

## **Two cases of pulsus paradoxus / by John Hay.**

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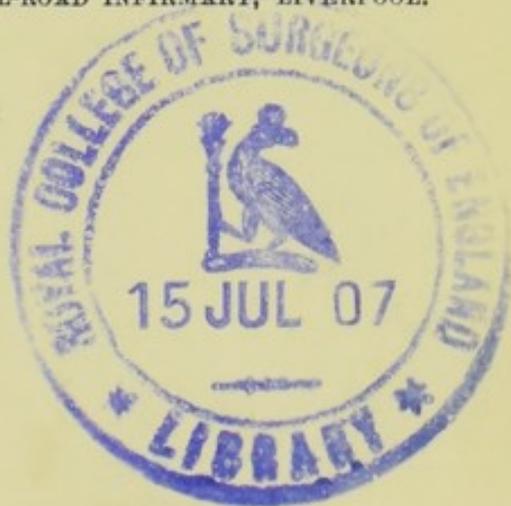
TWO CASES OF  
PULSUS PARADOXUS

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BY

JOHN HAY, M.B. VICT.

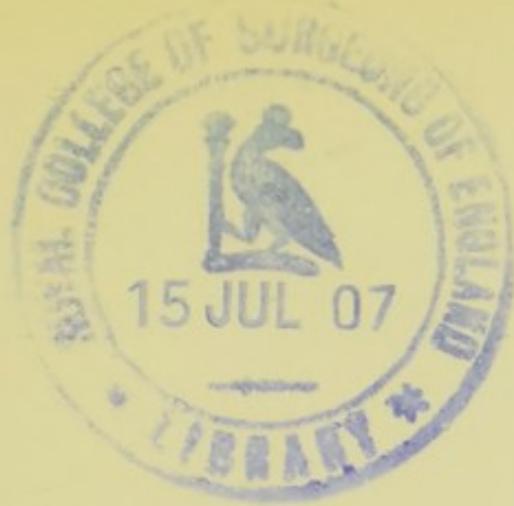
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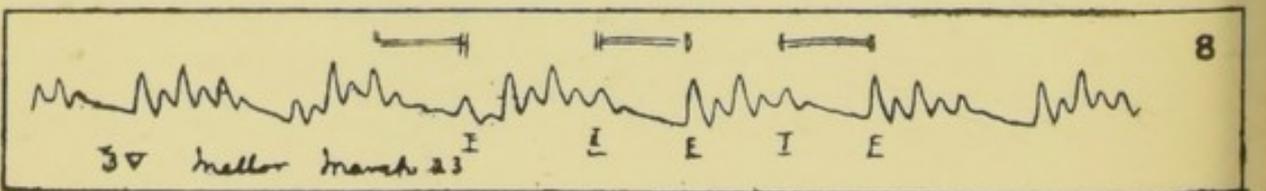
