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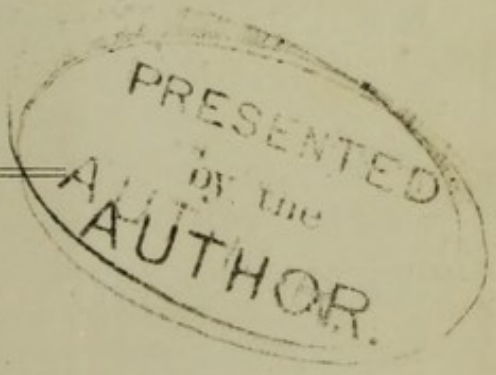
ON FIBRINOUS DEPOSITION IN THE HEART.

BY

BENJAMIN WARD RICHARDSON, M.A., M.D.,

MEMBER OF THE ROYAL COLLEGE OF PHYSICIANS;
SENIOR PHYSICIAN TO THE ROYAL INFIRMARY FOR DISEASES OF THE CHEST; LECTURER ON PHYSIOLOGY AND PATHOLOGICAL ANATOMY AT THE GROSVENOR PLACE SCHOOL OF MEDICINE; HONORARY FELLOW OF THE SOCIETY OF SCIENTIFIC MEDICINE OF BERLIN; AND OF THE PHILOSOPHICAL SOCIETY OF ST. ANDREW'S.



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Fibrinous Deposition in the Heart.

CHAPTER I.

ON opening the bodies of the dead, we find any blood that may be in the heart in one of three conditions.

(*a.*) As a coagulated red clot, but with no surrounding serum. In this latter respect, the blood differs from ordinary blood drawn from the body, and set aside for coagulation. It must not be assumed, however, that in the dead body serum does not separate. It does separate, the same as in cases where blood is drawn; but it appears to me that for some time after death an osmotic action is continued in the body, by which the serum is removed. When, some hours previously to the injection with wax, the vessels of the subject preparing for dissection are injected with a watery solution containing arsenic or sulphate of zinc, the solution thus thrown in does not remain in the vessels; it is absorbed and received into the soft parts, where the capillaries are so plentifully distributed. This same kind of absorption, I presume, removes the serum as it is extricated from the clot, thus leaving the *post mortem* red clot free of serum.

(*b.*) There is another condition of blood found after death, in which perfect fluidity of blood is maintained. This fluidity —which, by the way, is very rare—is met with in two classes of cases. In some instances, from chemical changes going on during life, the coagulability of the fibrine is destroyed. In cases of typhus—I mean true petechial typhus—this condition has been observed; it has also been seen in scurvy, yellow fever, and in examples where water has been largely introduced into the circulation in the lower animals. The fixed alkalies carried on to a poisonous result, will cause the same fact. Cyanide of potassium and antimony lead to a like phenomenon. If blood thus obtained be set aside, it will not coagulate, although placed in conditions the most favourable to coagulation. In another class of cases, the blood may be found fluid in the heart, not because the coagulating process is destroyed, but because the conditions favourable to coagulation are not presented. The law on this point seems to be this. If in any given case the life of a person is suddenly extinguished while yet the blood is in a state of perfect fluidity in the vessels, and if in the cause which produced death no portion of the closed circuit of the vascular system is opened, and if the vessels are themselves filled with blood, then the volatile alkaline solvent by which the blood has been held fluid is retained, and the blood remains fluid until it is exposed to the air. On such exposure, however, coagulation takes place; but, as the temperature of the blood falls after death to the temperature of its surrounding medium, the coagulation will be slow, as compared with that of warm blood drawn from a living man. The period required for coagulation, in short, will be the same as it would be in a mass of newly drawn blood suddenly reduced in temperature from 98° to the temperature of the dead. This condition of fluid blood after death is met with in instances where the death has suddenly occurred from shock, from chloroform syncope, from hanging and other varieties of asphyxia, and the like.

(*c.*) The third class of cases embraces those in which the blood is found in the heart with the fibrine separated; and as these are the cases of most interest to us at this time, I may leave the others without further note, in order patiently to trace out, and as nearly as possible in the order described, the following points.

I. The *various forms* of concretion in the heart, and the modes by which they are produced.

II. The *conditions of disease* under which concretions are produced, and the periods in the course of a disease at which they occur.

III. The *symptoms* indicating fibrinous deposition, and the point at which the deposition has taken place.

IV. The *value of a knowledge* of the subject of fibrinous deposition, in the practical treatment of disease.

I.—Forms of Fibrinous Separation.

The simplest kind of separation is described well in Fig. 1. The fibrine in this case lies on the superior surface of a red coagulum. It forms, in this way, a thin layer, shaped to the clot beneath. It is ordinarily convex above, where it comes into contact with the wall of the cavity; and concave beneath, where it is in contact with the blood. If the clot be turned over, so that the fibrine surface rest on the table and the red blood-clot look upwards, the red portion may be gently washed away by the stream from a wash-bottle, and the layer of fibrine may be left entirely separated. It will then be found to consist of a simple layer of white matter, varying in thickness from one to four lines, firm rather in character, and of a very pure white.

This form of clot may be met with in any of the cavities of the heart, or in the great vessels. If the heart is examined *in situ*, and the cavities are cut into from above, the fibrinous layer will meet the eye as soon as the cavities are laid open. The fibrine is separated, in short, from the under stratum of blood, the body lying on the back at the time of the separation. This form of concretion, though it may occur, as I have said, in any part of the heart, is most common in the auricles. Occurring in the pulmonary artery or aorta, the separation takes the form of a part of the circumference of a cylinder; the superior surface being convex, the inferior concave—modeled, that is to say, to an under column of red blood. I ask special attention to this first form of fibrinous deposition, whether it occur in one of the cavities of the heart, or in one of the large vessels. I should prefer to designate it, for clearness sake, as the *static* deposit, because it is formed from blood at perfect rest.

A second variety of fibrinous separation in the heart is that of a firm white striated mass, filling a cavity or a tube. In some instances, especially where the concretion exists in the auricle, and when it occurs in the course of an acute inflammatory disease, it may fill the cavity altogether, and even distend it. In other cases, although it may form the greater part of the matter contained in the cavity, and although it may itself be purely separated, it may be surrounded by a layer of red blood. This layer will not be found only beneath the concretion, but, as I say, surrounding it; this form of concretion is thus entirely distinct from that described under the first head. When concretions so formed lie in a vessel, as the pulmonary artery, they may extend very far into the ramifications of the vessel. Where they end as separations of fibrine, they are, as a general rule, tipped at their extremities by a layer of red blood, which may be washed off, leaving behind a fine pointed conical termination of fibrine. The concretion occurring in the manner thus described, is often beautifully modeled to the part in which it lies. If it lie in the auricle, it will be found with its surface marked by the little musculi pectinati, as shown at c in Fig. 2, taken from a recent specimen; or if it pass into the pulmonary artery or aorta, it will be found bearing the impress of the semilunar valves at the point where it traverses them, in the way marked in Figure 3, where a concretion (c) filling the right auricle, partly filling the ventricle, and passing into the pulmonary artery, is marked over the line of the semilunar valves with a distinct depression, cup-shaped, and originally an exact print of one of the semilunar curtains.

There is another form of deposition in which the surface is grooved more or less deeply. The groove thus exhibited may be simply curved, or spiral. The line of the groove, whatever that may be, indicates the course of what was once a column of moving blood; and, indeed, a portion of red blood will in most cases be found in the groove. The simply curved grooves are most frequently seen in concretions formed in the right auricle. Here the superior groove, running nearly straight (see A, Fig. 4), marks out a current of blood which, whilst life still existed, found its way from the superior vena cava through the auricular cavity into the auricle, and over the surface of the concretion. The inferior groove, on the other hand, very sharply curved, indents the under surface of the concretion, and takes the direction of the column of blood, which during life passed from the inferior cava through the auricular cavity into the ventricle. The straight and curved grooves of a concretion laid down in the right auricle are in almost every case presented as above. But there is one rare modification; I have seen it at most but twice, in which the concretion is divided into two distinct halves, one half lying in the upper part of the auricle, the other in the lower part, the streams of blood from the two cavæ separating the two fibrinous masses, and grooving them both, in the direction of the venous currents. In these examples, the concretions, superior and inferior, have a firm attachment to the auricular wall.

