PRIMARY ATYPICAL PNEUMONIA
THE AVERAGE CLINICAL PICTURE BASED ON 101 CASES *

PERPETUO D. GUTIERREZ, M.D.
FRANCISCO F. TANGCO, B.S., M.D.
AND
DOMINICO RUIZ, M.D.
Department of Medicine, College of Medicine,
University of the Philippines and the
Philippine General Hospital

It is only recently that most of us have given acceptance to that clinical entity first alluded to by Allen(1) in an analysis of 50 cases at Fort Sam Houston in 1935, characterized by a benign course, few physical signs, and X-ray evidence of a localized inflammatory process in the lungs—a form of respiratory infection, to designate which, he used the term "acute pneumonitis"; and which in 1938, Reimann(2) reported as atypical pneumonia, citing eight cases occurring in Philadelphia.

In 1942, Sison and co-workers read a paper(3) on the first cases reported locally. There was a temporary interest in the subject, but the majority of clinicians either were not convinced or considered the disease an extremely rare condition.

However, reports continued to come in of cases diagnosed as "lobar pneumonia" but which varied from the typical picture in certain respects. The most important difference was the gross disparity between the condition of the patient and the detectable physical findings. The patients were highly febrile, looked toxic, had a distressing cough and yet the internist, seeking verification through a meticulous physical examination, would find only some impairment of resonance, a few scattered rales and very little else. Moreover, the course of the disease was much shorter than the typical lobar pneumonia and more often than not, defervescence would be by lysis instead of by crisis. The difference in response to sulfonamides and penicillin was also striking.

Another facet of the problem became evident during the military campaign for the liberation of Manila. Shortly after the arrival of the American forces, cases of "atypical lobar pneumonia" began

* Read at the Thirty-ninth Annual Meeting, Philippine Medical Association, May, 1946.

to be reported in increasing numbers. In the light of the apparent increased incidence of this disease abroad during World War II(5) and of its admittedly high incidence in the Army(6) two questions may well be asked. Did the local population become more susceptible to the infection as a result of the breakdown of peacetime health safeguards, physical suffering and nutritional difficulties? Or were the people infected by carriers among the U. S. armed forces?

In an effort to throw some light on the main problem, this report is presented. We have studied the records of the 234 cases with the diagnosis of "lobar pneumonia" admitted to the Philippine General Hospital from July, 1945 to March, 1946. Of these, 101 cases deviated so clearly from the usual picture of lobar pneumonia and conformed to another recognizable pattern that we felt justified in setting them up as a distinct group.

Smith(7) has well summarized the present status of the entity, and we quote, "The increasing prevalence of an atypical form of primary pneumonia has commanded more and more attention . . . Much of the literature on the subject is puzzling or contradictory. There is no 'single criterion—clinical or laboratory—which characterizes the syndrome.'(8) Diagnosis is arrived at by a process of eliminating similar diseases of known etiology and attempting to check the patient's signs and symptoms against those of groups of cases previously reported. Unfortunately the literature on the subject is still too fresh for the relative value of each sign and symptom to have been worked out. Yet from the present maze of apparent contradictions among reported groups of cases, there is emerging a clinical picture sufficiently clear cut to permit reasonable diagnosis and tentative classification." Furthermore, these "several published accounts of a comparatively benign but special variety of pneumonitis conform so nearly to one pattern that there is little room for doubt that the disease should be considered as an entity."(9)

Nomenclature: We shall refer to the disease in this paper as primary atypical pneumonia. The designation assigned by the Commission on Pneumonia of the U. S. Army which probably applies to the largest standardized group of cases to be reported is "Primary, Atypical Pneumonia, Etiology Unknown."(10) It has also been referred to in literature as "Acute Pneumonitis,"(8) "Acute Respiratory Tract Infection, Type A," "Acute Interstitial Pneumonitis,"(11) "Bronchopneumonia of Unknown Etiology, Variety X,"(9) "Current Bronchopneumonia of Unusual Character and Undetermined Etio-

logy," 'Virus-type Pneumonia," "Viral Pneumonia," "Virus Pneumonia," and several others. Campbell et al believes it more nearly correct to call it "Acute Bronchiolitis with Associated Atelectasis."(12)

Predisposing Factors: In our cases, exposure to cold or rain was the most commonly mentioned factor. Fatigue also frequently preceded the attack, especially if there was subsequent exposure. Upper respiratory catarrh in the form of the "common cold" has apparently served to predispose some patients to the disease. Malnutrition did not seem to play an important role, as seventy four per cent of the cases were fairly nourished patients, about twenty per cent poorly nourished, and about six per cent were well nourished. None were emaciated.

