it should never be attempted to make a diagnosis by aid of the ophthalmoscope unless the appearances are so well marked as to admit of no doubt about their nature. In a very valuable paper in the *British Medical Journal* for 15th June, 1872, Dr. Broadbent says:—"According to my experience, while some degree of hyperæmia of the disc is common in tubercular meningitis, well-marked choking of the disc is rare, and I have never met with an extreme example, such as I have met with in cases of cerebral tumour."

In conclusion, Sir, it would seem to me that, while true congestion papilla is not pathognomonic of intra-cranial tumours, it is extremely rare as a symptom of any other intra-cranial disease. It has been observed in a few cases of meningitis, chiefly in children, so that its value as a symptom of cerebral tumour is even greater in adults than in young people.

ART. VII.—*The Pathogeny of Hæmorrhagic Infarction of the Lungs.*^a By GERALD F. YEO, M.D., Assistant-Physician to the Whitworth and Hardwicke Hospitals; Lecturer on Institutes of Medicine in the Carmichael School of Medicine.

FOR many years pathologists have been familiar with the hæmoptysis which is a frequent, and often a fatal, symptom of heart affections; and the pathological changes in the lungs, with which such hæmorrhage is usually associated, have been found to be strikingly similar in the majority of cases, and easily distinguished from the many other lesions which give rise to spitting of blood. Laennec,^b who first described the anatomical characters of the lung affection, called it *pulmonary apoplexy*, a name which has held its ground ever since, in spite of attacks from all quarters; and the likeness to the cerebral lesion, which suggested the term, is rather borne out than refuted by modern research. I do not, however, wish to uphold the name as quite satisfactory, nor will I adopt it, because of the indiscriminate manner in which it has been applied to various head affections—the word "*apoplexy*" being now used rather to express the clinical idea of a group of symptoms, than any single pathological process. The name "*infarctus*," which has been used for a long time in Germany, has a more

^a Read before the Medical Society of the College of Physicians, February 10, 1875. [For the discussion on this Paper, see page 243.]

^b *L'Auscultation Mediate, &c.* 1819. Tome II., p. 40.

definite meaning, and is more expressive of the condition at present under consideration.

The anatomical characters of the lesion differ somewhat according to the period which has elapsed since its commencement, as the hæmorrhagic centres go through a series of changes which are variously modified in each case by its individual peculiarity. A few days after their formation they appear as isolated spots of intense engorgement, varying in number—there being seldom fewer than three or four of notable size. They are situated in different lobes of a lung; or, in many cases, both lungs are affected. These nodular solidifications are extremely hard and resistant, almost black in colour, and perfectly airless. One of their most striking characteristics is their sharply defined margin, the lung tissue immediately around them being soft, pale, and elastic, and—at least in the early stages—to all appearances healthy; owing to its property of partially collapsing when removed from the body, it retracts from the indurated nodules, which are left standing out on the pleural surface like knobs, which prominence suggested the name *furuncular pneumonia*. Their size varies very much, though they never occupy more than a comparatively small part of a lobe, seldom attaining a diameter of more than three or four inches, and they are frequently not larger than a filbert nut.

Their shape is a point upon which I wish to lay particular stress, both as it is most characteristic and as it helps to explain their production. When situated on a flat surface of the lung, their superficial aspect is circular; but, when they include the sharp edges of the lung, which is a very common occurrence, the outline is distinctly wedge-shaped. When a section is made of them they are always found to present a typical cone-shape, the apex of which points to the root of the lung, and the base is situated at the surface. Even when placed deep in the structure of the lung, this cone-shape may be seen by splitting them in their long axis, which points to the entrance of the great vessels. The surface of their section is finely granular, and the deep black colour hides the markings of the lobules, which are generally so well seen in sections of pneumonic consolidations, from which the cut surface of the hæmorrhagic infarction may be distinguished by their homogeneous blackness. Another point of interest in connexion with their cause is, that the vessels ramifying in the cone, which enter its apex, are invariably filled with tough, adherent, dry fibrinous clots, even extending to

the very small branches. The bronchi are usually more or less filled with bloody mucus, which varies in character, according to the period at which the parts are examined.

