Case.	Age.	Red Cells.	Haemoglobin.	Colour Index.	Remarks.
			Per cent.		
1	31	6,600,000	97.5	0.74	Severe D.A.H.
2	36	6,465,000	99	0.76	Severe D.A.H.
3	26	6,275,000	99	0.79	Mild D.A.H.
4	24	6,135,000	93	0.76	Moderate D.A.H.
5	24	6,065,000	100	0.82	Severe D.A.H.
6	—	5,960,000	89.5	0.75	Chronic mild D.A.H.
7	27	5,915,000	91	0.76	Chronic severe D.A.H.
8	19	5,900,000	97	0.82	Severe D.A.H.
9	18	5,867,000	90	0.76	Chronic moderate D.A.H.
10	32	5,675,000	91	0.84	Severe D.A.H.
11	31	5,552,000	—	—	Moderate D.A.H.
12	25	5,526,000	92.5	0.83	Mild D.A.H.
13	27	5,495,000	89.5	0.81	Mild D.A.H.
14	34	5,110,000	92	0.9	Chronic mild D.A.H.
15	32	4,892,000	87	0.89	Severe D.A.H.

The average of the fifteen patients suffering from D.A.H. was 5,837,000, which is quite in excess of anything previously observed in healthy individuals, and is the more remarkable in view of the fact that these men were invalids, who had for the greater part spent a considerable time in hospital before the estimation was made. The average age of these patients was 28 years. One-third of these patients had a count of over 5,000,000, and more than one half (53 per cent.) had a count of 5,900,000 or over.

The haemoglobin content was for the most part below the normal 100 per cent., the average being 93.4 per cent. (ranging from 87 per cent. to 100 per cent.). Relatively to the number of red cells the haemoglobin content was low, the average colour index being 0.8.

The cases have been roughly described in the table according as they are severe or mild, chronic, or otherwise, and it is evident that no relationship can be established between these particulars and the red cell count. It is apparent that a high blood count may be found in a patient, such as Case 6, able to undertake a half-hour's physical drill and march three miles without pronounced discomfort, and that a low count may occur in a patient, such as Case 15, readily distressed and only able to undergo mild resistance exercises.

The leucocytes were not counted in this series of cases. The existence of a leucocytosis in cases of D.A.H. has been established and described by Mrs. Briscoe.³

Multiple observations made at intervals on other individuals indicate a degree of variability in the count, but this is a matter that requires further investigation; amplification of these observations is evidently desirable in other respects, but, my work having been interrupted, I publish these records as they stand.

It does not appear possible in the present state of our knowledge of the pathology of D.A.H. to suggest the significance of this polyerythrocythaemic condition.

REFERENCES.

¹ *Eines Klinische Pathologie des Blutes*, Jena, 1893. ² *Amer. Journ. Physiol.*, 1904, vol. x, p. 384. ³ Report on Cases of Soldier's Heart, Medical Research Committee, 1917.

THE first meeting of the Spanish National Medical Congress will be held at Madrid in April, 1918 (21st to 26th). The work will be divided among seventeen sections. The president is Professor Ramón y Cajal, well known as the author of works of the highest importance on the structure of the brain and spinal cord. In the summer of the present year he published an autobiography under the title of *Recuerdos de mi Vida* (Recollections of My Life). Santiago Ramón y Cajal was born in 1852, and studied at Saragossa, where his father was professor of anatomy. After graduating in 1873, he served as an army medical officer in Cuba. In 1881 he was appointed Professor of Anatomy at Valencia; in 1886 he became Professor of Histology at Barcelona; and in 1892 he was called to the corresponding chair at Madrid.

ACUTE TUBERCULOUS BRONCHOPNEUMONIA WITH PNEUMOTHORAX, SECONDARY TO TUBERCULOSIS OF THE PERI- BRONCHIAL LYMPH NODES.

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THE following case is reported because it illustrates the relation of certain more chronic tuberculous lesions to acute tuberculous pneumonia, and the relation of the latter to pneumothorax.

Pte. J. G., aged 18, was admitted at midnight on January 26th, 1917, with a diagnosis on his field medical card of "incipient trench feet."

Previous History.—He had been a farm worker. During his last few days at the front he had had a cough and pain in the right chest; in the ambulance train this pain was quite sharp.

Clinical Aspect.—The feet were slightly swollen and tender but nowhere was the skin broken. The temperature was normal on admission, and the pulse was 92; the respirations were not rapid, and he made no complaints referable to his chest. He was able to sit up in bed and wash himself, appeared perfectly comfortable, and soon dropped off to sleep. At 4 a.m. (January 27th) he awoke suddenly with very severe pain in the right side of the chest and upper abdomen. He was extremely dyspnoeic and cyanosed. The respirations were 40, the movements of the chest were very shallow, the pulse 132, and the temperature 102.6°; it was 105° a few hours later.