Etiology: The causative organism of primary atypical pneumonia has not been identified. Recent and continued studies of this condition makes one feel that primary atypical pneumonia may prove to be not a single disease entity, but rather a clinical syndrome with multiple etiology.(14) A number of etiological agents have been mentioned—a number of known viruses, particularly of the psittacosis group,(15) rickettsiae, fungi, a protozoan that produces toxoplasmosis,(16) Coccidioides immitis,(17) and certain bacteria. Atypical signs and symptoms of pneumonia have also been observed during the migration of some of the parasitic merozoites.(18) A streptococcus (identified as No. 344) has been isolated in 2 cases of primary atypical pneumonia which terminated fatally, and it was shown that in fifty-five of one hundred and one cases, the patients had an increased titer to this organism.

Any of the aforementioned agents could produce a similar clinical syndrome but they can be excluded with reasonable certainty in the great majority of cases.(5) On the other hand, it was demonstrated that bacteria free filtrates obtained from sputum and throat washings, presumably containing a virus, can induce primary atypical pneumonia in man.(19) The results of the mass of laboratory experiments directed at isolating the etiologic agent leads only to the conclusion that primary atypical pneumonia is at least initiated, if not caused in its entirety, by a filter-passing agent, presumably a virus. The role of the bacterium in its causation is obscure.(20) It is possible that a single agent may be the cause of many or even most of the cases in a given outbreak, or in a single locality.(21)

Epidemiology: It is the consensus that primary atypical pneumonia occurs in epidemics. "Moist speakers" or impolite coughers,

birds and animals have been named as sources or transmitters of infections.

A common viral agent may have caused involvement of as high as fifty per cent of the population of some communities.(22) It seems that not all persons exposed to the disease acquire it. Persons with mild types of primary atypical pneumonia gave rise to the severer type in others, and vice versa. Children and infants are thought to be more susceptible than adults, although the higher incidence is among young adults. All ages, however, may be affected.(13)

In our series, one half of the total number of cases was found at ages from sixteen to twenty-five years, and the remaining half distributed among the later years, the number of cases decreasing as the age increased.

Both sexes are affected, but in our series, males were affected about four times as often as females.

Persons who engage in trades which require heavy manual exertion are apparently more susceptible. Fifty-nine of our cases were in the laboring class.

There is a distinct seasonal variation. In America it is most common in the fall and early winter, the incidence being highest during cold, damp, changeable weather, without any relation to the incidence of influenza or the common cold.(13) Our figures show the greatest distribution during those months of sudden changes of temperature, in December and during the transition to the hot months, in February. (Fig. 2). We have at the time of this report no figures for the months not included in this series (April to June).

Outbreaks are fairly common in crowded areas as in armies, schools, orphanages and jails.(13)

Morbid Anatomy: Since our series did not include a single case with a fatal termination, we cannot give a first-hand description of the pathological findings, and we have to depend entirely on what has been mentioned in the literature.

Grossly, the lungs resemble an acute miliary granulomatous process.(23) It is crepitant with isolated areas of pink or gray consolidation that vary in size.(24) Atelectasis and emphysema in other parts may be seen.(13) There may be hemorrhagic areas.(25) Infarcts have been reported.(9)

Microscopically, the fundamental pulmonic lesion is an acute interstitial pneumonitis.(23) Small bronchi, bronchioles and alveoli may be filled by frank pus(26) or a thick exudate of mucus, desqua-

mated cells, monocytes and a few neutrophils and eosinophils. Necrosis and ulceration of the epithelium may be found in the bronchi and bronchioles, with cellular debris filling the lumen.(25) The mucosa of the bronchi is inflamed and congested and bleeds readily.(27) Alveolar tissue is edematous, thickened and infiltrated primarily by monocytes.(24) This mononuclear alveolar exudate is peculiar to the disease.(9) A hyaline-like lining may be seen in the alveoli.(24) There may be metaplasia of the alveoli.(28)

Thrombosis and necrosis of the blood vessels with periarteritic changes may be seen.(24)(29)

Inclusion bodies in the epithelial cells have been seen and described.(25) This, however, is not specific for viral infections, for they may be due to Haemophilus pertussis, pasteurella tularense, toxins, irritative chemicals, or the protozoa of toxoplasmosis.(30)

Lymphangiectasis was invariably found.(26) Bacterial stains of lung sections uniformly failed to reveal micro-organisms in affected alveolar walls, alveolar lumens, peribronchial tissues, lung septa or bronchiolar wall.