These appearances are often first noticed in the dead-room, as the difficulty of recognising their production during the life of a patient is extreme—for all the symptoms, which may be considered as characteristic of them, occur in cases of heart disease, without any such lesion of the lung being found after death. As the sequence of these clinical phenomena may guide us a little in arriving at a correct conclusion as to their cause, I shall briefly enumerate the leading symptoms.

The first indication, commonly, is an attack of sudden, intense localised pain, or stitch, in the side, followed by great distress, uneasiness and dyspnœa. After some hours (in two cases, of which I have notes, six and five hours respectively) more or less copious spitting of blood comes on. This is the most constant symptom; but the quantity of blood varies in different cases, as does also its colour, although in some cases the blood is extremely dark, which fact Graves^a and Walsh^b consider characteristic; and, no doubt, it is so when it has the sooty look they describe, but this is too seldom found to make the sign of much practical value. In cardiac cases the bleeding generally continues to the end, or at least for a considerably longer time than most forms of pulmonary hæmorrhage. In cases where the heart is healthy, the bleeding may stop in a day or two, sometimes being replaced by a copious discharge of purulent matter, showing the formation of abscess.

In most cases the physical signs of solid lung-tissue cannot be elicited, as the area is too small, and it is only when the hæmorrhage is actually taking place that the bubbling râles, described by Laennec, can be produced; I have never been able to detect them. In one case I could distinguish a small circumscribed area of dulness, over which there was audible a clear harsh friction sound. Some authors lay great stress on the sudden increase in the precordial area of dulness, which they explain by the sudden mechanical dilatation of the right heart from increased pressure in the pulmonary artery. This physical sign I have had an opportunity of observing in two separate cases in a most striking manner, but in each of them I was able to satisfy myself, by the *post-mortem* examination, that the dulness depended upon the solidification of

^a Clinical Lectures. 1864. Page 540.

^b Diseases of the Lungs. 1860. Page 416.

the edges of the lungs covering the heart, and not upon sudden distension of the right cavities.

The sudden occurrence of irregularity of the heart-beat is a sign of considerable value and great constancy. I have never been able to meet with the sudden disappearance of a well-marked murmur which has been mentioned by some as a sign.

Although the typical examples given in his description of the disease are associated with exactly the kind of heart affection which most commonly produces parenchymatous bleeding, Laennec^a does not attempt to connect the heart and lung lesions as cause and effect, but immediately after his description became known, writers seem unanimous in ascribing hæmorrhagic infarction to undue strength of the right side of the heart, just as the cerebral disease may be induced by hypertrophy of the left ventricle; at the same time Laennec's view, that the blood oozed into the air-cells from the terminal branches of the pulmonary artery, was generally accepted, which same explanation was given by Andral^b of a lesion under the title of "*Lobular hyperæmia*," the clots in the vessels being supposed to depend on the stagnation of the blood from its non-aëration.

The chief opponent of this view was Sir Thomas Watson,^c who took exception to all these explanations. Although I think his views are quite untenable, as they have been accepted by many of the English text-books, I shall describe them briefly. He considers valvular disease of the *left* side of the heart to be the immediate cause of the lung complication, and that the primary hæmorrhage is into the larger bronchi (from the bronchial artery), some of the blood being drawn into the air-vesicles during inspiration causes the blocking up of a limited region of the lung. This ingenious theory was founded on one single case of suffocation by hæmorrhage into the air-passages, in which the lungs were found to be studded with small nodular consolidations. As the case is not followed by confirmatory observations, I am inclined to reject the theory on the following grounds:—It is not in accordance with the common clinical history of the affection, as the hæmorrhage usually occurs some time after the dyspnœa, &c. There was not sufficient time for the development of infarctions in the case given by Sir Thomas Watson, as the patient was dead in "two minutes."^d

^a *Loc. Cit.* P. 49.

