larger cells resembled myeloblasts, and were difficult to place accurately in any group. Films stained by Leishman's method showed a dark basophil cytoplasm in certain cells with numerous azurophil granules. Some cells had an intensely basophil-stained cytoplasm. The nuclei were large, varying in shape. Some of the cells showed mitotic division with beautiful examples of monaster and diaster stages.

On November 11th numerous petechial haemorrhages were noted over the chest, and especially round the hair follicles. On November 14th the patient died, twenty-one days after the pyrexial attacks commenced and six days after his spleen was found to be enlarged. The lymph-gland groups enlarged coincidentally with the spleen, particularly in the cervical region.

[Slides of the blood, spleen, bone marrow, and liver were demonstrated.]

Case of Aplastic Anaemia.

J. W., a female aged 26, a cook, was admitted to Dr. Fleming's wards on the recommendation of Dr. John Craig, Edinburgh, on November 11th, 1920. The duration of the illness was from December, 1918, and her chief complaints were weakness and shortness of breath.

She had had no previous illness before 1918, except "headaches." Since 1918 she had a "blister-like" rash on

her face, shoulders, and chest, in 1919 lasting for three weeks. There was also severe pyorrhoea, which gave continuous trouble and required much treatment. Her mother and her only sister were anaemic, and both had a history of gastric ulcer. Her father died of pulmonary tuberculosis at the age of 35.

On examination the patient was stout and very pale, but with a lemon-tinted skin. She had the usual haemic murmurs and a slightly dilated heart. There were periods of pyrexia lasting for ten days at a time with no definite periodicity. The diagnosis of aplastic anaemia was made because, during the patient's stay in the wards, extending up to the time of her death on October 19th, 1921 (a period of eleven months), her blood never showed any normoblasts or megaloblasts. There was poikilocytosis, varying in degree from time to time, but no punctate basophilia and no polychromatophilia. Anisocytosis was peculiarly marked, there being many microcytes and megalocytes.

Direct transfusion proved the only really effective treatment, although the benefit obtained was temporary.

The post-mortem examination showed that an extraordinary degree of haemolysis had occurred.

[Slides of the blood, the bone marrow, etc., were demonstrated.]

A CASE OF INTRAPERICARDIAL ANEURYSM OF THE AORTA.

BY

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The patient, a labourer aged 48, attended hospital on August 10th, 1920, complaining of pain in the chest, on the right side especially, shortness of breath, and cough during the previous four weeks.

Physical Examination.

The findings at the physical examination were as follows:

A well-developed man, somewhat above the average in general physique; expression anxious, cheeks sunken, malar eminences flushed; obvious pulsation in carotid, temporal, brachial, and other superficial arteries. Chest wall well covered, normal contour except for visible pulsation to the right of the sternum in the third, fourth, and fifth spaces; area of cardiac dullness extends 4 inches to the left of the mid-sternal line to the apex beat, which is situated in the sixth intercostal space and $3\frac{1}{2}$ inches to the right of the mid-line at the level of the fifth rib and corresponding space. The apex beat is forcible in character, diffuse in extent; there is a heaving impulse to the right of the sternum of maximum intensity in the fifth, but also felt in the third and fourth spaces. On auscultation a short systolic and long diastolic murmurs are audible over the entire precordium, the latter being of maximum intensity at the aortic area, but clearly heard over the dull region to the right of the sternum, and conducted upwards into the vessels at the root of the neck. The arteries are thickened and tortuous, the radial pulses collapsing in character and synchronous; the jugular veins are unduly prominent in the recumbent position, otherwise there is no evidence of venous stasis or obstruction of either superior or inferior vena cava. The pulmonary excursion and air entry are normal over both lungs, no tracheal tug; no indication of pressure on vagus, phrenic, recurrent, or sympathetic nerve trunks. The electrocardiogram suggests left ventricular preponderance, but is otherwise normal as regards wave sequence and conformity in all the leads.

The x-ray examination defines the left margin of the heart sloping towards the apex region in the sixth intercostal space, $4\frac{1}{2}$ inches from the mid-sternal line; the aortic shadow is abnormally wide—namely, $3\frac{1}{2}$ inches; its margins are blurred and irregular; a pulsating swelling, similar in density to the cardiac shadow, and apparently continuous with this latter, is seen to extend $3\frac{1}{2}$ inches to the right of the mid-sternal line, its edge, sharp and clear, stretching inwards at its superior extremity to join the aortic shadow at an acute angle, its lower margin, distinctly above and separated from the diaphragm, blending imperceptibly with the inferior aspect of the cardiac shadow; the lungs appear to expand normally, and the range of diaphragmatic movement is unimpaired.