Other pathological changes that have been noted were hemorrhage of the adrenal glands;(25) acute splenic tumor;(8) acute follicular splenitis with necrosis of enlarged malpighian corpuscles of the spleen;(13) focal necrosis of the liver;(24) edema of the meninges; congestion of vessels and small focal hemorrhages;(26) mesenteric lymphadenitis;(22) and hyaline necrosis of the diaphragmatic muscles.(31)

Symptomatology: In our patients, cough and fever were the most common presenting complaints. A great number of the patients consulted the physician because of severe headache which apparently did not respond to patent "cures." Chest and/or back pains or generalized body pains were also fairly common complaints. A few patients apparently sought the help of the physician because of persistent fever which had lasted for over two or three weeks.

The incubation period varies from five(32) to twenty-six days.(33) Reimann mentions, however, that it may be as short as one to two days. Fourteen to twenty-one days, or more precisely, seventeen to nineteen days, is believed to be the average duration.(34).

The onset was insidious in thirty-six of our cases and sudden in the remaining sixty-five. Literature is conflicting on this. Some claim an insidious onset as the rule,(14) allowing only twenty-five to thirty-three per cent for cases with sudden onset, while others,

like Daniels, reported a sudden onset in all his cases.(13) Page and Title, however, who gave an eighty-three per cent incidence of gradual onset would consider a lapse of two or more days between the onset of initial symptoms and the patient's hospitalization as gradual.

Fever was the most common symptom in our cases. In eighty-eight cases fever occurred early, as a rule on the first day. In eight it was a late symptom, occurring on the fourth day or even later. In five patients, fever was not noted during the whole course of the disease.

All kinds of temperature curves were observed. It may be high or only moderately so, ranging from thirty-eight to 40°C., or it may be only low-grade throughout. In forty-two cases, the temperature tended to be more or less continuous with only slight fluctuations. In twenty-nine patients it was of a remittent or "swinging" character.(13). In eighteen cases the fever was distinctly intermittent, going down to normal in certain hours of the day only to rise up in a few hours or so. A "dromedary" type of temperature was observed in seven cases, where there was fever for one or several days, normal temperature for two days or so, then another rise with exacerbation of symptoms. Lusk and Lewis' cases as well as some of Adams' presented such a "biphasic curve." In eighteen cases in the series there was a slight rise (up to 37.5°C.) that was observed after the defervescence and the patient had been afebrile and apparently symptom-free for some time.

The fever lasted from two to twenty-six days, the majority (seventy-five cases), being from four to twelve days. It may, however, last for forty-three days or even longer.(2)

Defervescence was by lysis in eighty-four cases, and by crisis in fifteen, the other two cases went home, against advice, still running a temperature.

Cough was the next most common symptom, occurring in eighty-seven of our cases. In only ten of these was cough noticed rather late in the course. Many times it came in paroxysms and tended to be more distressing or disturbing at night. It was usually dry at first but productive later on. Expectoration was mucoid or muco-purulent, whitish, yellowish, or greenish in fifty-nine cases. A brownish color, giving a rusty appearance, was seen in only five cases during the first three days of illness and, after this time, in five other cases. Six cases noticed blood streaks in the sputum instead.

Headache, an important early symptom(7), was rather common and many a time a very early and most disturbing symptom. It usually affected the entire head although some localized it at the frontal or temporal regions. It has been variously described as throbbing, crushing, or tightening, or just a dull aching pain. It was frequently so severe as to impair sleep and appetite and cause restlessness, and may not be relieved by the ordinary analgesics.

Chest and back pain were noticed at the onset in over half of the cases, although in others (twelve cases) it appeared rather late.

In about a third of the cases, the illness was ushered in by chilly sensations and in twenty-three others by actual shaking chills. Some had recurrent attacks of chills, while in others there was only one attack, appearing rather late in the course of the illness.

Only about a fifth of the cases actually complained of dyspnea or of chest oppression, and in nine cases only later in the course of the disease.

Other early symptoms observed were epigastic or generalized abdominal pain, tympanism, general body aches, joint, bone, or muscle pains, nausea and vomiting, anorexia, impairment of sleep, epistaxis, and profuse perspiration. Six patients had jaundice, ranging from a faint icteric tinge of the sclera to considerable yellowing of the skin. Bowel disturbances were present in some, in the form of frequent bowel movements, while in others there was constipation. Urine tended to be highly colored. There was actual polyuria in two cases. Coryza was complained of in three instances, while dizziness was one of the most disturbing symptoms of two cases. One patient had maculo-papular eruptions early in the disease. Restlessness, semiconsciousness, psychosis, hoarseness, slurring of speech, and laryngitis were among the rarer symptoms. Hebetude and body weakness were more common in the later stages. (Table 2).

The pulse rate in our cases increased more or less in proportion to the rise of temperature, a rate of 110 to 120 per minute being frequent, and rates as high as 150 having been observed. Bradycardia was not noticed.

The respiratory rate was only slightly increased, rates exceeding thirty per minute being rather infrequent.

Herpes labiales has not been observed.