The spiral groove, whenever it is met with, is found in concretions which extend into the large vessels, as into the pulmonary artery on the right, or the aorta on the left side. This spiral groove I observed and pointed out at the Medical Society of London so far back as the year 1851. I have met with it frequently since that time; and in B, Figure 4, it is excellently shown from a fresh concretion. The groove thus formed is caused by a current of blood, which during life has made its way from the heart by a winding course around the fibrine cylinder.

There is another form of deposition, in which the centre of a

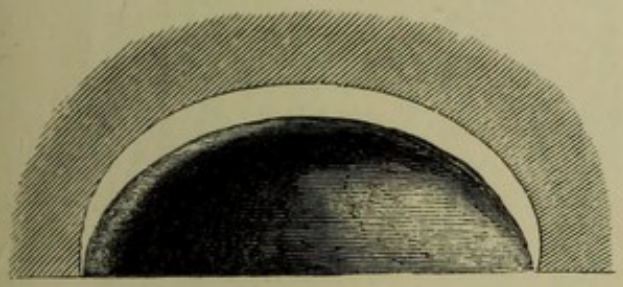


Fig. 1.—Deposition of Fibrine on the surface of a Coagulum.

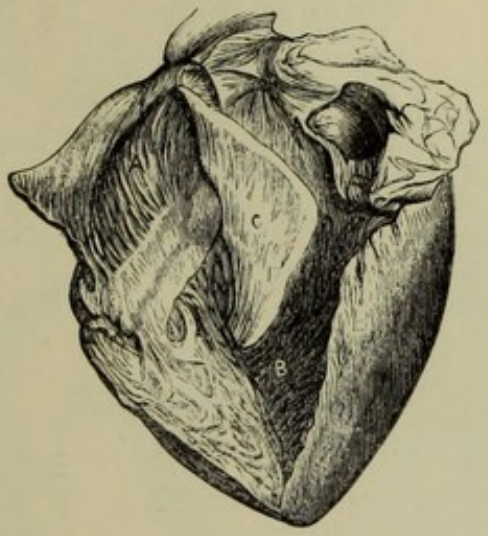


Fig. 2.—Concretion in the Right Auricle, marked by the Musculi Pectinati.

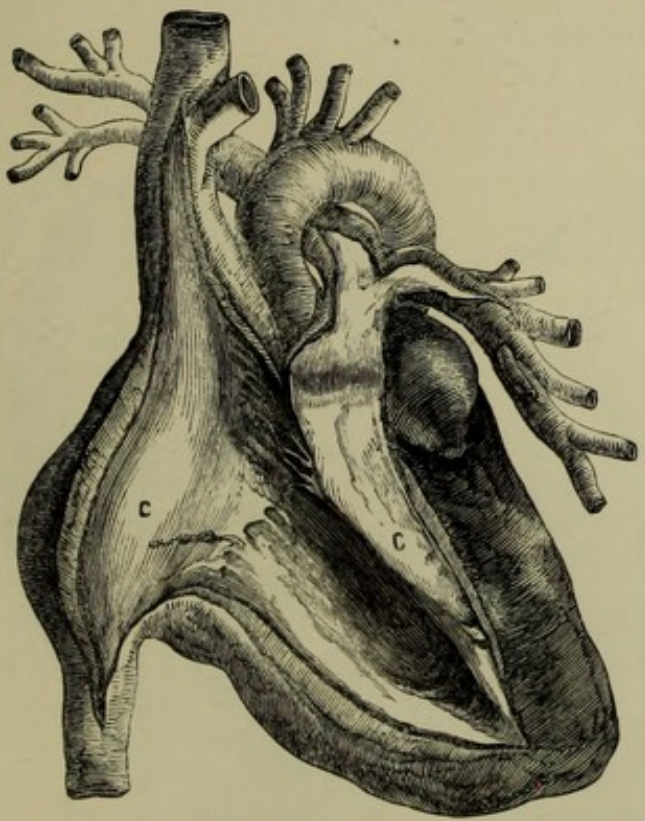


Fig. 3.—Fibrinous Concretion filling the Right Auricle, extending into the Ventricle and Pulmonary Artery, and marked by the Semilunar Valves.
C C. Concretion.

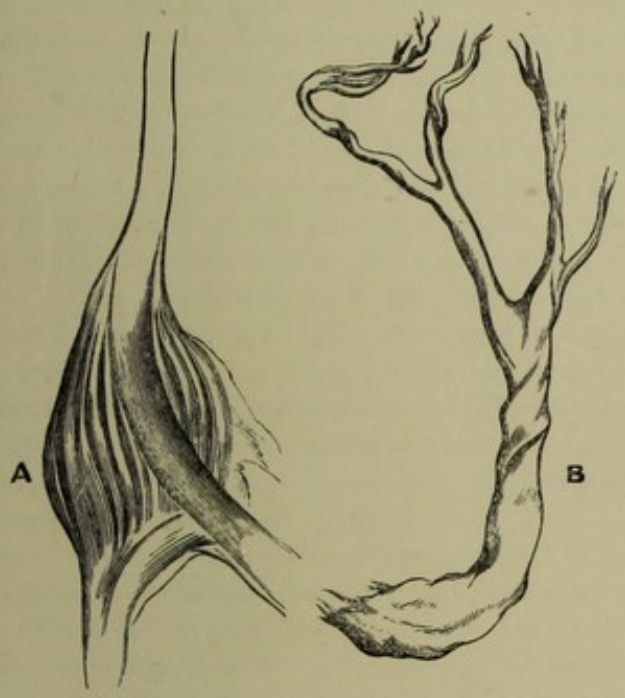


Fig. 4.—A. Fibrinous Deposit with simply curved groove; B. Concretion with spiral groove.

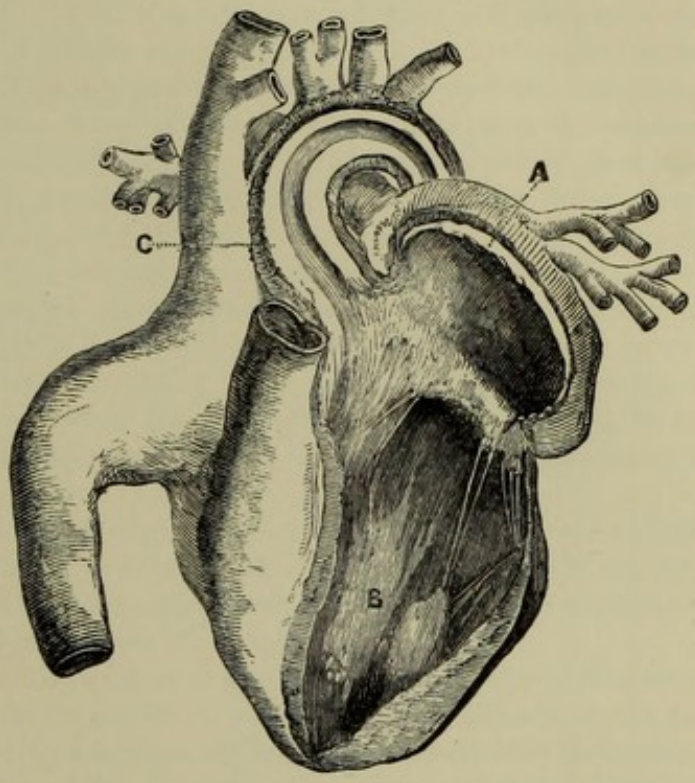


Fig. 5.—Tubular Concretion in the Aorta.