Simultaneous curves recorded on a rapidly moving drum from small receivers placed over the apex and the pulsating tumour to the right of the sternum indicate that the impulse in each region is an outward thrust, and measurement makes it clear that the movements are not synchronous; the apex beat, by a fraction under a fiftieth of a second, being the prior event.

The urine was normal in every respect. The Wassermann test was positive.

Discussion of Diagnosis.

The condition was thought to be an intrapericardial aneurysm of the aorta; and in arriving at this diagnosis one was largely guided by the following considerations:

1. The punctate heaving quality of the pulsation, which to palpation appeared synchronous with the arterial pulse.
2. The presence of aortic reflux, which, in view of the positive complement fixation test, would suggest syphilitic disease of the aortic stem, and the conduction of the diastolic murmur from its seat of production over the entire region of the tumour and upwards into the cervical vessels.
3. The fact that the aortic root and the first 2 inches of its ascending arch are situated within the confines of the pericardial sac.
4. That the direction of least resistance to the spread of a dilatation aneurysm proceeding from the aortic root would be downwards within the cavity of the pericardium.
5. The fluoroscopic appearances which identified the shadow of the tumours with that thrown by the heart and great vessels, and the distinct line of continuity which connected the marginal extremity of the tumour with the right-hand border of the ascending aorta; also the definite character of the expansile pulsation which seemed to warrant the exclusion of new growth or encapsuled pericardial effusion.
6. The absence of pressure signs, which, moreover, would be hardly likely to complicate the early stages of an aneurysm originating within, and governed in the matter of the direction of its spread by, the pericardial investments.

After-History and Progress.

Considerable relief to the subjective symptoms was noted during the first three months of treatment, for which, no doubt, physical rest was mainly responsible; there was, however, no material improvement; the aneurysm was palpably increasing in size, a note of February 18th, 1921, giving its outer border a distance of $4\frac{1}{2}$ inches from the mid-sternal line, and the patient was at that time complaining of sharp attacks of pain with the least effort, also cough and a feeling of suffocation, the latter aggravated by any attempt to assume the recumbent position. A month later the outer border of the impulse extended $5\frac{1}{2}$ inches from the mid-line, and examination at this time made it clear that pressure was being exerted on the right bronchus, and, further, that the superior vena cava was suffering obstruction. During April the symptoms increased in intensity; the cough, more especially troublesome at nights, the violent pain and the insomnia combining, at this time, to produce an extreme degree of debility and physical exhaustion; the outer border of the impulse was then to be found extending a distance of $5\frac{1}{2}$ inches from the mid-line; moist sounds were heard at both pulmonary bases; the air entry to the right lung was diminished.

During the last fortnight of life expectoration was profuse, and every paroxysm of coughing was accompanied by violent

and intense prostration. On May 13th the sputum became tinged with blood, and several small recent clots were expectorated. The patient died next day.

Autopsy Findings.

The two layers of the pericardium were found to be closely adherent, and numerous firm adhesions united the pericardial surface to the pleura on each side. The pleural cavities were in great part occupied by dense adhesions in the neighbourhood of the pericardium, but on separating these on the right side the cavity was found to contain a large amount of recent blood clot.

The heart was considerably enlarged; the aortic valve was incompetent; the cusps of the aortic valve, more especially the anterior and right posterior, were thickened, retracted, and distorted; there were no obvious vegetations; the wall of the aorta showed extensive atheromatous changes, the macroscopical appearances indicating an advanced degree of the process. Immediately above the aortic valve, at the junction of the anterior and right posterior cusps, a large saccular aneurysm measuring $4\frac{1}{2}$ inches by $4\frac{1}{2}$ inches arose; it was completely covered by the adherent layers of the pericardium, and it was about two-thirds full of laminated clot; its upper aspect was adherent to the right pleura and lung, which had suffered much dislocation, and here it was that the aneurysm had ruptured into the adjoining pleural cavity and pulmonary tissue.

THE PRESENT VALUE OF THE WIDAL REACTION IN THE INOCULATED SUBJECT.