Physical Findings: Physical findings initially were often confusing.(35) A patient may be bright and relatively comfortable only to show abundant physical findings, and another may look acutely ill and reveal little on physical examination.

The face was flushed in a few cases. Conjunctival injection was marked in about ten per cent of cases. In those cases where there was apparently either a toxic hepatitis or a concomittant hepatic involvement, scleral icterus was noted. Dilatation of the alae nasae on inspiration, as Campbell emphasizes, was conspicuous by its infrequency.(12) A dirty, furred tongue was rather frequent.

Slight rigidity of the neck was observed in one case.

Examination of the heart was essentially negative. In two cases, however, there was a functional murmur in the mitral area and in one case there was an apparent slight increase in the area of cardiac dullness. Accentuation of the second pulmonic sound was not infrequently observed.

The lung findings are very interesting. It is characterized by the great disparity between the complaints, the physical findings and the roentgenographic picture. In only 10 patients was there an appreciable limitation of the expansion of the affected side. In eighty-eight cases, the lesion was definitely patchy in nature, appearing apparently as if only a portion, or portions, of the lobe was affected. Only impairment of resonance was appreciated in seventy-one cases, and dullness appeared only in twenty-one cases, while in the remaining nine, there were no percusory findings. Muscular hyper-irritability was found in two cases. Frequently, tactile fremitus was only slightly increased, in some it was decreased, and in a number there was no appreciable change.

Decreased breath sounds were rather common and were apparently at some time or another the only finding in some cases. Harsh breath sounds were appreciated in some cases and were absent in others. Bronchial breathing, brochophony, and whispered pectoriloquy were elicited in only a few instances. Rales were, as a rule, scanty, and may be crepitant, subcrepitant, sonorous, or sibilant. (Table II).

Any lobe of either or both lungs may be affected but basal lesions are most common—seen in eighty-six patients in our series, of which left-sided lesions were found in thirty-two cases, thirty-six cases with right-sided affection, and bilateral basal lesions in eighteen (Table IV).

Abdominal tenderness at one region or another was almost invariably elicited in those cases where abdominal pain was a symptom. In three cases, there was even some degree of rigidity.

Splenomegaly was appreciated in four cases but in all of these there was a very strong history of malarial infestation at one time

or another. Slight enlargement of the liver was observed in three cases without any attendant splenomegaly nor any history of malaria, and in one case where there was splenomegaly and malarial history.

Laboratory Examinations: There seems to be a tendency to slight anemia, over fifty per cent of our cases having counts of three to four million red blood cells per cu. mm. About twenty per cent had more or less normal counts. The hemoglobin content was on the average seventy to seventy-five per cent.

In the early part of the disease, half of our cases had counts ranging from 12,000 to 18,000. The greater bulk of the remainder had higher values, the highest count in 2 cases being over 35,000. There were, however, counts in the early stage below 7,000. There was a polynucleosis of from 76 to 95 per cent. Inadequate staining facilities, however, prevented us from ascertaining how much of this figure is made up of eosinophils, which may constitute a considerable percentage.(36) In the later part of the disease, there was observed, as a rule, a distinct and many times abrupt fall of the count, about two-thirds of the examined patients giving a count of from 7,000 to 13,000, of which seventy-one to eighty-five per cent were polymorphonuclears. The lowest count observed in the later stages was 4,200 and the highest 20,000. One case gave a count of 23,000, and another 35,000.

Urinalysis was essentially negative. In a few cases, there were traces of albumin, occasional hyaline casts, rare to few red blood cells, rare to few pus cells; in two cases there were abundant pus cells. Urinary findings were usually present during the highly febrile period, and disappeared soon after the drop of the temperature.

In those with suspected or manifest jaundice, elevation of serum bilirubin values was observed, Bilirubin I going as high as 0.641 mgm./cc. and Bilirubin II as high as 4.983. These values gradually went down to normal, pari passu with improvement of the case.

In all those cases where sputum examination was done, pneumococci were not identified. The mere presence of pneumococci in the sputum, however, does not necessarily rule out atypical pneumonia. There are reports of the isolation of the pneumococci in the sputum. These are not considered the causative organism since they were of the higher types.(12)

Roentgenological Findings: Inadequacy of supplies did not allow us to have the desired X-ray studies. Nonetheless, the examinations performed yielded very interesting and gratifying results.

The findings were very variable. We had cases where there was diminished transradiancy or diminished aeration of the affected side. This may appear more or less homogeneously over the whole lobe or field or may be in patches. Other findings were marked diffuse perihilar shadows; prominence of lung markings; cottony shadows which may appear like bronchopneumonic patches or congestion. A mottled appearance may also be observed. The shadow may also resemble lobar pneumonia but, as Green and Eldridge note, without obscuring the vascular and osseous markings.(44)

Complications: Complications of primary atypical pneumonia are apparently uncommon.