^b *Anat. Path.* 1830. P. 299.

^c *Lectures on Prin. and Prac. Physic.* 1857. Vol. II. P. 152.

^d *Loc. Cit.* P. 156.

It seems physiologically impossible that the air-vessels could be so tightly filled with a viscid fluid like blood, by a few inspiratory efforts, as to displace the reserve, residual, and permanent air from them. This view does not explain many of the *post-mortem* appearances.

The chief anatomical peculiarities of the infarction in the lungs remain then but poorly explained, for any distant cause of engorgement, such as mitral obstruction, should affect the entire lung equally, and therefore cannot adequately account for any of the constant characteristics of the lesion, which I shall now recapitulate :

- α. Sharp demarcation from surrounding tissue.
- β. Plurality of the centres of engorgement.
- γ. Their scattered distribution in the lungs.
- δ. Their regular conical shape.
- ε. The dense thrombi in vessels.

The key to these difficulties was first found by Virchow, who, by his exhaustive researches on embolism, threw light on so many otherwise inexplicable pathological processes, that they may be considered to have created a new era in pathology.

He found by experiment that solid particles of various kinds, when introduced into the circulation, acting as artificial emboli, were carried along with the blood-stream till they reached a vessel too small for their reception; here they became impacted and occluded the vessel. If they are introduced into a vein, they of course pass by the right heart into the lungs, where they are arrested; and if introduced into an artery, they are stopped on their way to some part of the general capillaries. That the presence of free fibrinous masses, or emboli, is common in the human subject, he also proved, and explained how they arise by the projection of a growing end of a thrombus from a tributary into a larger vein, where it is broken off by the passing stream of blood. The impaction of such an embolus causes changes in the part supplied by the plugged artery, the intensity of which depends upon many circumstances, the most important of which are—

1st. The freedom of anastomosis enjoyed by the distal part of the plugged artery, there being a certain number of organs whose terminal branches have no anastomotic connexions—namely, the lungs, spleen, kidneys, and brain.

2nd. The properties of the substance forming the embolus,

which produces various results according as it is more or less mechanically or chemically irritating.

3rd. The efficiency of the valves in the veins of the heart.

The varieties in the vascular arrangement in different organs is so great, and the emboli may vary so immensely in size and properties, that we cannot be surprised at a long list of results which may follow the process. The simplest of these occurs where there is a perfect system of anastomoses, such as the vessels of the limbs, where pain and passing *œdema* are the only effects; when the substance forming the embolus is of an irritating nature it sets up local *inflammation*; or, if the plug be very large, *gangrene* may follow—the most common result of a simple embolus being, however, an *infarction*, which commences as congestion, and the part soon becomes dry, dense, and fibrinous. In many cases and situations these infarctions become so intensely engorged that they acquire the name *Hæmorrhagic*, when they remain as black masses such as those I have described as occurring in the lungs. In other cases the hæmorrhagic stage passes on rapidly to suppuration, when we have the so-called *metastatic abscess*. In other cases, again, the plugging of the vessel may be followed by *atrophy* of the part supplied.

Virchow's observations were confirmed by a host of experimenters on the Continent, and there now no longer exists a shadow of doubt that the plugging of a vessel causes certain changes which are capable of explaining all the lesions attributed to them; and since Cohnheim's recent work on the subject,* the greatest sceptic must admit these views, as that pathologist showed it was easy, with the aid of the microscope, to watch, step by step, the process of infarction taking place in the transparent tissues of a frog, where he found the changes following the sticking of a wax embolus to be divisible into the following stages:—

- α. Temporary dilatation of the vessels.
- β. Peculiar oscillating movements in the stream of blood, with a tendency back towards the embolus, which produces crowding.
- γ. *Œdema* of the tissue and emigration of the white corpuscles.
- δ. Hæmorrhage from the capillaries.
- ε. Absolute stand-still of blood.