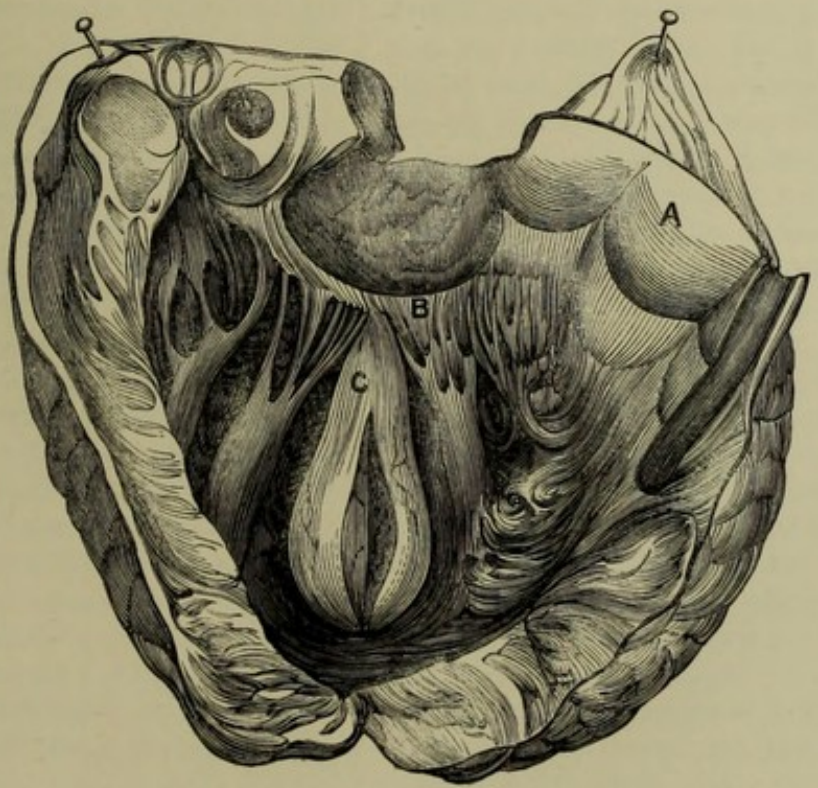


Fig. 6.—Organic Adhesion of a Fibrinous Concretion to the Heart.
A. Portion of Aortic Valves; B. Mitral Valve; C. Concretion.

concretion is bored by a column of red blood. Here the concretion takes the form of a hollow cylinder. (Fig. 5, c.) This modification is rare; and when it occurs, it is found in the commencement of one of the two large trunks, the pulmonary artery or aorta. I have met with it very clearly marked, but chiefly in the aorta. It is worthy of note, too, that in examples yielding tubular concretion, the containing vessel is generally dilated, and roughened on the inner surface from atheromatous deposit. From the rarity of this form of concretion, I infer that a dilated or roughened tube is favourable as a base-work for the deposit. When thus formed, the fibrine is firmly adherent by its circumference to the inner surface of the artery; and the thickness of the fibrine cylinder is due to a series of layers, laid down from the circumference towards the centre. The diameter of the column of red blood in the centre, in all the instances I have seen, has been about that of a goose-quill. The red blood thus situated is in the coagulated state.

This form of concretion gave rise to the myth held by the old writers, that serpents might exist, and were, in fact, to be sometimes found, in the great blood-vessels. In the Library of the Medical Society, there is a quaint paper by an old London Æsculapian, Dr. May, giving a serious account of a serpent which he had removed from the great aorta of a patient who died under his care. That which shocked our historian most was the observation that the creature had an alimentary canal and a distinct anal orifice. He gives even a drawing of the creature, specially indicating the termination of the alimentary tube. The drawing, as we read it in these days, is a very correct representation of a hollow fibrinous tube, with a cord of red blood running through the centre. (See Note 1.)

In one or two instances, I have met with a concretion having conical points, in which, on vertical section, I found a thin cord or film of red coloured substance running centrally and in the line of the concretion. I therefore thought that the tubular character of many concretions might be hidden in some instances by a contraction taking place at the two ends of the concretion. I now believe, however, that this opinion requires to be withdrawn; and herewith I withdraw it. In Fig. 5, I have supplied, from nature, a beautiful illustration of the character of the true tubular concretion. In the case from which the sketch was made, the concretion (c) was laid down in the aorta, that vessel being dilated at its commencement, and having its inner surface much roughened. The concretion, some days in formation, was at last loosened by the force of the ventricular contraction acting on a tube, becoming smaller and smaller in its diameter: suddenly carried away altogether into the narrower part of the artery, it produced almost instantaneous death.

There is yet another form of deposition of the hollow character, in which the fibrine is laid down in the cavities of the heart in such a way as to encircle the inner surface of the cavity, and, by illustration, to cause a new and false endocardial lining. I have attempted to indicate this mode of deposition also in Fig. 5, at points A, B. In these instances, as I have once seen, the deposit may entirely cover the inner surface of an auricle, may be perforated by the entering vessels, and may contain enclosed in it a clot of red coagulum. In other examples, much more frequent in number, the ventricular cavity is found lined in patches (Figure 5, B) with an extremely fine web of fibrine, so thin as to be transparent, and easily looked over if sought for hastily, but easily to be seen and raised with the forceps, if sought for with precision and care.

Lastly, it is to be observed that fibrinous concretions have some point or points of adhesion to the walls of the part in which they are laid down. The adhesions are of two kinds—(*a*) mechanical, (*b*) organic.

a. The mechanical adhesions are produced by an interlacing of the fibrine with the muscular fibres of the heart, or with the tendinous cords extending, in the ventricles, from the columns to the valves. These adhesions are most firm in the auricles, and particularly in the right auricle, where they interlace so intimately with the musculi pectinati that considerable force is often required to separate them. The concretion, indeed, if force be used, will tear off at its base, but will not unwind itself from the muscular fibres. The auricula of the auricle is a spot in which this kind of adhesion is very strongly brought out; but I have seen, also, a very firm attachment in the right ventricle, the base of the fibrine mass being closely interwoven with the muscular structure. It would seem, too, that an adhesion thus existent may last for many months, nay, for years, without any organic connection being produced. My friend Dr. Sayer was so kind as to show me on one occasion a concretion removed from the body of a lady, who for some years had suffered, at intervals, with the symptoms of obstruction of the right side of the heart. She died at last suddenly, in one of these attacks; and, after death, the concretion I refer to was found in the right ventricle, and extending into the pulmonary artery. It could not be separated from its attachment to the ventricle except by being torn across; it was unusually firm in consistence, and had all the characters of a concretion long present in the heart; but there was no evidence of the mass having undergone organisation.

b. The organic adhesion of a concretion to the walls of the heart, or to the valvular appendages, is, in very rare examples, fully marked. In Fig. 6 there is presented an illus-

tration of this nature. Here the concretion assumed a globular shape; it was attached by a small pedicle to the posterior curtain of the mitral valve; on dissecting it down, I found that a small blood-vessel passed through the pedicle, and, indeed, there was a distribution of vessels throughout the whole mass. The patient from whom this was removed was an aged woman, who had long suffered from signs of cardiac disease, indicating obstruction on the left side of the heart. She died suddenly, from the fibrinous mass having been carried forcibly upwards into the infundibulum, and from the arrest in the arterial circulation necessarily resulting from so great an obstruction.

Such, then, are the broad anatomical characters of the fibrinous deposits met with in the heart and great vessels adjoining the heart. I called the first of these varieties a static formation, because it is produced from blood at rest. The remainder, one and all, differ from the static product in that they are the result of a dynamic force; that is to say, they are formed from blood in motion.

II.—Physical and Chemical Characters of Deposits.

From the consideration of the general forms of fibrinous deposits in the heart, it is instructive to turn to the deposits themselves, to remove them, and to examine their minute structure with as much care as can be bestowed upon the inquiry. In such an investigation, we may notice points relating to colour; consistency: structural outline; microscopical appearances; weight; and chemistry.

Colour. The colour of a fibrinous deposit varies from a dull red, approaching closely in tint the red of a voluntary muscle soon after death, to a perfect and pure white. This difference depends entirely upon the relation existing between the amount of hæmatine or colouring matter of the red blood-corpuscle and of separated fibrine. In cases where the separation is very rapid, some amount of coloured corpuscles, varying according to the period of separation, may be entrapped in the fibrine, when the concretion will be more or less red in colour. In other cases, where the fibrine separates very slowly, particle by particle, if I may so say, the concretion will be entirely free of coloured matter, except perhaps on its external surface, where it may have been left in contact with a little remaining red blood; and, divided in any direction, will convey the idea of a pearly looking mass throughout.

Consistence. The consistence of a fibrinous concretion is not invariably the same. In some examples—in cases, for instance, where, as we shall afterwards see, the concretion is the last fact in the course of a fatal disease, the determining fact of the death—the substance of the mass is soft and almost gelatinous in its character; while modeled to the part, and completely filling a cavity or tube, this clot may be so soft as scarcely to be removable without breakage. If it is cut across, it seems as if it were infiltrated with dirty thin fluid; and, placed on the water-bath so as to dry up, it is left as a mere film or thread on the drying plate. This condition of clot is only to be expected in cases where there has been some pre-existent asthenic long continued disease.