In our series we had a few. In six cases with scleral icterus and increased blood bilirubin values, there must have been at least a toxic hepatitis. Whether this is due, or not, to a primary hepatic involvement by the same etiologic agent, we are not in a position to determine. Suffice it to mention that in these cases the jaundice and the serum bilirubin values diminished and returned to normal as the patients improved.

Diaphragmatic pleuritis was observed in eight cases with an audible rub and referred abdominal pain. Meningismus was seen in two cases with negative spinal fluid findings. There was toxic psychosis in one patient who had complete abatement of symptoms with the recovery from the respiratory condition.

Evidence of pleural fluid was detected on the twelfth day in one case which on tapping yielded a thin sero-sanguinous exudate with four per cent albumin, 6,250 cells per cu. mm., with 66 lymphocytes, 21 polynuclears, 1 eosinophil, 6 macrophages, and 6 mesothelial cells. It was negative for any micro-organisms. This patient had an uneventful recovery and did not require a second tapping.

Prognosis: In the absence of any serious complication, concurrent or superimposed, prognosis is generally good. Reports of deaths, however, may be met in literature. The mortality rate in an Army camp with 1,862 cases was reported at 0.26 per cent(35) while among civilians, the rate is estimated at 2.4 per cent.(31)

Of the one hundred and one cases in the series, seventy were discharged "recovered," symptom-free and clear of any physical findings; twenty-nine were discharged "improved"—patients who were afebrile for sometime, completely symptom-free and with a normal blood picture, but still exhibiting some pulmonary physical findings, which may be in the form of persistent impairment of resonance,

bronchial breath sounds, or some moist rales; and two discharged against advice, "unimproved"—still running a temperature and with signs and symptoms.

The period of confinement ranged from three to forty days, the greater number staying in the wards for from one to two weeks.

Summary: The extremes and variations in the clinical and laboratory data of one hundred and one cases of primary atypical pneumonia were presented and compared with those reported in the literature.

REFERENCES

(1) ALLEN, W. H., "Acute Pneumonitis," Annals of Int. Med.: 10: 441, 1936.

(2) REIMANN, HOBART A., "An Acute Infection of the Respiratory Tract with Atypical Pneumonia," J.A.M.A., 111: 2377 (Dec. 24) 1938.

(3) SISON, A. B. M., JIMENEZ, V. B., and HERRERA, F., JR., "Virus Pneumonia," submitted for publication in the Jour. P.I.M.A. in Oct., 1942.

(4) IDSTROM, L. G. and ROSENBERG, B., "Primary Atypical Pneumonia," Bull. U. S. Army Med. Dept., No. 81 (Oct.) 1944.

(5) Commission on Acute Respiratory Diseases, "Atypical Pneumonia." Am. Jour. Med. Sciences, 209; 55 (Jan.) 1945.

(6) SMITH, RICHARD H., "Primary Atypical Pneumonia, Etiology Unknown," The Average Clinical Picture Based on Thirty-seven Original Cases," Annals Int. Med., 20: 890 (June) 1944.

(7) DINGLE, J. H. and FINLAND, M., "Primary Atypical Pneumonia of Unknown Etiology," New England. Jour. Med., 227: 378-385 (Sept. 3) 1942 Quoted by Smith, R. H. (5).

(8) LONGCOPE, W. T., "Bronchopneumonia of Unknown Etiology (Variety X)," A Report of Thirty-two Cases with Two Deaths," Bull. Johns Hopkins Hosp., 67: 268-305 (Oct.) 1940. Quoted by Schmitz (13).

(9) Official Statements, "Primary Atypical Pneumonia, Etiology Unknown," War Medicine, 2: 330-333 (1942).

(10) SMILE, D. R., SHOWACRE, E. E., LEE, W. F., and FERRIS, H. W., "Acute Interstitial Pneumonitis, A New Disease Entity," J.A.M.A., 112: 1901-1904 (May 13) 1939.

(11) CAMPBELL, T. A., STRONG, P. S., GRIER III, G. S., and LUTZ, R. J., "Primary Atypical Pneumonia; A Report of Two Hundred Cases at Fort Eustis, Virginia, J.A.M.A., 122: 723 (July 10) 1943.

(12) SCHMITZ, R. C., "Primary Atypical Pneumonia of Unknown Cause," Arch. Int. Med., 75: 222 (April) 1945.

(13) PAGE, S. G., JR., and TITLE, C. R., "Primary Atypical Pneumonia," Virginia Medical Monthly, 71: 305 (June) 1944.

(14) LEVINSON, D. C., GIBBS, J., and BEARDWOOD, J. T., JR., "Ornithosis as a Cause of Sporadic Atypical Pneumonia," J.A.M.A., 126: 1079 (Dec. 23) 1944.