He was almost immediately transferred to the "pneumonia hut," where there is an abundance of fresh air. The apex beat of the heart could be seen in the fifth interspace 14.5 cm. to the left of the middle line, and on percussion the right border was found 2.5 cm. to the left of the left border of the sternum. The heart sounds were clear and no murmurs were heard. The right side of the chest did not move. Tactile fremitus was much decreased over the right lower axilla and base, and at the base behind there was an area of flatness extending upwards for about five inches. Above this in front the percussion note was hyper-resonant. Over the areas of the middle and lower lobes the breath sounds were amphoric, and accompanied by tinkling sounds; over the upper lobe in front the respiratory murmur could scarcely be made out. The amphoric breathing was transmitted to the left of the sternum, and was marked at the level of the second costal cartilage. On the right side, behind, the breath sounds were poorly heard. The *bruit d'airain* was heard over the right chest, and the succussion splash was readily made out. On the left side there was no dullness, but the respiratory murmur was harsh, although nowhere "blowing."

A diagnosis of right-sided pneumothorax was now made, and this was thought to have been caused by the rupture of a tuberculous cavity in the lung. Pneumonia also was thought to be present. A very grave view of the case was taken, but the patient held out until February 2nd. The course of the disease was not without interest.

January 27th: A small amount of fluid was withdrawn from the right base. January 28th: The temperature was 104°, respirations 32, pulse 118. Blood was seen in the sputum. January 29th: Temperature 99.5°, respirations 32, pulse 120. The amphoric breathing was not heard so high up on the right side of the chest, but above this there were numerous rhonchi. No change in the position of the heart was made out, so the pneumothorax was thought not to be "valvular" in nature. January 30th: Temperature 99°, respirations 30, pulse 124. The patient became slightly delirious and was much cyanosed. The upper level of flatness in the right thorax had apparently not changed. The amphoric breathing was still distant and heard on the right side only over a small area close to the lower end of the sternum. Over the right upper lobe the respiratory murmur was inaudible. February 1st: Temperature 97°, respirations 36, pulse 100. There was no blood in the sputum. The patient was not so cyanosed and his pulse was stronger. The apex beat was seen at a point 10.5 cm. from the middle line—that is, a little nearer its normal position. The amphoric breathing at the pulmonary cartilage was not so pronounced. Although there was still pain in the right chest, it was thought that the pneumothorax was decreasing. Over the left upper lobe the breath sounds were still harsh, and numerous moist râles were heard. February 2nd: The patient was again very cyanosed, with much wheezing, and was unconscious most of the time. No blood in the sputum. The apex beat was in the same position as on the previous day. The amphoric breathing was again heard higher up on the right side of the chest and at the base of the heart to the left of the sternum. Still no increase in the area of flatness was observed. Sharp moist râles were heard over the left upper lobe, with prolonged expiration. Numerous bubbling râles were heard in the left axilla. It was thought that the patient had tuberculosis of both lungs. Temperature subnormal, respirations 40 to 48, pulse 120. He died at midnight of cardiac failure.

I am indebted to Major Lawrence J. Rhea, C.A.M.C.,

for the following notes on the pathological aspect of the case:

Pathological Report.—Report on the fluid withdrawn from the right pleural cavity on January 27th: "There is about 10 c.cm. of turbid yellow fluid. The predominating cells are polymorphonuclear leucocytes, although there are a considerable number of endothelial cells and lymphocytes. Smears stained with carbol-fuchsin show numerous tubercle bacilli. These organisms are mostly extracellular in position, although some of them have been ingested by the endothelial leucocytes. There are also a few small diplococoid organisms." Report upon the examination of the sputum of January 28th and 30th: "A number of selected particles from the sputum were stained for tubercle bacilli, and after careful and prolonged search only two were found."

Post-mortem Examination.—The skin and pectoral muscles were dissected in such a way as to make a water-tight pouch bounded by the ribs and the dissected tissues. This pouch was now filled with water and a puncture made beneath the water between the second and third ribs into the right side of the thorax. Air, under slight pressure, bubbled through the water. The right thoracic cavity contained a large amount of turbid fluid. The right lung was greatly compressed and forced upwards and inwards against the bodies of the dorsal vertebrae. Over the parietal pleura, especially at the angles of the ribs and over the upper surface of the diaphragm on the right side, there was a layer of plastic lymph several millimetres in thickness, which was easily removed. The visceral layer of the pleura also showed a lymph exudate which was thickest over the posterior surface of the upper lobe. The mediastinum and its contents were pushed to the left. The cause of the pneumothorax—that is, the communication between the respiratory passages and the pleural cavity—was not at first evident, but after removing the lymph exudate from the lower part of the posterior surface of the right upper lobe a small perforation about 2 mm. in diameter was found in the visceral layer of the pleura. The right upper lobe was crepitant in its upper but not in its lower half. A longitudinal section was made through the lower part of the upper lobe and showed the following: There is a band, about 5 cm. in width, of acute tuberculous bronchopneumonia extending from a point close to the hilum of the lung to the periphery of the lower part of the upper lobe. This band follows the general direction of one of the branches of the bronchial tree. For the most part the small greyish-white areas of tuberculosis vary in size from about 0.5 mm. to 10 mm. in diameter. One small area of tuberculosis, just beneath the visceral layer of the pleura, has broken down, and by involvement of the overlying pleura has caused the tiny perforation already described. This sharply limited area of involvement of the lung is explained as follows: The lymph nodes about the hilum of both lungs are enlarged, and are the seat of old non-active tuberculosis. There is, however, one node on the right side which shows changes due to active disease. This node, which is firmly attached to one of the bronchi, has undergone acute necrosis and has led to the perforation of the bronchus. As a result, there is established an opening from the medulla of a tuberculous lymph node directly into a bronchus. The area of diseased lung corresponds to that supplied by the perforated bronchus. There is no gross evidence of old tuberculosis in either lung. The left lung shows septic bronchopneumonia. The right side of the heart is dilated.