There is an opposite state, in which the fibrinous clot is laid down rapidly and in the course of diseases very acute and sthenic, in inflammatory diseases, such as croup and pneumonia. In these, the concretion may be, indeed generally is, so firm that it cannot be removed from any attachment it may have without some tearing either of the fibre of the concretion itself, or of the fibre of the heart. Concretion of this kind is not unlike, in colour and firmness, the coagulated albumen of the egg after hard boiling. From this it differs, nevertheless, in that, when broken across, it is not fractured in a clean clear surface, but in a jagged fibrous form. If a fibre of such a clot be pulled, it will be found to possess a considerable degree of elasticity, and to curl up like the tendril of a vine, something in the way that the elastic or yellow tissue curls when cut across, extended, and then set free. Nothing can be more distinct, in short, than the difference in character between this form of clot and that already named as gelatinous. Both are produced by separation of the same constituent of the blood; it will be surmised, therefore, that this constituent, in the course of disease, undergoes great modification in its nature. The fact is so; and, inasmuch as death may occur at different periods in various diseases, so there may be observed, as the result of an inquiry extending over many cases, shades of consistency in all degrees, from the gelatinous and watery to the firm and fibrous.

Again, a fibrinous concretion may be found in two states. It may be firm and tough on its exterior, and soft, pulpy, or even creamy, in its centre. It was at one time assumed that, in clots having these appearances, the matter present in the centre of the concretion was pus. Mr. Gulliver was the first to point out the error of the said conclusion. He determined the fact that the fluid matter so situated is identical with softened fibrine; and that, when fibrine removed from the body is subjected to a temperature of 96°, it begins to soften in forty hours, and looks to the naked eye like purulent matter, but may be distinguished from that matter by the microscopical characters. He adds—I believe I am using his own words—"the softening of coagulated fibrine is an elementary pathological condition, frequent, distinct from suppuration, and constituting many of

those cases called suppurative phlebitis." I am fully of opinion that Mr. Gulliver, in these remarks, has indicated with great precision an important but simple pathological fact. Concretions containing pulpy matter, like those gelatinous in structure, are found only in cases of the asthenic type—in cases where the fibrine may be said to be diseased or imperfectly developed.

Structural Outline. In a great number of concretions, there is found a peculiarly well marked *laminated* condition of the deposited mass; *i. e.*, when divided, the mass bears the character of having been composed in exact order, in layers laid down one upon the other, either from the centre towards the circumference, or from the circumference towards the centre. When a striated concretion is formed in an auricle or ventricle, and assumes a globular form, the layers are encircling; they are disposed, to use a common simile, like the coats of an onion, but of course, being extremely thin, are not so obvious to the sight. Still the striated character is so well traced in many examples, that the arrangement is to be discerned even by the eye alone. When the striated deposit occurs in the pulmonary artery or aorta, being then laid down as a cylinder, the layers are also circular, running the whole length of the cylinder. The *tubular* concretion is in this way formed of a series of layers, the innermost of which encloses the contained red coagulum.

Whenever in fibrinous deposit, layer upon layer, in the manner described, the red colouring principle of the blood pervades the mass, the appearance presented bears the closest analogy to a portion of voluntary muscular fibre. This analogy is so perfect that, in one case, I knew a gentleman who, on meeting with one of these formations in the pulmonary artery, mistook it at first sight for a portion of muscle. A mistake of this kind is necessarily soon dispelled on close examination; but I point out the analogy as a means of conveying the plainest description of a coloured striated deposit.

Microscopical Characters. The microscopical characters of a fibrinous deposit present but few peculiarities. In clots perfectly free from colour and firm in structure, the microscopical form is that of a closely woven fibrillar network, uniform, or nearly so, in appearance throughout. Interspersed amongst the fibrillæ, one occasionally sees, at considerable distances apart, colourless cells resembling the white blood-cells, and indeed not to be distinguished from them. When the clot is coloured, there are to be seen, in the meshes of the fibrine, red corpuscles also, their amount varying with the depth of the red coloration. The softened matter found in the centre of some concretions presents the appearance of white cells, mixed up with disintegrated fibrils and a considerable quantity of granular substance of a brick-dust shade. In concretions lying in a cavity, and having a *mechanical* attachment, these are the only microscopical characters with which I am conversant.

In the case of a concretion becoming organised, a more complex microscopical field is presented. I have had one opportunity of examining such a clot; and the appearances in that instance were those of an areolar web rich in white cells, and having vessels ramifying in great abundance through every part.

I say that I have seen but one illustration of an organised concretion. This is true; but I had, on a second occasion, brought before me a firm growth, adherent to the wall of the left ventricle of the heart by a broad base, and presenting, as regards position and outline, the idea of a fibrinous concretion having an organised attachment to the muscular wall. But, on examination, I failed to detect any sign of blood-vessel; I found only a very loose network enclosing lymph-like cells; and, as the history of the case gave me no clue bearing towards the view of a deposit of fibrine from the blood at any period, I am left doubtful on the point whether or not this was a form of fibrinous organisation. I should rather be inclined to think that it was not, but was a new growth developed from the endocardial surface.

Weight. The weight of a fibrinous concretion is an important matter; for the weight, ascertained both before and after drying, gives us data by which to form an estimate in discussing the question of the *ante mortem* and *post mortem* origin of concretion. I have paid considerable attention to this question of weight; and the mean result of the labour leads to the fact that there is the greatest possible difference in the actual amount of fibrine in different concretions—a difference which does not in any way depend on the relative size of concretions, but on their consistence, or, in other words, on the amount of water present in the mass.

I once removed a large soft gelatinous concretion, made up almost entirely of water, from the right ventricle and pulmonary artery of a man who died from chronic alcoholic poisoning, and found it, when washed of red blood and externally dried with blotting-paper, to weigh 215 grains. Removing fifteen grains with the scissors, I let the remaining mass of two hundred grains dry at a gentle heat until it ceased to lose weight. The result was a residue barely turning the balance against six grains. The concretion, therefore, in this example, contained, as it existed in the heart, so little as 3 per cent. of fibrine.

In other forms of concretion, the amount of fibrine present is very large. I removed from the right ventricle of a girl, 14 years of age, who died of acute rheumatic fever, a dense con-

cretion, which, washed first with water, and then with ether to take up fatty matter, weighed in the moist state exactly 144 grains. This concretion, dried gently until it ceased to lose weight, yielded not less than 12·5 per cent. of fibrine. In another specimen of concretion, taken from the left ventricle and aorta of a man, a concretion which in the moist state weighed, at the time it was removed from the heart, 170 grains, I found, after perfect drying, a percentage of 11·18 of fibrine. In a third example, a concretion weighing 43 grains, obtained from the auricle of a child that had died from an acute pneumonia, yielded so much as 30·3 per cent. of fibrine. And in a fourth example, where a concretion weighing 27 grains in the moist state was found in the right auricle of an infant, a proportion of 30·2 per cent. of fibrine was yielded. In this last case, the fibrine had been enclosed in the auricle for many days before death, and was unusually firm.

These differences in the amount of water present in a concretion depend on the quality of the fibrine, and on the time elapsing from the commencement of the process of deposit and the termination. As the soft or gelatinous concretion is common to that class of cases where long continued exhaustion marks the end, so, on the contrary, that which retains so much more of solid matter is the product of a disease of the acute inflammatory type, in which the fibrine is in excess in relation to its solvent, and possesses active contractile force. The difference in weight is therefore not remarkable; for, in the last named concretion, the water is made to exude in some measure by the contraction of the mass during its deposition.

Chemistry of Concretions. In so far as I can ascertain, the concretions met with in all cases are pure specimens of ordinary fibrine. According to Mulder and Franz Simon, fibrine of inflammatory blood may be considered in a higher state of oxidation than normal fibrine; and the terms binoxide and tritoxide of protein have been used to express this higher oxidation. The hypothesis of a separate body in the form of protein is now given up; and, in so far as the action of reagents on inflammatory fibrine proves a fact, it proves that this fibrine is the same as common fibrine obtained from healthy blood. Concretions of all kinds, as far as my experiments indicate, are composed of matter soluble in all alkaline solutions, and in acetic, citric, and butyric acids; of matter that may be precipitated from acid solutions by ferrocyanide of potassium; and of matter insoluble in pure water, ether, and alcohol. Butyric acid dissolves the concretion most energetically. The same rules obtain in regard to fibrine in its ordinary condition. I would, however, make one observation in relation to the amount of fat present in certain of these deposits. In concretions formed during acute inflammatory disorders, there is often to be seen on the external surface a distinct coating of an oily nature, which can be removed effectually by ether, and which sometimes is not confined to the surface of the clot, but extends through it. It is true there is always a small amount —about 2 per cent.—of fatty matter in fibrine; but in these examples the normal amount is very considerably exceeded. It may be doubled, or even trebled. The excess seems to me to be due to the same general producing causes as those which determine the increase of fibrine; viz., quickened respiration and circulation, and more active combustion—hypercausis. In cases where concretion occurs during exhausting diseases, there is no such increase of fatty substance.