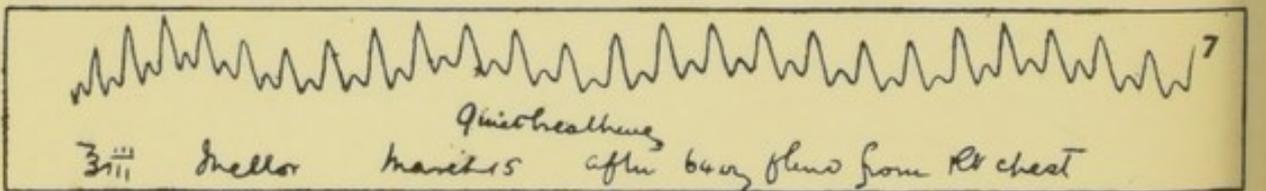
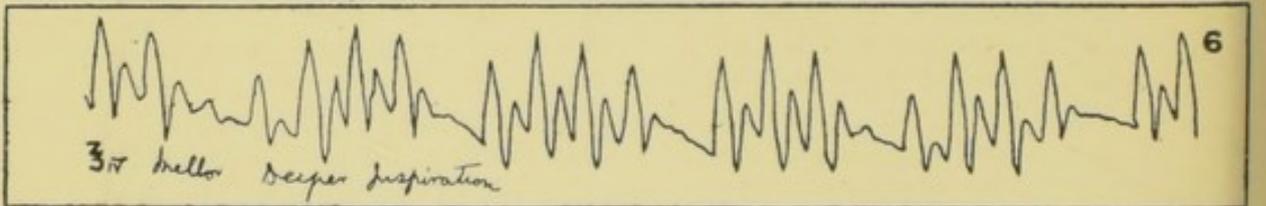
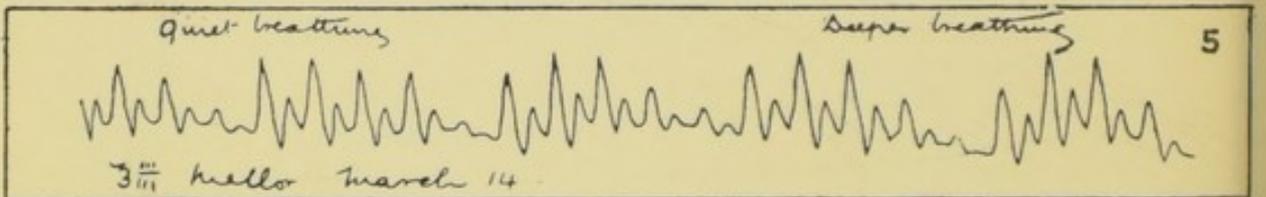
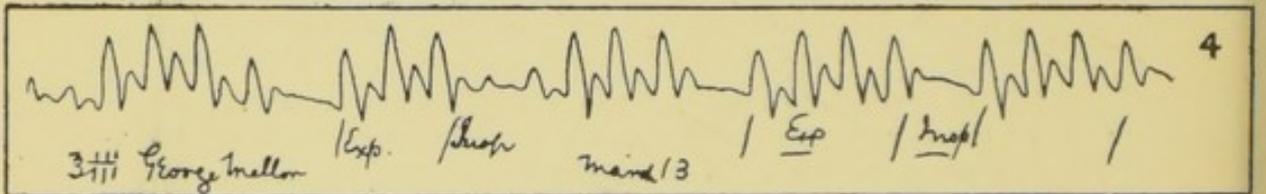
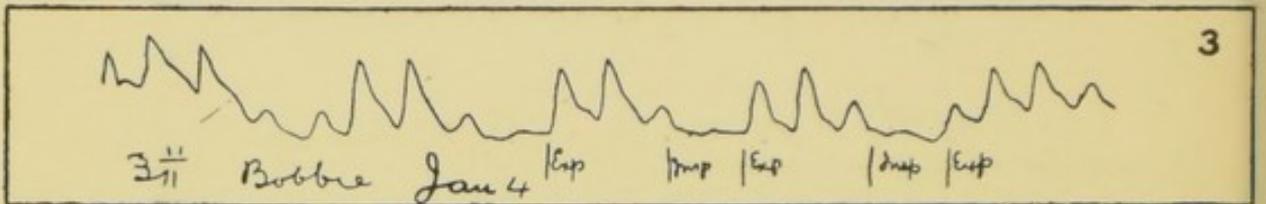
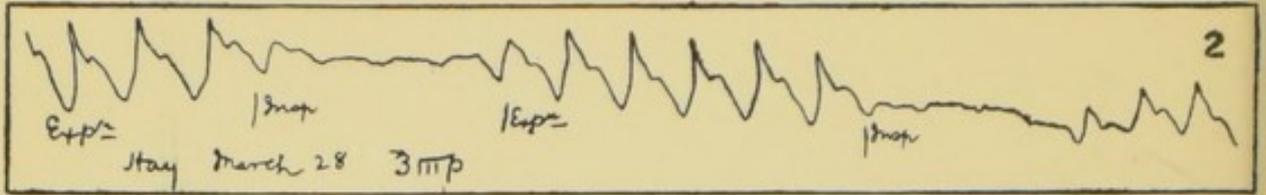
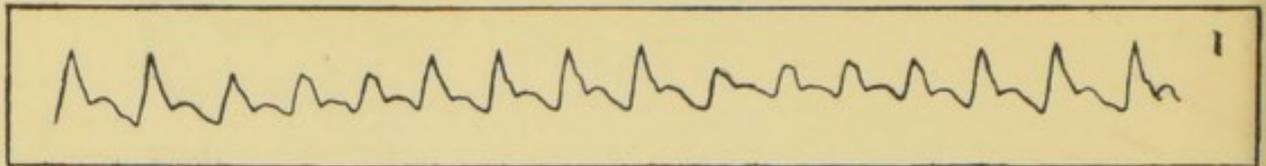
## TWO CASES OF PULSUS PARADOXUS.