(15) PINKERTON, H. and HENDERSON, R. G., "Adult Toxoplasmosis," J.A.M.A., 116: 807 (Mar. 1) 1941.

(16) GOLDSTEIN, D. and MCDONALD, J. B., "Primary Pulmonary Coccidioidomycosis," J.A.M.A., 124: 557 (Feb. 26) 1944.

(17) KELLER, A. E., "The Clinical Manifestations, Treatment and Prevention of Ascaris," J. Tennessee M. A., 25: 346 (Sept.) 1932. Quoted by Schmitz(13)

(18) Commission on Acute Respiratory Diseases, Army Epidemiological Board, "Transmission of Primary Atypical Pneumonia to Human Volumeers": J.A.M.A., 127: 146 (Jan. 20) 1945.

(19) Commission on Acute Respiratory Disease, Army Epidemiological Board, "Present Status of Etiology of Primary Atypical Pneumonia," Bull. U. S. Army Med. Dept., 4: 494 (November) 1945.

(20) Editorial, "The Virus of the So-called 'Virus Pneumonias'," New Eng., J. of Med., 228: 729 (June 3) 1943.

(21) Editorial, "'Acute Pneumonitis' Virus," J.A.M.A., 116: 1222 (Mar. 22) 1941.

(22) Editorial, "Primary Atypical Pneumonia," J.A.M.A., 126: 18 (Dec. 30) 1944.

(23) KNEELAND, Y., JR., and SMETANA, H. F., "Current Bronchopneumonia of Unusual Character and Undetermined Etiology," Bull. Johns Hopkins Hosp., 67: 229 (Oct.) 1940. Quoted by Schmitz(13).

(24) ADAMS, J. M., "Primary Virus Pneumonitis with Cytoplasmic Inclusion Bodies," J.A.M.A., 116: 925 (Mar. 8) 1941.

(25) GOLDEN, A., "Pathology of Atypical Pneumonia," Bull. U. S. Army Med. Dept., 81: 64 (Oct.) 1944.

(26) DINGLE, J. H., ABERNETHY, T. J., BADGER, G. F., BUDDINGH, G. J., FELLER, A. E., LANGMUIR, A. D., RUEGSEGGER, J. M., and WOOD, W. B., JR., "Primary Atypical Pneumonia, Etiology Unknown," Am. Jour. Hygiene, 39: 67 (Jan.) 1944. Quoted by Schmitz(13).

(27) LONGCOPE, W. T., "Atypical Virus Pneumonia," Bull. New Eng. M. Center, 4: 21 (Feb.) 1942. Quoted by Schmitz(13).

(28) THOMAS, H. M., JR., "The Role of Alpha Hemolytic Streptococcus in Pneumonia," Bull. Johns Hopkins Hospital, 72: 218 (April) 1943. Quoted by Schmitz(13).

(29) SABIN, A. B., "Toxoplasmic Encephalitis in Children," J.A.M.A., 116: 801 (Mar.) 1941.

(30) OWEN, C. A., "Primary Atypical Pneumonia: An Analysis of Seven Hundred and Thirty-eight Cases Occurring During 1942 at Scott Field, Ill.," Arch. Int. Med., 73: 217 (Mar.) 1944.

(31) KAMIN, H. N., "Virus Pneumonia," Illinois, Med. Jour., 83: 41 (Jan.) 1943.

(32) GOODRICH, B. E., and BRADFORD, H. A., "The Recognition of Virus Type Pneumonia," Am. J. M. Sc., 203: 163 (Aug.) 1942.

(33) a. DINGLE, J. H., ABERNETHY, T. J., BADGER, G. F., BUDDINGH, G. G., FELLER, A. E., LANGMUIR, A. D., RUESEGGER, J. M., and WOOD, W. B., "Primary Atypical Pneumonia, Etiology Unknown," War Medicine, 3: 223 (Mar.) 1943.

b. CAMPBELL, E. T., "Primary Atypical Pneumonia and Malaria," War Medicine, 3: 249 (Mar.) 1943.

(34) VAN RAVENSWAAY, A. C., ERICKSON, G. C., ROH, E. P., SIEKERSKI, J. M., POTTASH, R. R., and GUMBINER, B., "Clinical Aspects of Primary Atypical Pneumonia (A Study Based on 1,862 Cases Seen at Station Hospital, Jefferson Barracks, Missouri," J.A.M.A., 124: 1 (Jan. 1) 1944.

(35) CONTRATTO, A. W., "So-called 'Atypical Pneumonia' Among College Students," New Eng.J. Med., 229: 229 (Aug. 5) 1943.