All his observations were confirmatory of Virchow's explanation

of the process, and throw new light on the immediate cause of the hæmorrhage—a point which Virchow does not at all satisfactorily explain.

The explanation usually adopted was that given by Ludwig. He believed that the blood corpuscles, which had free access to the distal end of the plugged vessel through the neighbouring capillaries, became lodged there, and so occluded some of the capillaries, while others formed a kind of blind end to the collateral arteries. In the latter set of capillaries the tension rose to that of the vessels leading to them, and this their delicate structure was unable to bear, and accordingly allowed the escape of blood. This view, which hardly bears critical physiological examination, seems rendered more improbable by Cohnheim's observation that the hæmorrhage occurs before the coagulation of the blood in the vessels; and his experiments led him to adopt another view—namely, that the constant renewal of the blood in the vessels was necessary to sustain the perfect function of their wall, and he concluded that capillaries lost their function—*i.e.*, their power of governing the retention of the blood—when deprived of a constant supply of fresh blood. However, he was unable to detect any corresponding structural change as evidence of such a process of starvation. According, then, to Cohnheim, the order of events would be:—

1. Retardation of the blood-stream.
2. Œdema and emigration of white corpuscles.
3. Increasing stasis from pressure of œdema, &c.
4. Starvation of blood-vessels—first, the capillaries; then veins; and, lastly, arteries.
5. Impairment of their function, causing inability to retain the blood.
6. Hæmorrhage.

If we adopt, then, the embolic process as the cause of the infarction of the lung which accompanies the hæmorrhage in heart disease, we must find a constant and ready source of origin for the emboli. For this, in the majority of cases of dilatation, we have not far to go, as in the right auricular appendix, or among the trabeculæ of the right ventricle, there is generally an abundant supply of small coagula, formed where the movement of the blood is most retarded, as it has now been ascertained that just as the wall of the vessels must have a fresh supply of blood to retain their perfect life, so the blood must be kept repeatedly in contact with the healthy wall of a vessel to prevent its coagulation. These cardiac thrombi

sometimes appear pedunculated, when they are called *polypi*, which, if once detached, must be carried into the branches of the pulmonary artery, where they set up the changes I have attempted to describe.

In a case, then, of disease of the heart which terminates fatally by pulmonary infarction, the probable chronological sequence of events may be thus enumerated:—

1. Prolonged impediment to the pulmonary circulation, frequently caused by valvular disease of the left side of the heart.
2. A certain amount of injury to the pulmonary vessels, caused by the constant obstacle to the flow of blood.
3. Dilatation and hypertrophy of the right heart, which always follows the persistent impediment to the pulmonary circulation.
4. Coagulation of the blood, which commonly occurs on the surface of the ventricle or in the auricular appendix.
5. The detachment of a portion of such a fibrinous clot.
6. An embolus is thus formed which sticks in a branch of the pulmonary artery.
7. *Hæmorrhagic infarction* and bleeding into the air passages, in the manner already described.

These variations and hæmorrhagic tendency depend, I believe, on (*a.*) disease of the pulmonary vessels, and (*b.*) hypertrophy of the right side of the heart, circumstances which cannot be imitated by experiment, and hence Virchow could never produce typical pulmonary apoplexy in the lower animals. Thus, besides the properties of the embolus, the anastomoses of the artery, and the supply of valves in the veins of the part, there are two conditions to which experimental pathologists seem not to have ascribed sufficient importance, for they doubtless materially influence human pathology.

If we examine carefully the morbid appearances in various cases of pulmonary infarction, we find differences in the anatomical characters and results, which can hardly be explained by the circumstances which govern experimental infarction. Thus, in some cases depending on cardiac disease, the hæmorrhage is very profuse, and even the tissue ruptured; while in others, no doubt rarer cases, the bleeding is comparatively slight, and the infarction dries up, evidently with a tendency to form a mere fibrinous induration.