UNDER certain conditions the pulse becomes distinctly smaller during inspiration or completely disappears and this form of pulse is named the "pulsus paradoxus." It is not of common occurrence, though probably not so rare as is usually supposed, often being looked upon simply as an irregularity of the pulse whilst the rhythmical character of the irregularity is not perceived. This condition is usually bilateral but is occasionally not so, an interesting case of this nature being recorded by Dr. Thomas Harris of Manchester in *THE LANCET* of April 22nd, 1899, p 1072.

As to the value of the sign, opinions differ greatly, but I think that we may take it that pulsus paradoxus is not of *itself* pathognomonic of any condition, though it cannot be denied that it does exist in perhaps its most marked form in certain cases of indurative anterior mediastino-pericarditis; but there are other conditions in which it is sometimes present, such as pericarditis with effusion uncomplicated by mediastinitis, also in pleuritic effusion and in patients suffering from great inspiratory dyspnoea.

The following two cases illustrate to some extent (1) these three conditions as causative agents; (2) the relief afforded by thoracenteses in cases due to pleural effusion; (3) the association of the pulsus paradoxus with a pulse of low tension, as pointed out by Sir William Broadbent, who drew attention to, and emphasised the fact that, the *high tension* pulse hardly reacts to variations "which produce extraordinary perturbation in the *low tension* pulse"; and (4) the possible short duration of the paradoxical pulse. This is probably due to the fact of its dependence on low arterial blood-pressure—if from some cause the blood-pressure falls in a certain case the pulse becomes paradoxical. Stimulate the heart, contract the peripheral arterioles, and the pulse becomes normal in its rhythm with the increase in arterial

PULSE TRACINGS TO ILLUSTRATE THE CASES.



## DESCRIPTION OF PULSE TRACINGS.

(See opposite.)

1. From normal adult, showing the effect of deep respiration. During inspiration the radial pulsation becomes more frequent and the percussion wave not so forcible. Pressure two ounces.
2. From normal adult, showing the effect of deep inspiratory efforts with closed glottis. Pressure three and a half ounces.
3. Tracing illustrating Case 1. Transient pulse condition caused by marked dyspnoea associated with temporary low arterial tension. Pressure two ounces.
4. Tracing illustrating Case 2, March 13th. Quiet breathing. Pressure three ounces.
5. Case 2, March 14th. Quiet breathing, but somewhat deeper than 4. Pressure three ounces.
6. Case 2, March 14th. Deeper breathing. Pressure four ounces.
7. Case 2, March 15th. Quiet breathing. Shows the effect of the withdrawal of 63 ounces of fluid from the right pleural cavity. Pressure three ounces.
8. Case 2, March 23rd. Pulsus paradoxus returned. No corresponding accumulation of fluid in the right pleural cavity. Pressure five ounces.

tension. This was exemplified recently in a case necessitating venesection on account of great cyanosis due to labouring right heart, the cause being emphysema complicated by an attack of bronchitis. As the arterial tension was good and the pulse (radial) fairly strong, showing that the left ventricle was in good working condition, venesection was performed. Some 22 ounces of blood were drawn off before the man experienced relief. The radial pulse was then soft and regular in time and force and exhibited a rhythmical variation with respiration becoming hardly perceptible during inspiration. As the arterial tension increased again the pulse resumed its normal characteristics, the *pulsus paradoxus* only having been observed for a period of 20 minutes.

CASE 1.—The patient, a male child, aged three years, when in perfect health was attacked by acute bronchitis. This was accompanied by slight rise of temperature, rapid respiration and pulse, the inspiration being short and catchy and the expiration being more prolonged and wheezy. His pulse though frequent remained regular in time and force and of fair tension. One evening I noticed that the pulse was much softer and that during inspiration it could not be felt. I then took some pulse tracings, one of which is reproduced and which shows the relationship between the pulse and respiration (*vide* No. 3). On the next morning the pulse was again normal, being perfectly regular in time and force and not varying with respiration. The important point is that the tension was good. It was no longer a soft pulse. The respirations were somewhat less rapid, but were not markedly different from the condition of the previous evening. The convalescence was rapid and uninterrupted, there being no repetition of the abnormal pulse condition.

