Sydenham Society, says: "The jaundice from mental emotion is developed much more rapidly than is even the case in complete closure of the ductus choledochus." He assigns two causes which may produce this effect—"1, by interruptions of the circulation of the blood through the liver, by the influence exerted by the nerves upon the calibre of the portal vein; or, 2, by interruptions to the heart's action, the respiratory and the renal circulation."

Now undoubtedly, if mental emotion of a depressing character, acting thus on the circulation, can produce jaundice—a fact, I think, sufficiently established—it does not appear at all improbable that great losses of blood, producing as they do weakness, irregularity, and obstructed circulation, should lead to the same result.

Again, there are many cases of jaundice recorded resulting from snake bite and pyæmia. Whether they depend on deranged innervation, affecting the circulation through the liver and great vessels, or whether from a change in the condition of the blood, we have not sufficient data to determine. My own opinion is, that the phenomena I observed were due to congestion of the liver, just at that period when reaction had set in. What is really the effect on the circulation of profuse hæmorrhage? I do not mean the immediate effect in those cases where death occurs directly after excessive bleeding. We then find all the venous and arterial cavities empty; but this is not the case shortly afterwards, if the patient should survive. Rapid absorption of watery fluid takes place. It is increased in its proportions even during the abstraction from an artery or vein. Thus Dr. Zimmerman (vol. iv, p. 389) found, in bleeding dogs, that the last drawn portion of the blood contained 12 or 13 parts of water in 1,000 more than the blood first drawn; and Polli notices a corresponding diminution in the specific gravity of human blood during venesection, and has suggested the only possible explanation of the fact; namely, that during bleeding, the blood-vessels absorb very quickly a part of the serous fluid with which all the tissues are moistened.

Nature therefore endeavours, and partially succeeds, in restoring the volume of the circulating fluid. But I may say, in the words of the poet, "quantum mutatus ab illo." It is no longer the same highly stimulating and nutritive fluid; besides which, the weakened condition of the great vessels and cavities of the heart peculiarly favour that condition, which we here denominate passive congestion, and which Andral and the French pathologists designate asthenic or passive hyperæmia.

Now we all know that jaundice may be produced by congestion of the liver of a passive character, as well as from more active congestion arising from over-stimulation. In the former case, the jaundice may first arise in the liver where the bile is reabsorbed into the circulation. These cases I therefore regard as the result of passive congestion coming on with febrile reaction. In themselves, they only form a minute fraction of the tendency to visceral congestion induced by hæmorrhage, or any other debilitating causes, such as the long exposure to high temperature, and the consequent feebleness, the depressing passions, animal poisons, and many other causes which will immediately suggest themselves to your memory.

I think it probable that, if the records of venesection were ransacked, we should find abundant evidence of subsequent congestion, not only of the liver, but of many other viscera. The experience of every medical man teaches him that œdema is a common consequence of excessive losses of blood. This probably is owing, in some measure, to the watery condition of the blood before alluded to, but also in no insignificant degree to the atonic condition of the heart and great vessels, whether from defect of proper nutrition, or from loss of nervous energy.

There is one other theory I think it right to mention in relation to this subject. The one on which I have hitherto dwelt supposes the cases to have resulted from hepatic congestion secondarily to the change in the blood. But you are doubtless aware that many pathologists believe that the cause of jaundice should be sought in a morbid condition of the blood without any abnormal condition of the liver. Frerichs says:—"Breschet, to whom belonged the honour of having first supported the derivation of the bile-pigment from the blood by direct proofs, bluntly expressed this view as to the origin of jaundice as follows: 'Je présume ainsi que l'ictère est occasionné bien moins par la bile, que par le sang.' A similar expression is made use of by Dubreil: 'La teinte ictérique est la suite d'une modification malade des parties constituantes du sang, peut-être de la matière colorante portée sur le serum.' This theory has acquired fresh support from the investigations of Virchow upon pathological pigments. These investigations have proved that, under certain circumstances, a yellow substance is formed from the hæmatine, which, in relation to solvents and reagents, bears a close resemblance to cholepyrrhin.