III.—Order of Construction of a Concretion.

Those concretions which merely coat a mass of red coagulated blood, are produced exactly in the same manner as is the buffy coat on inflammatory blood newly drawn from the body. There is a simple separation taking place slowly, but *en masse*. Hence this form of concretion is not disposed in layers—it is one layer in itself. But in concretions such as were described in figures 2 to 6, the method of construction is essentially different. In these, the first step in development consists in the laying down of a portion of fibrine, in such a manner as to form a fixed point or basis for future deposition. Two special points in the cardiac cavities are favourable to such primary formation. The auricula of the auricle is one such point; the portion of the ventricle immediately below the infundibulum on either side is another; nevertheless, there may be separation commencing at any part of the cavity where, from irregularity of surface, and the presence of muscular bands or tendinous cords, the fibrine can interlace and hold fast. In forms of chronic cardiac or aortic disease, a groundwork for a concretion is offered in a surface of roughened valve or atheromatous artery. Any way, in whatever part it may be situated, or however slight its extent, there is a surface of attachment from which the further development of the deposit takes place; and, unless this fact is recollected, it is impossible to understand the peculiar courses which a concretion may assume. The first particles of concretion laid down at a fixed spot, the progress of its increase is modified according to the time allowed for separation, and the condition of the blood. When the deposition commences in the earlier stages of a sthenic disease, the deposit laid down, particle by particle, during each passage of the laving blood, is arranged under the direction of the blood-currents into one or other of those forms which have been already explained. Taking the auricula of the right auricle as its basis, a concretion will go on gradually forming, and will encroach largely on the cavity without interfering with

the course of the two venous currents. In this way is formed that variety of concretion which, as we have seen, sometimes fills the auricle exclusively, and even distends it. In the acute inflammatory diseases of children, this concretion is frequently formed.

If, again, the concretion take the posterior surface of the auricle as its basis, it will by its steady advancement anteriorly come at last into contact with the venous currents, and will be grooved by them—will be grooved above in a long curved line, *i. e.* in a line directed from the superior cava into the ventricle, and below in a line indicating a sharp curve from the inferior cava into the ventricle.

Or, if the concretion take its origin from two points of attachment in the auricle—from the anterior inner surface as well as the posterior—we then after death discover the double concretion, with the two columns of blood lying between the two deposits, and grooving both.

Once more, the concretion, taking the auricle for its base, may extend into the ventricle, and, following the course of the circulatory channel, may make a rapid sweep into the infundibulum and pulmonary artery on the right side, or the aorta on the left side. Thus continuing, its ramifications may extend into the smaller branches of the pulmonary artery, or far along the aortic curvature.

Concretions arising in either ventricle take their insertion from the ventricular wall, and, almost invariably following the course of the blood-currents, pass into the great arterial trunks leading from the ventricle. The concretion thus running along the main trunk is either tubular in character, or is entirely surrounded by blood, or grooved spirally.

There is yet another condition, in which the deposition, being more slowly carried on and more extended in its character, occurs in the form of a layer, having no one point, but a wide spread surface. By this form of deposition are produced the hollow concretions—those which line a cavity or a vessel, and enclose in their centres red blood.

Lastly, there is that concretion which undergoes *organic* development. In all instances of this nature, there are, in my opinion, preceding changes in the endocardial lining. There are injection of the subserous layer, œdema, exudation of plastic matter, with rupture of surface, and production of a point on which the fibrine of the free blood finds not only a basis, but a basis directly connected with the endocardial membrane. In process of time, vessels from the broken membrane are thrown out, and, extending through the fibrinous matter deposited by the blood, furnish it, if the patient live long enough, with the materials for a new and special organic growth, possessing a permanent and independent existence.

IV.—Deposition before and after Death.

Whether the separation of fibrine from the rest of the blood in the heart, as it is found in many instances after death, is an act *post mortem* or *ante mortem*, is a question, which for more than a century and a half has caused more disputation than almost any other in pathological science.

Harvey seems to have been acquainted with "polypi" (for so the old men characterised fibrinous concretions) in the heart; and Malpighi knew that the white clots found in the heart after death in inflammatory diseases, were the same as that crust which forms on the surface of inflammatory blood. Kirckringius, a writer of no mean repute, opposed the view, that the "polypi" were formed before death. His hypothesis was ably met in 1684, by a countryman of our own, Dr. William Gould, who, in the *Philosophical Transactions* for that year, described fibrinous deposit with remarkable accuracy. He not only denoted the symptoms which attend the progress of deposition, but pointed out the diseases in which deposition is most imminent; and even the further fact, that there may be deposited a form of concretion from which portions of fibrine may be borne away, and, being carried into the extreme parts of the circulation, may plug up the finer vessels, and stop the nutrition of the part beyond. Morgagni was well conversant with concretions of the heart. He also knew their source. The principal and peculiar substance of which they were made, he taught to be the same as that which forms the crust of blood in inflammation These concretions, he adds, are sometimes begun and even completed in the living body. He thought they might commence in cases of protracted syncope. We are now pretty well assured, all the world over, that there is such a thing as deposition of fibrine from the blood in the living body. It is also admitted by those even who see most clearly the full importance of this admission, that there is such a thing as separation of fibrine from the blood after death in certain cases. Let us consider both these positions with candid care.

Firstly, then, we may learn whether a concretion has been formed before or after death by examining the concretion, and by observing its relations to the surrounding blood.

There is one true and unmistakable form of *post mortem* concretion; that, namely, which I have marked first in my list of illustrations. I mean that separation in which the fibrine lies in a thin layer on an under stratum of red coagulated blood. Everything here points to the fact that the fibrine has left the blood while the source from which it came was in a state of rest. No other explanation of this form of concretion is possible.

But, in reference to every other description of concretion which I have given, the evidence is equally clear in support of the view that they, whenever found, are of *ante mortem* date. I do not at present discuss how long before death any one of them may have been produced—that will constitute a subject of inquiry further on; but I say for the moment, that they are produced before death, and that they can only be explained as resulting from blood in motion. Where, for instance, there is a separated clot filling the whole of a cavity, such as the right auricle, it is certain that the clot must have been formed antecedently to death; for the simple reason, that there is no blood left in the cavity from which the clot could have been produced after the final result. When the clot is grooved, we have again demonstrative proof that it was of *ante mortem* date; because the groove, whether curved or spiral, is in the line of a blood-current, and is, in fact, made by the transit of blood, as the bed of a river is made by its current. If the concretion is a hollow cylinder, containing red blood in the tube, the evidence in favour of the origin of the concretion from blood in motion is equally conclusive; for, as the tubular concretion is never met with except when the deposit takes place in a cylinder, as the pulmonary artery or the aorta; as the tubular concretion is never grooved spirally; as the centre of the tube is filled with blood; as these tubular concretions (in every case, at all events, in which I have met with them) show evidence of having been broken away from a basic attachment; and lastly, as they entirely fill the vessel in which they are found,—there can be but one reading of their origin, which is, that they were laid down from their circumference layer by layer; that they formed, in fact, a false and increasing tubular lining to the inner part of the vessel; and that the red blood found in the centre is the last remaining portion left there at the moment of dissolution.

It is but fair, nevertheless, for me to state that a hypothesis has been projected, to the effect that the tubular concretion may be produced after death. Dr. Humphry of Cambridge, in a most able essay on the *Coagulation of the Blood in the Venous System during Life*, assumes that a tendency to separate coagulation of fibrine will produce its effects after death in much the same manner, and in the same position as during life; and that it will thus lead to the formation of fibrinous clots upon and near the valves, and in the interior of the vessels. In this manner, Dr. Humphry further assumes, a tubular lining of fibrine may be laid down in the pulmonary artery after death, and be moulded upon the sigmoid valves, while the remainder of the blood in the vessels is fluid. This remaining blood, subsequently coagulating, would form a soft dark cylindrical clot, inclosed in and continuous with the fibrinous tube.