BY

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An acutely febrile case was recently admitted to the Princess Alice Hospital, Eastbourne, on which I was asked to perform a Widal reaction. The technique used was Dreyer's macroscopical method with the Oxford standard cultures. The result read as follows:

CASE I.

	Serum Dilutions.			
	1 in 25.	1 in 50.	1 in 125.	1 in 250.
Typhoid cultures ...	+	+	+	+
Paratyphoid A ...	Trace	—	—	—
Paratyphoid B ...	—	—	—	—

The reading was "strongly positive for typhoid."

The patient was an ex-soldier, inoculated for enterica during the war, the last time most likely in 1917. The clinical signs were those of continuous fever with some physical signs in the chest, and a provisional diagnosis of pneumonia had been made. The case was subsequently regarded, on the strength of the Widal reaction, as typhoid fever, probably pneumonic in type, it being assumed that the war-time inoculins had died away by this time.

The day after the test the patient was removed to the Borough sanatorium as an infectious febrile case. He died some days later. Clinically the symptoms were not typical of enteric, and a *post-mortem* examination was performed. Miliary tubercle was found to be the cause of death. It was then suggested by Dr. W. G. Willoughby (M.O.H. for Eastbourne), under whose care the case had been, that the positive Widal reaction might have been due to residual effects of war-time inoculation. I reported the case to two leading authorities for their opinions. One of them held that miliary tubercle may accentuate any existing agglutinins. He also pointed out that miliary tubercle and enteric may coexist, and that he had had a case of this kind where Peyer's patches showed no signs of inflammation yet where culturally 50 per cent. of the colonies in the mucous membrane of the lower part of the small intestine agglutinated with paratyphoid B serum in high dilutions, the blood of the same case also giving a highly positive Widal reaction to paratyphoid B, on which alone a diagnosis was made. The second authority offered the opinion that it was quite possible that "residual"

agglutination could account for my results, but that one would expect, if this were so, a lower titre of agglutination than 1 in 250 after four or five years unless the man had given a very high response to his inoculations.

In order to prove what degree of residual agglutinins occur normally I estimated the agglutinin reactions to typhoid, paratyphoid A, and paratyphoid B of five healthy ex-soldiers, with the following very instructive results:

CASE II.

	Serum Dilutions:			
	1 in 25.	1 in 50.	1 in 125.	1 in 250.
Typhoid cultures ...	+	+	+	+
Paratyphoid A ...	—	—	—	—
Paratyphoid B ...	—	—	—	—

This case had had Flexner dysentery in 1916, and agglutinated Flexner's bacillus up to 1 in 250.

CASE III.

	Serum Dilutions:			
	1 in 25.	1 in 50.	1 in 125.	1 in 250.
Typhoid cultures ...	+	+	+	Trace
Paratyphoid A ...	+	Trace	—	—
Paratyphoid B ...	—	—	—	—

CASE IV.

	Serum Dilutions:			
	1 in 25.	1 in 50.	1 in 125.	1 in 250.
Typhoid cultures ...	+	+	+	Trace
Paratyphoid A ...	—	—	—	—
Paratyphoid B ...	Trace	—	—	—

CASE V.

	Serum Dilutions:			
	1 in 25.	1 in 50.	1 in 125.	1 in 250.
Typhoid cultures ...	+	+	—	—
Paratyphoid A ...	—	—	—	—
Paratyphoid B ...	—	—	—	—

CASE VI.

	Serum Dilutions:			
	1 in 25.	1 in 50.	1 in 125.	1 in 250.
Typhoid cultures ...	+	+	—	—
Paratyphoid A ...	—	—	—	—
Paratyphoid B ...	—	—	—	—

We thus have a fairly complete answer to our queries. Fifty per cent. of the cases agglutinate typhoid up to 1 in 250, so that such a high response is no uncommon thing in the inoculated after four or five years. Paratyphoid agglutinins are very slight in all cases as compared with typhoid residua. The explanation of this no doubt lies in the greater strength of the typhoid vaccine given and possibly also because this was used alone at first during the war.

I cannot agree that the existence of miliary tubercle accentuated any potential agglutination titre, otherwise the paratyphoid titres in Case I should have been higher, but of course one cannot generalize too much from these few cases.

The essential point of the matter is that a positive Widal reaction in the inoculated subject, even some four or five