(36) GUNDERSEN, S., "Primary Atypical Pneumonia of Unknown Etiology," New Eng., J. Med., 231: 697 (Nov. 23) 1944.

(37) HELWIG, F. C. and FREIS, E. D., "Cold Autohemagglutinins Following Atypical Pneumonia Producing the Clinical Picture of Acrocyanosis," J.A.M.A., 123: 626 (Nov. 6) 1943.

(38) EMERSON, K., JR., ET AL., "Chloride Metabolism and Plasma Amino Acid Levels in Primary Atypical Pneumonia," J. Clin. Investigation, 22: 695 (Sept.) 1943. Quoted by Schmitz(13).

(39) RAKE, G., EATON, M. D., and SHAFFER, M. F., "Similarities and Possible Relationships Among Viruses of Psittacosis, Meningipneumonia, and Lymphogramuloma Venereum," Proc. Soc. Exper. Biol. and Med., 48: 528 (Nov.) 1941. Quoted by Schmitz(13).

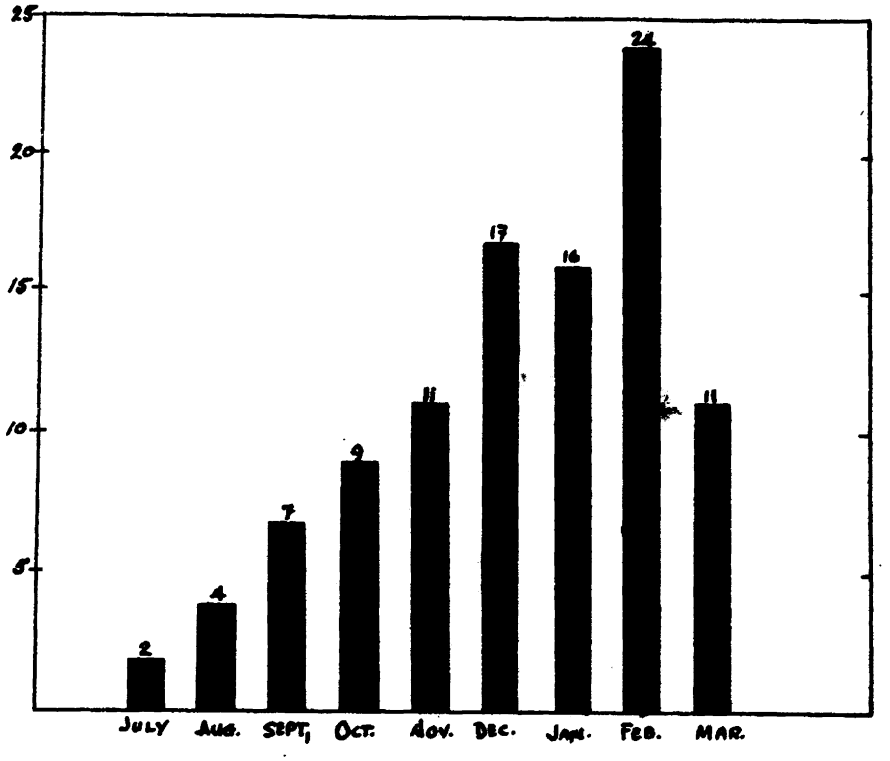
(40) GALLAGHER, J. R., "Bronchopneumonia in Adolescence," Yale J. Biol. and Med., 7: 23 (Oct.) 1934. Quoted by Schmitz(13).

(41) SMITH, J. H. "Virus Pneumonia by Contrast with Other Types," Virginia Medical Monthly, 70: 353 (July) 1943.

(42) OFFUTT V. D., "Diagnosis and Treatment of Primary Atypical Pneumonia," Virginia Medical Monthly, 71: 431 (Aug.) 1944.

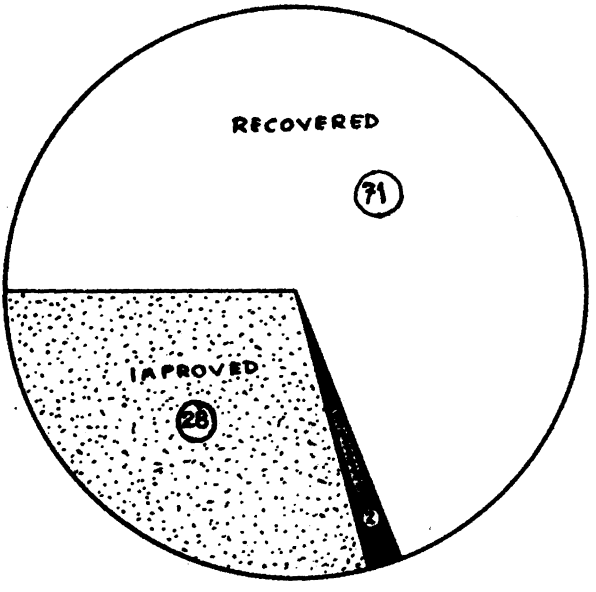
(43) GREEN, D. M. and ELDRIDGE, F. G., "Primary Atypical Pneumonia, Etiology Unknown," Military Surgeon, 91: 503 (Nov.) 1942.