I am sorry that I am obliged to dissent entirely from this conclusion of Dr. Humphry. I dissent because, from my observations of the coagulation of fibrine in the static condition, it never separates from blood in the manner suggested; it always separates in such way as to crown the blood from which it was produced. True, blood left in the pulmonary artery, blood very rich in fibrine, may yield after death a thin coat of fibrine; but such coat will express but half a cylinder, and will lie on the upper surface, like the buffy coat of inflammatory blood.

Further, the true *ante mortem* fibrinous tube, as I have described it, is not a mere film of fibrine inclosing a large cylinder of red coagulum. Such a separation I have myself never seen. The hollow cylinders I describe are made up almost entirely of fibrine in layers; the red blood, not more than the size of a quill in diameter, and quite incapable of yielding such a surrounding mass, running along the centre. The same observations apply to those forms of separation in which the fibrine lines the whole of a cavity, as the auricle, and incloses a loose red coagulum.

The firm adherence of a concretion to the walls of the heart is another proof of an origin before death. To produce such adhesions, as we have seen already, even though the adhesion be only mechanical, motion is required. The pulsating heart must *churn* out the fibrine; and it is in this act that the firm interlacement takes place. Owing to this fact, we find that, in the separation of fibrine which takes place after death, there is no adhesion of the fibrinous layer to the cardiac surface; yet it is clear that, if separation were the result of mere contact, there would be in these, as in other cases, a firm adherence. If the connexion betwixt a concretion and the heart be organic, however slight the connexion may be, the evidence is of necessity all the stronger, not only of the existence of the concretion prior to, but of an existence *long* prior to, death. This argument is so entirely obvious, that it is unnecessary to push it further.

I have one further word to say on the distinction of fibrinous concretions formed before and after death; this relates to the structure of the deposit itself. If a concretion be laid down in one layer, it may have been formed before death, though it need not necessarily have been so formed. But if a concretion be laid down in distinct layers, such concretion bears its own evidence of having been constructed in layers, step by step, from the blood. Or, if a concretion found in a newly dead body (in a body twenty-four hours dead) be changed in its centre into a soft purulent looking mass, then the proof is carried with the concretion, that the concretion was formed in the living body, and at a period of at least twelve or sixteen hours

before death; for it takes forty hours to produce the softened change in fibrine at a temperature of 98°. I am not sure, indeed, that the change would progress at all at the temperature of the dead body; but, this point waived, certain it is that it would not occur within forty hours after death.

Lastly, some indication of a concretion being formed before death is to be gleaned from the mode in which it is moulded to the parts surrounding it. I agree with Dr. Humphry, that a *post mortem* concretion may bear the impress of that part of the cardiac wall against which it is left in contact; for I have found, by some recent inquiries directed to this point, that if gelatine dissolved in warm water be run into the vena cava, so as to fill the right side of the heart and the pulmonary artery, and if the solution be left to gelatinise, and the heart be then opened—the concretion, for so I may call it, of gelatine will be found bearing a slight impression of all the parts of the heart against which it has lain. At the same time, when the impression made on the concretion is very distinct, indicating that contraction has been made upon the deposit, and when with this modeling there is adhesion, as was the case in the concretion delineated in Fig. 2, then the presence of the impression is corroborative evidence of a separation during life.

To sum up. The proof of a *post mortem* concretion is its position—*on the upper surface of a red coagulum*. The proofs of an *ante mortem* clot are—(*a*) The fact of its filling a cavity; (*b*) the facts of its being grooved externally by a blood-current, or bored by a current through its centre; (*c*) its being firmly adherent to the heart either by mechanical or organic tie; (*d*) its structure being laminated, or its containing in its centre broken up fibrine; (*e*) the fact of its being deeply indented by surrounding structures.

Are there any other proofs wanting in support of the view that deposition of fibrine takes place during life? To some minds, such proofs may be required; and so I proceed to them.

In continuance, then, I remark that the *weight* of a concretion is in many cases proof direct of the *ante mortem* nature of the deposit. The position stands as follows. The amount of fibrine in healthy blood is—I take an extreme point, in order to make the argument fairest—3 parts per 1000. The right side of the heart of an adult man, including auricle and ventricle, is capable, I find, in the relaxed state after death, of holding from 1400 to 1500 grains of blood. To make it contain this quantity, however, it is necessary to tie the inferior cava, to fix a funnel tube into the superior cava, and a straight tube into the pulmonary artery. The organ must then be held with the base upwards, and must be filled through the funnel tube until fluid appears in the tube in the pulmonary artery. The right side of the organ will thus be charged with fluid, and that to an extent rarely if ever met with as a *post mortem* result. The amount of fluid required for this state of fulness will be, as I say, 1400 to 1500 grains; let us accept 1500 grains as the amount. Granting, then, that the heart were left at death thus charged with blood containing the natural amount of fibrine, the fibrine, however it might be coagulated—as a red coagulum, as a white concretion, or as a mixture of the two—could not exceed in weight 4·50 grains.

The greatest increase in the amount of fibrine in disease is up to 12 parts per 1000. I do not myself understand how blood so charged with fibrine could circulate; but, as the statement has once gone forth that such a blood was found, let us take that as the extreme example.

But I have found in the right auricle of the heart of a child, dying of pneumonia, thirteen grains of fibrine, which would represent 4333 grains of blood in health. Admitting, therefore, that the right auricle of this child was capable of holding not less than the auricle of an adult heart, and half as much as the whole of the right side—auricle and ventricle together, I mean—and that after death the auricle was left distended with 750 grains of blood, then it would follow, on the hypothesis that the concretion was formed after death, that the blood left in the heart contained 13 grains of fibrine in 750, or at the rate of 17·3 parts per 1000, which is absurd; for a blood so loaded with fibrine could not circulate at all.

In the above sentences, I have made over to the *post mortem* hypothesis of concretion more by double than any one of its own exponents could claim. For, in the case I have quoted, for illustration, the auricle in reality had a dimension only capable of holding, at full distention, 240 grains of blood, and the auricle and ventricle together but 700 grains. Further, there was no red blood either in the auricle or in the ventricle, and, in fact, nothing save fibrine combined with water; but I have thought it best to extend an absurd limit to the *post mortem* hypothesis, in order the more effectually to show its absurdity. I could labour out the question of weight from at least a dozen more cases from similar data, but the result would be simply a repetition. Any one desiring to follow out the same mode of proof, has only to remove a concretion from the heart of a patient dying at the acme of an inflammatory disease with the symptoms of concretion, as I shall give them in the end, and to proceed as follows:—to weigh the concretion in its moist state; to dry it gently on the water bath till it loses weight no longer; to weigh again, and take the last number as the amount of fibrine. The dimensions of the heart being then calculated, and the amount of blood in the cavity of the heart in which the concretion was found also calculated, the excess

of fibrine over the proportion of 3 per 1000 of blood may be readily ascertained.

It remains for me to adduce one other proof of the fact that fibrinous concretions may be formed during life. This proof, reserved to the last, may be considered the most direct, because it appeals to the visual sense. It is this: that in an animal fibrinous deposit in the heart may be induced—induced by experiment, by what I have elsewhere called the production of disease by synthesis; that the presence of such concretion being diagnosed by symptom, the animal may be made insensible to pain by a volatile narcotic; and that then, the respiration being sustained artificially, and the chest laid open, and the pulsating heart laid bare, the heart may be opened, and the concretion found there present, and removed. I have succeeded in various ways in thus producing concretion: I have succeeded by arresting a large tract of the circulation, as by passing a ligature beneath the trachea, carrying its ends back behind the vertebral column, and by the tourniquet screw cutting off the cerebral circulation. I once narcotised a dog with sulphuretted hydrogen; and, when the animal was entirely insensible, I allowed him to remain in the open air for several hours. He was still insensible at the end of nine hours, but was evidently sinking: I then laid open the heart, and found in the pulmonary artery a distinct fibrinous concretion. [See Note 2.] But the best way of performing this synthetical experiment is to place a carnivorous animal in a closed chamber, and to fill that chamber steadily and constantly with pure and freshly made oxygen. A carnivorous animal so placed will live, I find, for a period of from twelve to thirty-six hours. When the animal begins to fail, or, in other words, to sink, there is manifested in all his movements the signs of obstruction of the heart—signs which will hereafter be more carefully indicated. The sinking fully set (for, before the sinking, there is no concretion, the prostration being the mere result of the obstruction), narcotic vapour is introduced into the chamber, and, insensibility complete, the vivisection is made. I have extracted large concretions from six animals under this condition. The colour of the clot has varied in these cases from red to perfect white. In one instance, a cat being the subject of experiment, the concretion was of pure white, and filled and distended the right auricle, and passed into the ventricle, and passed into the pulmonary artery. In this instance, the sinking symptoms were allowed to go on until death was imminent. Nevertheless, the heart was actively contracting when it was exposed, and continued to contract for nearly twenty minutes after the concretion was removed from its cavities.