Here I think the dyspnoea was the active cause, but so long as the arterial tension remained fairly good no paradoxical pulse showed itself; however, on the occasion of the tension dropping and the pulse becoming soft, then the dyspnoea was able to produce a definite effect, the result being the *pulsus paradoxus*.

CASE 2.—A man, aged 40 years, was admitted into hospital on Feb. 8th, 1899, suffering from broncho-pneumonia complicated by pericarditis with effusion. He had been ill for four days previously to admission. His condition was critical and he had an irregular temperature ranging between

99° and 104° F. The pulse-rate was 130 and the respirations were from 40 to 60 per minute. Under treatment he improved considerably up to Feb. 21st. From that date he slowly began to lose ground, his most prominent symptoms being marked restlessness, a very troublesome cough, considerable dyspnoea, and inability to sleep in the recumbent posture; towards the end he became delirious. His temperature remained a little above normal, the dyspnoea increased, and he died suddenly on March 26th. The definite improvement in the general condition up to Feb. 21st was associated with the gradual clearing up in the lungs. Following this, however, the pericardial condition and the cardiac changes accompanying it became the main factors in the case and were the direct cause of death. On March 13th I made the following note: "The patient is still suffering greatly from dyspnoea, cannot lie down, and is occasionally attacked by great difficulty of breathing, the number of respirations increasing to 70 per minute. During these attacks he becomes almost frantic. They are not associated with marked cyanosis. The præcordial dulness is increased in area, extending two inches to the right and five and three-quarter inches to the left of the middle line, together making a transverse dulness of seven and three-quarter inches. There is dulness in the first left interspace extending one and three-quarter inches from the middle line. No friction rub is to be felt on placing the hand over the præcordium, and only an indefinite cardiac pulsation. The apex beat cannot be seen or felt, and the heart sounds are practically inaudible. There is occasionally a short systolic murmur in the fourth and fifth spaces. There are râles to be heard in the chest, more particularly over the lower lobes, and a pleural friction rub over the right middle and lower lobes. There are definite signs of fluid at the right base, the dulness reaching as high as the ninth spine. The liver is eight and a quarter inches in nipple line and is enlarged and tender. The spleen measures four inches in long diameter. The veins of the neck and arms are tense and engorged, but there is no definite cyanosis." The left pupil was dilated and larger than the right, a condition which I have noticed in several cases of acute pericarditis with effusion. His radial pulse was soft, frequent, and varied rhythmically with the respiration.

Tracings Nos. 4, 5, 6, and 8 in the diagram show well the "pulsus paradoxus." They were, with the exception of

No. 6, taken with the patient breathing quietly. During the time that No. 6 was taken the patient was asked to breath more deeply. The five tracings all show a condition of low arterial tension, Nos. 4, 5, and 6 being distinctly hyperdicrotic. It is noticeable that where the paradoxical condition is most marked, as in No. 6, there the pulse is most hyperdicrotic.

On March 15th 63 ounces of clear fluid were withdrawn from the right pleural cavity. This was followed by considerable improvement in breathing and the patient passed a comfortable night. Pulse tracing No. 7 was taken on the evening of March 15th following the thoracentesis and shows a definite improvement. There is certainly a distinct respiratory curve but otherwise the tracing might pass for that of a normal pulse of low tension.

The above alteration in the pulse was undoubtedly brought about by the removal of the fluid from the chest and although the fluid did not re-accumulate, so far as can be judged by physical signs, the paradoxical condition of the pulse returned as markedly as ever.

Tracing No. 8 was taken on March 23rd, three days before the man's death; he was breathing quietly, though rapidly. The above tracings are all from the right radial artery; others were taken from the left radial artery presenting the same condition. It was impossible to obtain any tracings from the anterior tibial artery on account of the œdema about the ankles and feet. Unfortunately no necropsy was permitted.

I would like here to express my indebtedness to Dr. Nathan Raw, as it is through his kindness that I am able to publish these cases.