If we analyse the various events in the development of dilatation of the right side of the heart, we find that the injurious influences

which generally commence at the left side must pass to the right by means of the pulmonary circulation, and the vessels themselves must surely become more or less deteriorated or diseased by the transmission of such mechanical impediment; and as I have constantly found material changes in the walls of the pulmonary vessels, in cases where there has been copious bleeding, I believe that deterioration of the walls of the pulmonary vessels is a very important factor in its production.

The second important circumstance influencing the amount of hæmorrhage is the strength of the right ventricle, which is usually more or less increased by the same cause which produces the dilatation; though the hypertrophy is often too slight to attract special attention, it becomes a most active agent in causing bleeding where the vessels are diseased, and the tension within them increased by a portion of the usual blood channels being occluded.* I believe that either, or both, disease of the pulmonary vessels and hypertrophy of the right ventricle, are essential to the production of those profuse bleedings which so often prove fatal in heart affections.

The conclusions we may be allowed to draw from the foregoing remarks can be thus briefly recapitulated:—

That the commonest cause of hæmoptysis in cardiac disease is pulmonary infarction produced by embolism.

The occlusion of a vessel by an embolus fully explains the anatomical characters of the infarction and the occurrence of hæmorrhage.

That the origin of the embolus may usually be traced to the cavity of the right side of the heart, where fibrinous clots are frequently formed *intra vitam*.

That the alteration in the capacity and strength of the right side of the heart is commonly the immediate cause of the embolism.

That disease of the smaller branches of the pulmonary artery is commonly associated with the change in the right side of the heart, and is an important agent in regulating the amount of the hæmorrhage which takes place.

* Several specimens of infarctions were shown, illustrating the anatomical characters of the affection. Also the hearts of two patients who had died of rapid pulmonary hæmorrhage. In both of these, the right cavities were dilated and hypertrophied in a most unusual degree. The pulmonary arteries showed disease of their coats in the larger branches.

In one of the hearts there was no sign whatever of disease on either the mitral or aortic valves, and the orifices were normal in size; the left cavities were enlarged, but slightly so when compared with those on the right side.

That bleeding from the bronchial mucous membrane cannot produce, or even simulate, true hæmorrhagic infarction.

That valvular disease of the left side of the heart is a very common starting point of all these pathological changes, but that such disease is by no means an invariable concomitant of pulmonary apoplexy.

ART. VIII.—*Dilatation of the Stomach.** By ARTHUR WYNNE FOOT, M.D.; Junior Physician to the Meath Hospital.

THE following case is one of great dilatation of the stomach, consequent upon narrowing of the pylorus, the result of an ulcer situated in that neighbourhood. It was characterised by periodic vomitings of large quantities of acid, fermenting liquid, rich in sarcinæ. The patient derived much relief from the washing-out of the stomach with alkalised water, and died apparently from an attack of sub-acute peritonitis, for which, however, there was no obvious cause, and which did not appear to be in any way attributable to the use of the stomach-tube.

The subject was a country boy, aged nineteen, of the labouring class, who was admitted to the Meath Hospital under my care, 28th Oct., 1874, complaining of “a windy swelling” rising in his abdomen, and at intervals ending with a copious vomiting of liquid so sour as to injure his teeth, also of heart-burn, “a drunkenness in his head” (*vertigo dyspeptica*), and frontal pain. He further mentioned that he felt “a working and boiling” in his stomach; that this sensation and his almost constant heart-burn were relieved for the time by vomiting; that the vomited matters hissed and frizzled in the basin; that he felt his stomach as if it were a bag; that it “joggled” while he walked, so that he used a belt to support it. His appetite was very defective; his bowels generally acted once a week, and on one occasion not for twelve days. He suffered greatly from flatulence (gastric); the wind always escaped upwards—never downwards. His teeth were perpetually “on edge” from the sourness of the periodic vomitings. Such were the symptoms he complained of on admission.

On personal examination he was found to be deficient in flesh and strength. He presented the *facies dyspeptica*, a dull, spiritless

* Read before the Medical Society of the College of Physicians, February 10, 1875.
[For the discussion on this Paper, see page 245].