I have thus offered, I trust, not a theory, but a complete demonstration, of the fact that fibrinous deposition is an event possible previous to death. I shall have to trace out, in a succeeding chapter, the symptoms indicative of concretion, whereupon the opportunity will recur of adding further proofs to those already supplied.

CHAPTER II.

CONDITIONS FAVOURABLE TO FIBRINOUS DEPOSITION.

FROM points connected with the structure of fibrinous deposits, and the evidences that these deposits are in many instances laid down prior to death, we pass naturally to consider *those conditions of the system which are favourable to deposit*—in other words, the diseases in the course of which deposits are formed. To understand clearly the question here involved, we must consider the modifications to which the fibrine of the blood is subjected under various diseased states, and especially those modifications leading to deposit.

1. In various conditions the fibrine is, or seems to be, diminished in the blood. These conditions are indicated in diseases in which the hæmorrhagic tendency prevails. True typhus, and yellow fever, are diseases of this nature. The effects of many poisons, as the cobra poison, the vegeto-alkaloids, the alkalies, and several organic acids, are manifested in this apparent deficiency of fibrine. In these cases, the blood, as Plato would express it, "is no longer under the natural guardianship of its fibres"; so it exudes into the soft structures, giving rise to petechial spot, black vomit, melæna, hæmaturia, and, in fact, to any form of hæmorrhagic discharge, according to the degree of pathological change.

I have said that, in these cases, the blood seems to be deficient in fibrine. I say *seems* to be deficient, because I wish to guard myself from saying absolutely that the blood is so deficient; and I would impress this qualification earnestly, because, from some late inquiries on the effects of alkalies and alkaloids on blood containing, when drawn, the normal amount of fibrine, the same apparent decrease of fibrine has been produced by the mere addition of these agents in excess to the blood. We need not, therefore, bind ourselves to the idea that, in diseases attended with great fluidity of blood, less fibrine is really produced in the system; we may rather infer that, as all evidence tends to prove, there are conditions in which the blood is surcharged with a body having the power of holding fibrine in solution—of reducing the fibrine, in fact, into a form of albumen. In such condition of blood, whether my explanation of its cause be correct or not, this that I am about to say shall be correct: the blood, while in the condition named, never, either before or after death, yields a fibrinous separation. Ordinarily, it does not coagulate even as red clot, but at most it becomes a thick treacly mass, like newly drawn blood treated with an alkali and afterwards mixed with albumen.

2. There is a second variety of blood, not widely removed from the above named kind, in which there is nevertheless a peculiar tendency to separation of fibrine; and this without any necessary increase of the fibrine itself, or the presence of those systemic states in which the combustion of the body is increased. The older writers were accustomed to notice this separation of fibrine in scurvy. They spoke of the stream of blood in that disease as having two colours, a purple and a white; and of a loose buffy coat forming on such blood. I am convinced to proof that the condition here expressed exists—exists sometimes, as it would seem, by hereditary predisposition; at other times, by acquired disease, especially by disease derived from peculiarities of food. I do not pretend to be master of all the facts connected with this subject, but I will narrate such few as I know. I once attended a child covered from head to foot with purpuric spots—a miserable puny child, cold always, and emaciated. This child died suddenly; and I traced the cause of the sudden death clearly enough to a large concretion of fibrine which had been laid down in the right auricle. I also knew an instance of extreme anæmia terminate in a similar way; the only difference being, that the concretion had formed in the pulmonary artery, and not in the auricle. In a case of purpura at this very time under my observation, there is every disposition to effusion of blood under the skin, and large purpuric blotches are produced from the slightest cause; but, some time after this effusion has occurred, there is a hardening of the matter at the effused point;

and it is observable that the blood of this patient, although it is so easily effused, is not incapable of coagulation when it escapes from the body.

3. There is another condition, in which the fibrine of the blood undergoes *relative* increase. It may be difficult at first to understand what is meant by relative increase. I mean by it, then, a state in which the fibrine is not abnormally increased, nor the system under any condition for the production of an increase of fibrine, but in which, the fibrine remaining normal, other constituents of the blood are below the usual standard. This condition is not uncommon.

When water, the grand menstruum in which the fibrine is held in solution, is reduced in amount, such reduction will favour materially the tendency to deposit. It may seem, at first sight, that this statement runs counter to that which was made above relative to anæmia; but there is no contradiction. Let me explain. Water is, as I have said, the menstruum in which the fibrine is held in solution. But fibrine will not remain in solution in water simply; the water must be the menstruum also for an agent having the power of dissolving the fibrinous constituent. This agent, as we now have proof beyond every kind of dispute except sheer cavil, is an alkali of the volatile series. Now, in order that fibrine should remain in solution, there must be a fixed relationship existing between the menstruum, the solvent, and the substance dissolved. If the substance dissolved is but dissolved, and no more, as fibrine is in blood, precipitation of fibrine can be induced by addition of more water, because, in this addition, we distribute the solvent over a larger surface; we weaken, that is to say, the solvent power of the solution; and the result necessarily is, that the dissolved substance is proportionately precipitated. On the other hand, if from a solution of fibrine, as in blood, we draw off by osmotic action the watery part, together with the solvent, the fibrine is necessarily increased in relation to the portion of water and solvent left behind. In both cases alike, there is, therefore, a tendency to the deposition of fibrine. Out of the body, I can produce in experiment all these phases; and in the bodies of the dead we constantly meet with the results of these changes, if we are observant observers.

The best marked cases in which the fibrine is found separated after death, as the result of loss of fluid from the system, are those where, during life, there has been an exhausting and rapid flux from the alimentary canal. In cases of cholera, the fibrinous deposit so commonly found after death is formed in this way; and the same may occur in other disorders affecting the alimentary system. I have seen them, as so produced, in death from mesenteric disease; and once in a debilitated person, after a fatal purging, induced primarily by self-dosing and overdosing with black draught. Deposition, brought about by loss of fluid, will further happen from rapid elimination through other channels than the bowels, as from the skin in the colliquative sweating of phthisis.

Once more; the fibrine may be relatively increased, not in proportion to the water, and not in proportion to the mass of blood, but in proportion to its own solvent. The old writers were accustomed to state that concretions were the result of what was called languor of the circulation. We cannot adhere to that phraseology at this time of science, because the phraseology is imperfect; but we may, nevertheless, accept that these writers did truly express a partial explanation of a very interesting fact. In many cases of great prostration of the body and of the powers of the circulation, where, notwithstanding, life is slowly destroyed, the blood making languid way for many hours; in such cases (and I may add, in the majority of such cases), the fibrine is left deposited in the heart. The cases themselves include: (*a*) deaths from shock, where the life is not extinguished at once, but in which the act of death has been very considerably prolonged, say for a period of from three to four days; here the fibrine may be separated in a firm and decided mould; (*b*) deaths from the prolonged effect of some poisons, which depress the heart without adding any solvent substance to the blood, such as opium; and (*c*) those instances of disease where death is preceded by great degenerative change, and by slow and certain exhaustion. I have met with *ante mortem* concretions in examples of this nature, after death from mere senile decay, fatty degeneration of the heart, and slow alcoholic poisoning. The concretion, however, in these cases, is often but indifferently developed. It is gelatinous in character, very heavy at first from the presence of water, but containing in reality a very small amount of fibrine. Dr. Gould, whom I have noticed once before, laid great stress on debility as a cause of deposition. He opined —and the opinion has been reiterated by Dr. Meigs of Philadelphia—that, owing to the stasis produced in the blood during a fit of syncope, the commencement of a fibrinous deposition may be traceable, in some instances, to an attack of syncope. Nor is it to be denied that many arguments lend a colour to this view of stasis as a cause of concretion. The observation made by Lancisi respecting the mode in which fibrine is deposited in aneurism, the first point of deposit in the sac of an aneurism being that at which the stasis is most marked, was alone of sufficient importance to establish the stasis hypothesis for a long period; and the more so, because this hypothesis is not opposed to the truth, but is only faulty in that it does not express the whole truth. The position of the question is as follows:—When blood yet fluid is placed entirely

at rest in a closed cavity, and under a pressure equal to that of the circulation, there is no separation of fibrine. On the contrary, blood so enclosed is in the best condition for its fibrine to remain in solution. Here is an argument, then, in direct opposition to the pure stasis view; flatly disproving it, indeed, as a bare hypothesis. Blood, therefore, from which *ante mortem* concretion is formed in the asthenic, is not blood in a state of absolute rest. No; it is blood undergoing slow and languid motion, in process of which the volatile solvent of the fibrine, a product of an active nutrition, is in the first place imperfectly supplied, and, secondly, is eliminated in the expirations of the skin and lungs, to the loss of the blood. The relationship existing between the fibrine and its solvent being thus destroyed, the fibrine is precipitated, and is found in the separated condition after almost all forms of death from asthenia, excepting those where a solvent poison is present as part of the disorder.