(44) RACKER, E., ROSE, S. P., and TUMEN, A. O., "Pneumococcic Pneumonia Resembling Primary Atypical Pneumonia," Am. J. M. Sc., 209: 496 (Apr.) 1945.



Monthly Distribution of the 101 Cases of the Series

N.B. There were no figures obtained for April, May, and June



Condition on Discharge

TABLE I.—*Symptoms*

	First 3 days	*Later*
Fever	88	8
Cough	77	10
Chest pain	57	7
Mucoid expectoration	54	5
Back pain	44	5
Headache	40	3
Chilly sensations	31	
Chills	23	5
Dyspnea	21	9
Abdominal pain	11	4
Vomiting	10	4
Muscle pains	6	
Icteric sclerae	6	1
Bone pains	6	1
Anorexia	6	4
Rusty sputum	5	5
Blood streaks in sputum	4	2
Nausea	4	1
Coryza	3	
Epistaxis	3	
Dizziness	2	
Impaired Sleep	2	1
Body weakness	2	6
Chest oppression	2	
Epigastric pain	2	1
Frequent urination	1	1
Highly colored urine	1	2
Incoherence	1	1
Insomnia	1	1
Skin eruptions	1	
Psychosis	1	
Profuse perspiration	1	1
Semiconsciousness	1	1
Tympanism	1	
Restlessness	1	
Laryngitis	1	

TABLE II.—*Physical Examination*

Nutrition:	
Well nourished	6
Fairly nourished	75
Poorly nourished	21
Emaciated	0
Head:	
Flushed face	2
Dilatation of alae nasae	8
Coated tongue	32
Injected conjunctivae	8
Icteric sclerae	10
Neck:	
Slight rigidity	1
Chest:	
Heart:	
Murmur, soft blowing	2
Enlarged, 6th I. S.	1
Accentuated 2nd pulmonic sound	21
Lungs:	
Inspection:	
Limited mobility of affected part	10
Percussion:	
Muscular hyper-irritability	2
Dullness	21
Impaired resonance	71
Palpation:	
Tactile fremitus increased	69
Tactile fremitus normal	11
Tactile fremitus decreased	21
Auscultation:	
Breath sounds weak	68
Breath sounds absent	3
Breath sounds harsh	5
Bronchophony	4
Bronchial breath sounds	10
Whispered pectoriloquy	0
Rales:	
Crepitant, abundant	15
Crepitant, few	60
Subcrepitant, few	33
Subcrepitant, abundant	10
Sonorous	5
Sibilant	9
Rub	5
Abdomen:	
Splenomegaly	4
Hepatomegaly	4
Rigidity	3
Tenderness:	
Right hypochondrium, slight	4
Left hypochondrium	2
Right iliac	1
Left iliac	1
Epigastrium	1

TABLE III.—*Character of Fever*

	No. of cases
With slight rise after falling to normal	18
Continuous:	
High	22
Moderately high	20
Remittent:	
High	12
Moderately high	17
Intermittent:	
High	6
Moderately high	10
Low	2
Dromedary	7

Completely afebrile	5
With slight rise after falling to normal	18

TABLE IV.—*Lung Involvement*

	Patchy	*"Lobar"*
Right:		
Upper lobe	3	1
Middle lobe	5	0
Base	28	4
Upper, middle	0	0
Middle, base	4	2
Left:		
Upper lobe	2	0
Base	26	4
Both lobes	2	0
Bilateral:		
Upper lobes	1	0
Bases	17	1
Mixed	1	0
TOTAL	89	12

TABLE V.—*Differential Criteria*

	Pneumococcic	*Atypical*
Onset	Abrupt	Slow
Cyanosis and Dyspnea	Frequent	Rare
Herpes	Frequent	Rare
Pulse rate	Rapid	Normal or slightly accelerated
Respiratory rate	Accelerated	Normal
Physical signs	Impaired resonance	Very slight change in resonance
	Bronchial breathing	Rare bronchial breathing frequent rales
Sputum gross	Rusty	Greenish mucoid
Sputum microscopic and culture	Pneumococcic	No predominating organism
White blood cell count	High	Normal
X-ray finding	Dense consolidation	Stringy and mottled type density
Crisis:	Frequent	Rare
Response to Sulfonamides	Good	None

CAMPBELL ET AL; J.A.M.A. 122: 723; July 20, 1943.