in mm. in First Hour	
	Westergren	Wintrobe
9 { i ii	105 95	13 7
10	92	36
11 { i ii	100 90	54 14
13 { i ii	115 80	43 40
14	79	55
16 { i ii	101 93	43 31

is obvious, in particular two patients (Cases 9 and 11) had normal Wintrobe rates when the Westergren rates were over 90 mm. in one hour.

The occurrence of similarly anomalous results of sedimentation rate estimations, an increased rate by the Westergren method being accompanied by a normal or only slightly increased Wintrobe rate, has been reported by Gilmour and Sykes (1951), Goldberg *et al.* (1952), Goldberg and Conway (1952), and Alexander (1953). The Westergren method in each case provided a better index of the clinical state of the patient. Factors which appear to play a part in producing the discrepancy include the chemical properties of the anticoagulant used (Goldberg *et al.*, 1952), the method of filling the Wintrobe tube (Alexander, 1953), and dilution of the blood (Lewi, 1954). The height of the column is especially important when very high sedimentation rates are being looked for; using the Wintrobe method, it is impossible with non-anaemic blood to obtain a fall of more than about 55 mm. owing to the packing of the red cells in the shorter (100-mm.) tube.

The association which we believe exists between very high sedimentation rates and primary atypical pneumonia is not related to one particular epidemic, but to the disease in general, because Cases 1-10 occurred sporadically, without obvious epidemiological connexion with one another. They were seen in two hospitals in London and one in Southampton over a period of five years. We do not suggest that all cases of primary atypical pneumonia show a very high sedimentation rate, nor that every case of pneumonia with such an E.S.R. will be found to have a raised cold agglutinin titre. The finding of an E.S.R. of over 90 mm. in one hour in all but three of the 16 cases of pneumonia with significantly raised cold agglutinin titres reported here is, however, remarkable enough to justify further statistical studies, and meanwhile to lead to this diagnosis being considered in any patient with an unusual pneumonia who has a very high sedimentation rate.

Summary

Thirteen out of 16 patients with pneumonia associated with significantly raised titres of cold agglutinins in the serum had Westergren sedimentation rates of over 90 mm. in one hour when first examined.

Nine of these patients were sporadic but consecutive cases of clinically atypical pneumonia, of whom seven had an E.S.R. of over 90 mm. on admission and four had rates of 120 mm. or more at some stage of their illness. These levels are much higher than those commonly seen in acute pneumonia.

The high sedimentation rates are not due to the excessive aggregation of red cells produced by the cold agglutinins. The plasma protein abnormalities which occur do not appear to be unusual or specific to this form of pneumonia.

Such very high rates can be demonstrated only by the Westergren method; the Wintrobe method may give disproportionately low or even normal results.

If a patient with acute uncomplicated pneumonia has an E.S.R. of over 90 mm. the diagnosis of primary atypical pneumonia should be considered, especially if there are any other unusual features.

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PRIMARY ARACHNOID CYSTS

REPORT OF TWO CASES

BY

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Richard Bright (1831) gave a short account of cysts arising in relation to the surface of the brain, and there is little doubt that some of them were cysts of the arachnoid, as the accompanying reproduction of his original illustration shows. A long time elapsed before any further noteworthy contributions to this subject were made.

de Martel and Guillaume (1930) reported seven cases of arachnoid cysts in the posterior fossa and stressed the sudden onset of headache with neck stiffness, suggesting the diagnosis of meningitis or subarachnoid haemorrhage. Mullin (1932) described two cases in which a cyst in the cerebellopontine angle produced a syndrome resembling that of an acoustic neurinoma: arachnoid cysts are commonly found associated with acoustic tumours, but in Mullin's cases the internal auditory meatus was clearly exposed and a tumour excluded. The membrane was not examined microscopically; therefore it is possible that the cysts were ependymal in origin as in a case described by Hardman and Jefferson (1938).

Characteristics

Tönnis, at the meeting of the Society of British Neurological Surgeons in Berlin in June, 1937 (unpublished), described his experience of eight cases of "congenital cysts of the cisterns" simulating intracranial tumours. This was a most valuable contribution in which the characteristics of arachnoid cysts were defined as follows: The membranes correspond to the meninges in their structure; signs of inflammation in the cyst wall are absent; the cystic fluid is clear and usually xanthochromic, and coagulates spontaneously after removal.

McConnell and Douglas (1939), writing on intracranial cysts, included examples of what they termed "subarachnoid cysts." Their illustrations make it clear that the outer wall of the cysts was composed of arachnoid, and the inner wall of pia mater.

Three examples were cited: in the first the cyst occupied most of the right anterior fossa, in the second the anterior half of the right temporal fossa, and in the third the midline of the posterior fossa. In all three instances it appeared as though the underlying brain was deficient. Macroscopically the arachnoid and pia forming the cyst walls were normal. The fluid of the cysts was not analysed and there was no microscopical examination of the membranes.

Reproduction of Richard Bright's illustration of an arachnoid cyst in the left Sylvian fissure. (Kindly lent by Mr. James Hardman.)

Horrax (1924) reported some cases of Cushing's of mid-line cysts of the posterior fossa causing increased intracranial pressure. The cysts, however, appeared to be dilatations of the cisterna magna, and they were attributed to inflammatory adhesion of the membranes, though evidence of inflammation was lacking in most of the cases; examples of cysts in the cerebellopontine angle associated with otitis media were also encountered. Cases similar to Cushing's were described by Allen and Corkill (1937); in two of them the cysts were apparently separate from the cisterna magna and were therefore true arachnoid cysts. These authors also favoured the inflammation theory, though no histological or other evidence was given to support it.

Successful removal of a circumscribed cyst of the leptomeninges involving the spinal cord and cauda equina was reported by Spiller, Musser, and Martin (1903). The walls of the cyst were described as transparent, but again no histological examination was carried out; in a subsequent paper, however, Spiller (1909) attributed such lesions to inflammation, and hence the origin of the term "circumscribed serous spinal meningitis." Spiller was supported in his belief by Horsley (1909), who reported his cases under the title of "chronic spinal meningitis"; he suggested that in some instances syphilis or gonorrhoea was the responsible infection.

Stookey (1927) believed there was no justification for the view that arachnoid cysts are of inflammatory origin, though he reported his own case under the title of "Adhesive Spinal Arachnoiditis."

Scrutiny of case records in the literature lends abundant support to Stookey's opinion, but histological reports on the membranes are rarely given. Therefore it seems worth