Comments.

1. This case demonstrates a well-known fact—namely, that the finding of pneumothorax clinically, and of numerous tubercle bacilli in the pleural effusion, does not compel us to diagnose extensive disease of the lung.
2. The amount of fluid in the pleural cavity was underestimated.
3. The changes in the position of the heart and the changes in the clinical signs, especially that of the amphoric breathing, may, we think, be explained. The return of the apex of the heart towards the middle line, and the decrease in the area of amphoric breathing, were evidently due to the fact that the air in the chest was being absorbed after the perforation of the lung had been closed by lymph. The fact that the amphoric breathing was again heard higher up on the chest, may possibly be explained by the greater compression exerted upon the residual air in the pleural cavity by the increasing amount of fluid.
4. The source of the infection of the lung was found.
5. The area of lung involved corresponded to the distribution of that bronchus which had been perforated by an ulcerating tuberculous lymph node.
6. The production of the pneumothorax was due to a factor which plays such an important rôle in many diseases, namely, the accident of position of one of the small foci of acute tuberculous bronchopneumonia.

To Dr. George E. De Schweinitz, now serving as a major in the United States Medical Reserve Corps, has been assigned the task of compiling a handbook on ophthalmology for the use of the Surgeon-General of the army.

NOTES ON TRENCH FOOT.

BY

CAPTAIN R. T. DOBSON, R.A.M.C.

UNDER the exigencies of modern campaigning winter warfare imposes on the troops holding the line conditions to which, previous to the winter of 1914, no armies had been subjected, and to counteract which no measures had been devised, with the consequence that in that winter an enormous proportion of the troops were incapacitated by the various lesions to which as a whole the term "trench foot" is applied.

As the conditions favouring the occurrence of these became better understood, and measures were devised to combat them, the losses from the occurrences of trench foot became less serious in each succeeding winter.

Whilst in charge of the arrangements for preventing and treating lesions of the feet and legs occurring in the troops occupying a certain area, all the cases of so-called trench foot passed through my hands and came under my care; this gave me exceptional opportunities of studying the various kinds of lesions included under the term.

There were three distinct types. The first, which comprised about 15 to 16 per cent. of the cases, commenced with symptoms of marked constriction of the blood vessels, causing the feet to become in the first stage numb, cold to the touch, and strikingly bloodless in appearance. The second type, comprising 4 to 5 per cent. of the cases, commenced as ordinary chilblains. The third type, which comprised the remainder of the cases, arose as septically infected lesions of the skin, and were obviously due to neglect of ordinary cleanliness or ill-fitting foot-gear, or both. The first two types are to a certain extent unpreventable, but the third should be quite avoidable with ordinary care.

The period during which the observations were made extended from the beginning of November, 1916, to the later part of February, 1917. The first two-thirds of this period was very wet with low temperature, whilst the latter part was dry and frosty with a temperature much lower than normal, a hard frost prevailing most of the time.

During the rainy period the trenches and ground were very wet, necessitating the almost continuous wearing of thigh gum boots. In the dry frosty weather the men were able to revert to the use of their leather ankle boots and putties.

The greatest influx of cases occurred towards the end of the wet period, although some of the more severe cases occurred during the dry frosty weather.

The cases of the first type mentioned below, those occurring during the wet period, resembled closely the milder cases of frostbite, under which term the cases occurring during the dry frosty weather should properly be included.

Four per cent. of the cases gave a history of having suffered from a mild form of Raynaud's disease; 50 per cent. gave a history of having been troubled by persistent cold extremities, blueness of the hands or other signs of poor capillary circulation. About 30 per cent. of these were also subject to chilblains, whilst about 10 per cent. gave a history of having suffered from chilblains only.

The wearing of gum boots did not appear to prevent entirely the onset of the complaint, since 80 per cent. of the total cases included under the term trench foot and occurring during the wet period were wearing them. In fact, where they were worn continuously during the tour in the trenches, as was the practice with most of the men, they soon became wet inside from condensed perspiration, and the clothing covered by them became soaked by perspiration also, thus subjecting the legs and feet to the very conditions the boots were devised to avoid. These conditions were aggravated by any active work which caused them to perspire undertaken by the men whilst wearing gum boots. On the other hand, the lack of exercise to which the exigencies of trench warfare subjects the men whilst in the front line also has a harmful effect on the circulation, in the lower limbs especially.

The age of the men did not appear to have much influence on the onset or course of the complaint, but the length and thoroughness of their physical training did, as it more easily affected those who had come from sedentary