"Zenker and Funke have brought forward fresh arguments in favour of the intimate relation subsisting between the bile-pigment and the red matter of the blood, by shewing that a modification of the colouring matter of the bile, viz., bifurvine, can easily be transformed into hæma-

toidine, a derivative of hæmatine. This would appear to indicate the possibility of a direct transformation of hæmatine into cholepyrrhin."

Whether the facts I have brought before you are best accounted for by the former or the latter theory, I am not in a position to decide; but of their resulting from each other I have very little doubt.

✓ CASE OF RUPTURE OF THE HEART.*

By GEORGE MAY, JUN., ESQ.,
Surgeon to the Royal Berkshire Infirmary.

A. B., aged 61, during the night of September 23rd, was seized with acute pain in the epigastrium, extending through to the back. For two or three evenings he had complained of slight uneasiness in the chest: he attributed this to indigestion, and prescribed for himself some quinine. Soon after the pain commenced, both arms became cold up to the elbows. I saw him early in the morning, and remarked the anxious expression of his countenance and the tumultuous action of the heart, the sounds being distinct, with a frequent but feeble pulse, which never fell below 120 in a minute. The pain ceased after a few hours, and did not return. During the following night, the breathing became very hurried, with congestion of the lungs. From this time, the symptoms varied but little. He could not lie down or turn on his left side. There was increased dulness on percussion over the cardiac region. The last day or two of his life, his legs became swollen; and he died on October 11th, suddenly, seventeen days from the commencement of his illness.

POST MORTEM EXAMINATION, twenty hours after death.—The body was fat. The pericardium was filled with soft clotted blood, but no serum. The anterior wall of the lower half of the left ventricle was slightly adherent to the pericardium. The heart was moderately covered with fat, and pale; the anterior wall of the left ventricle was very thin, not thicker than cartridge-paper, and supported by a layer of partly organised deposit, which was firmly adherent to it. Near the auriculo-ventricular septum were two small openings in the anterior wall, about large enough to pass a No. 6 bougie. There were atheromatous deposits in the aorta.

I can only find one case, recorded by Dr. Quain in the *Pathological Transactions* for 1861, in which the patient survived more than a few hours; and in that case there was no distinct proof of rupture. That the illness depended on the heart, and would probably end fatally, was early diagnosed; but, as I had not heard of any case of rupture in which the patient survived more than a few hours, the true nature of the illness was not detected.

In a case of rupture of the left ventricle which I presented to the Society some years ago, death occurred within one hour.

In early life, my patient had lost his sight from small-pox, and for the last few months had ceased to take exercise, not having left the house during that time.

The case seems to have been one of aneurism of the left ventricle, associated with fibroid degeneration. The thinning of the wall of the ventricle was partly remedied by a deposit of fibrine; but this, not taking place close to the mitral valve, allowed rupture to take place. The escape of blood into the pericardium caused adhesive inflammation, but did not close the aperture; and the patient existed in a condition similar to that produced by effusion of serum into the pericardium. The smallness of the aperture was the reason why he probably escaped immediate death. Until his last illness, he had never suffered from any symptoms of inflammation of the heart.

✓ ANEURISM OF THE HEART.*

By T. L. WALFORD, Esq., Reading.

MR. G., aged 62, died October 1st, 1868, having first complained of the illness which caused his death in November 1865. He complained, when taken, of severe pain in his chest and arms. He passed an agonising night—at one time on the bed, at another on the floor, shifting his position continually, in vain attempts to get ease. All subsequent attacks came on in a similar manner. He was needy in circumstances, but inactive in disposition, and was strictly temperate.

The symptoms generally present were: shortness of breath, which was easily disturbed; pain in the chest; oppression; and inability to lie. Of the sounds of the heart, only one was observed at the apex; a slight *bruit* was believed to be heard at a distance. The least exertion made him pant as if he had been running. The pulse was quick, 100, without firmness. Such was the record in January 1866.

* Read before the Reading Pathological Society.