one or other apex, and some adhesion between the visceral and parietal pleura; evidence of some ancient and forgotten pleurisy is common enough. Let us now picture our Territorial standing beside his horse and facing, as is right and proper, the tail end of the animal; he lifts his left foot to place it in the stirrup, closes his glottis to make the effort of mounting, and at that moment the animal's head, a blunt heavy instrument (such as the medical jurists delight in when giving evidence in the courts), strikes him violently on the left chest.

The glottis being closed, all the mechanical requirements are fulfilled for the passage of the lines of force across the left chest through the mediastinum to produce a sudden and violent compression of the right lung.

Now our postulate gets to work, and it is easy to imagine this transmitted force to be sufficient to cause a slight tear in the visceral pleura where, owing to the adhesion, it is unable to slide over the parietes.

But my readers will ask, Why no pneumothorax till twenty hours later?

Here we are helped by our early efforts at mending punctures in tyres, when we found to our disgust that though the slit in the tyre appeared to be efficiently closed, yet a little extra mechanical strain caused the patch to yield and the puncture to recur.

We can therefore, I think, easily picture the slit in the visceral pleura still closed by the adherence between the two pleurae, but so lightly and weakly that a little extra strain would cause the patch to give.

Then, again, we have twenty hours of silent and unnoted work on the part of the traumatic inflammation set up by the tear still further weakening the already inefficient patch, and in the early morning the man closes his glottis to make the effort of getting out of bed (no slight effort to a weary soldier, as we all find), the patch gives way, air rushes into the pleural cavity and the catastrophe arrives at its final consummation.

I trust that the above "reconstitution" will hold water, if not air; at any rate it has the virtue of coming straight from the horse's head, if not directly from its mouth.

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**TRAUMATIC ANEURYSM OF THE HEART.**

By Major R. Priest,

Royal Army Medical Corps.

On March 31, 1927, Private C., aged 21, while serving in India received an accidental wound by a 0·22 bullet which passed through the upper right arm and entered the chest wall in the region of the fifth rib in the posterior axillary line.

Prior to this he had always been a healthy and active soldier. After receiving the wound he fainted and was admitted to hospital, where he
complained of pain in the chest and exhibited some hæmoptysis. After about nine days all signs and symptoms disappeared and he appeared to have made a good recovery, but two months later he was admitted to hospital for paroxysms of cough, and on examination he was found to be anæmic and his liver was enlarged. There was also ascites and oedema of the legs. Examination of the heart showed some enlargement and a systolic murmur was heard at the apex.

He was transferred to England and reached the Queen Alexandra Military Hospital, Millbank, on November 15, 1927. On admission he showed generalized anasarca, much free fluid in the peritoneal cavity, fluid at the bases of the lungs with paroxysmal cough and dyspnæa. He was much relieved by paracentesis abdominis.

Clinical examination of the heart showed much enlargement transversely to the right and left of the sternum. There was no thrill and no diastolic shock. On auscultation at the apex systolic and diastolic bruits were heard all over the precordium and the diastolic bruit could be traced from the aortic area, down the left border of the sternum to the apex. In addition to these bruits, which were constantly present, there were inconstant and more superficial murmurs which were not quite synchronous with the heart’s systole and diastole, heard best at the base, suggestive of a superadded pericarditis. Other concomitant clinical signs of aortic incompetence, such as collapsing pulse, capillary and retinal pulsation, were noted. There was no history of rheumatism or of other previous illness which would ordinarily account for a condition of endocarditis, and there were no clinical or pathological evidences of such.

After paracentesis, the liver was found to be much enlarged and exhibited expansile systolic pulsation. The clinical picture was therefore that of chronic cardiac failure.

The urine showed occasional traces of albumin, but no casts and no red blood-cells were seen in the deposit at any time; the quantity passed varied greatly from 34 to 120 ounces daily.

The blood examination showed hæmoglobin 64 per cent, red blood-cells 4,800,000, leucoocytes 12,500, polymorphs 80 per cent, small lymphocytes 4 per cent, large lymphocytes 8 per cent, eosinophiles 1 per cent, transitionals 7 per cent. The Wassermann reaction was negative.

An X-ray examination showed the presence of a bullet lying near the heart and appearing to move during respiration.

The ascitic fluid was non-inflammatory. The central nervous system appeared normal and the optic discs unaffected. The temperature throughout was normal except for an occasional moderate rise.

During the subsequent course of persistent progressive heart failure it was noted that the systolic and diastolic bruits gradually assumed the character and regularity of a machine, and strongly suggested the presence of some unusual intercommunication between the left and right side of the
heart. Also the more superficial pericardial friction sounds became much less constant.

As regards diagnosis, after eliminating infective endocarditis and other causes of heart failure, the opinion arrived at was that the heart had been damaged in such a manner as to cause an arteriovenous intercommunication, the site of which could not be ascertained during life.

In spite of all dieting, the administration of many cardiac stimulants, cathartics and frequent paracentesis abdominis, the heart muscle finally gave out and the patient succumbed on April 23, 1928, thirteen months after receipt of the bullet wound.

Major W. R. O'Farrell, R.A.M.C., performed the autopsy and found that the pericardium was adherent all over the surface of the heart, particularly at the base. The heart itself was very much enlarged. The right auricle was greatly dilated and the tricuspid opening admitted five fingers. A communicating channel was found leading from the right auricular wall into the aorta, the opening being situated just above the cusp of one of the semilunar valves. The channel was not of uniform calibre, but a distinct sac was present between the auricular wall and the opening into the aorta, the lesion being of the nature of a varicose aneurysm. No wound of entry into or exit from the heart could be found, this being obscured by the resultant adherent and thickened pericardium. The bullet, a small one about the size of a 0.22 bore, was firmly encapsuled in dense fibrous tissue situated in the lung substance at the root of the lower left bronchus. The other post-mortem findings were those of chronic cardiac failure. Conclusion drawn from post-mortem: A gradual development of chronic mediastino-pericarditis and an aneurysmal connection between the aorta and the right auricle, through which parts the bullet must have passed before coming to rest in the left lung, close to the bifurcation of the bronchi.

The interesting features of the case are the comparatively lack of symptoms of cardiac damage immediately after the injury, and entire absence of symptoms thereafter for nearly two months.

The track of the bullet was probably not at first sufficiently patent to allow of much intercommunication between the aorta and the right auricle, but as time progressed the channel became more and more open and direct, thereby throwing gradually increasing work upon the heart to maintain efficient circulation.

I should like to express my thanks to Major W. R. O'Farrell, R.A.M.C., for making the post-mortem examination and the report thereon.