4. The last condition favourable—most favourable—to fibrinous separation, is that in which the blood undergoes an absolute increase of fibrine—that condition which the old men were content to designate as disease "attended with sizy blood", and which we moderns, following Franz Simon, call "hyperinosis" or superfibrination. We include under this head a large group of diseases marked by inflammatory symptoms, and always running an acute course; pneumonia; croup; diphtheria; acute rheumatism; erysipelas; inflammatory diseases of all the serous and mucous tracts; the inflammatory reactive fever succeeding on surgical operations; inflammatory lesion in the uterus and peritoneum after parturition; and cerebral congestive excitement.

In thus naming certain local symptoms indicating what we are pleased too often to consider as pure diseases, I write to make the subject intelligible. In my opinion, these local changes are all secondary to one grand systemic derangement, a primary and essential feature of which is increase of fibrine—hyperinosis.

Still farther, I suggest that there are cases in which the hyperinosis is not only preexistent to any local manifestation of disease, but in which it is so marked, and the deposition of fibrine in the heart as a consequence of the increase is so rapid, that death takes place from the obstruction, before any local change is developed. Instances of this nature, while they are not by any means numerous, are, as a general rule, overlooked. There are, nevertheless, to be found in our literature certain extraordinary examples of this nature, worthy of careful notice. One of the very best observers of disease in the last century, Dr. Huxham, in a letter written by him to Dr. Mortimer, describes what was sometimes called an epidemic of cardiac polypi. In this letter, it is narrated that several seamen, brought during cold weather into England from the West Indies, were seized with short coughs without expectoration, continual and violent palpitations of the heart, intermitting trembling pulses, anxiety, and pain and sinking at the heart, so that they could not lie down; they had heavy dead countenances, some pains in the side, but very little fever. Twenty men thus soon died. Two of these men were opened, and in their hearts were found monstrous polypi, tough and adherent to the walls of the ventricle. One of these weighed an ounce. They were on both sides the heart; the largest on the right side.

A still more remarkable illustration of a similar epidemic is described in the *Edinburgh Annals of Medicine* for the year 1800, by another equally good observer, Dr. Chisholm. I have often noticed this valuable paper, but now I will give it almost entire. It is entitled "A Short Account of the Epidemic Polypus in Granada in 1790." The sufferers were negroes, who were subject to peculiar local influences at the time of the seizure. These influences Dr. Chisholm first describes. The foreground was the open sea, with an extensive and bracing beach of sand. On the left was a hill of considerable size and ascent, the bare sides of which, with the reflecting surface of the sea, produced in dry seasons an immense degree of heat. The background was a marsh, extending from the sea to the mountains. From this marsh deleterious vapours were exhaled. Immediately behind the marsh, a ravine began, through which rushed a current of wind of great coolness. The negro houses of the plantation were built on the hill on the left, chiefly on its slope and towards the edge of the marsh. The negroes were, therefore, at once exposed to excessive heat, a cold chilling current of air, and the miasma of the marsh. Their diet was chiefly vegetable food. They had been employed, immediately before the appearance of the disease, in clearing the surface of the marsh, and in holing land for the reception of cane-plants. They were much given to the destructive habit of eating a species of pipeclay very abundant in Granada.

The disease commenced in the end of September, was most prevalent in the latter part of October, and went off in November. The whole number of the sick was about forty, of whom seven died.

The symptoms were, pain at the pit of the stomach and head, and difficult respiration, attended with dry skin, small quick pulse, and slight dry frequent cough. No febrile heat accompanied these symptoms. The surface, on the contrary, was remarkably cool; but there was a heaviness and dulness of the eye, anxious features, and depressed spirits. This state continued for three days; then the pulse became quick, from 120 to 140, and intermitted, attended with a penetrating

pungent heat, which produced a pricking sensation on the hand of the person feeling the pulse.

The disease now became somewhat intermittent, the intermission lasting eight or nine hours. During the paroxysm, the struggle for breath, the aggravation of all the other symptoms, and *the very quick*, interrupted, and evidently visible, as well as audible palpitation of the heart, produced a scene of uncommon horror. The paroxysm was succeeded by a cold clammy sweat, and a state of approaching syncope. The second paroxysm generally put a period to the existence of the patient. The disease was also distinguished, during the latter stage, by an almost constant, disagreeable clammy sweat, overspreading the face, the upper extremities, and the body as low down as the scrobiculus cordis, all below remaining parched in a most remarkable degree. The disease seemed sometimes inclined to terminate by a metastasis. One instance of this was remarkable, wherein a patient, after labouring under all the symptoms peculiar to the disease before the intermittent period, found himself all at once, and without an evident cause, relieved of them; but he perceived at the same instant an excruciating pain a little above the elbow, and nearly about the middle of the thigh. He continued ever afterwards quite free of all the symptoms of the polypus; but they were succeeded by a large abscess in the parts in which he felt the pain. That in the arm disappeared gradually; but the other became so large as to occupy the whole of the under part of the thigh. The cure was effected by passing a seton through the whole length of the tumour; by the use of two dozen of Madeira wine, bark, and a calomel pill with opium, three times a day.

The audibleness of the palpitation of the heart in these cases, Dr. Chisholm remarks, may be considered an exaggeration; but in one instance, particularly, a gentleman (Mr. McSween), to whom the negroes belonged, heard distinctly the palpitation from an adjoining room.

Dr. Chisholm states that, at the onset of the epidemic, he knew not how to act until dissection showed him; and adds, that having no suspicion of the heart being the seat of the malady, he did not examine that organ in the first two bodies opened; but finding the brain and all other organs in a state of health, he opened the heart in the third body, and discovered what he considered might be the cause and seat of the disease. In the right ventricle of the heart he found an enormous polypus, which extended considerably into the pulmonary artery. On extracting it, the body which was contained in the ventricle, measured two inches in breadth. In the fourth body there was a large polypus in the right and left ventricle, besides one in the right auricle. The hearts of the fifth, sixth, and seventh were just similar; and in these five, except one, where the lungs were diseased, no other morbid appearance could be found.

It is interesting to remark, as an aid to our therapeutics, that Dr. Chisholm, after he had discovered what he considered to be the cause of death, viz., the concretions in the heart, changed his plan of treatment. On the very first appearance of symptoms, he took blood, and then freely salivated, with mercury. The results, he adds, were most happy; not one that was salivated died.

I can offer, on my own part, no such a narrative as that given by Dr. Chisholm; but I can very definitely refer to not fewer than six well marked cases in which death, taking place rapidly, with no other foreboding symptoms than a rigor and succeeding slight febrile heat, was traceable by the *post mortem* inquiry to deposit of fibrine in the right side of the heart; the deposit filling the auricle, to the exclusion of blood, and extending, in the course of the circulation, from the auricle into the ventricle and pulmonary artery.

In other instances, as we have seen, the deposit of fibrine is subsequent to the development of one or other of those forms of local mischief to which we assign a specific name. The obvious features of such cases are distinguished by the presence of the local change, but the essential characters are the same in all examples, that is to say, the fibrine is abnormally increased. The increase may extend from the normal two and a half or three parts per thousand, to six, nine, or it has been said to twelve parts per thousand. This latter figure is possibly derived from an error in analysis, but the fact of a great increase is without a shadow of doubt; the result of the increase is also often equally striking. The excess of fibrine, beyond a limited extent, cannot remain in suspension in the blood, and the heart, under such conditions—if so plain but expressive a term be allowed me—*churns* out the fibrine, and becomes the receptacle of the separated mass.

I can give, at this moment, no explanation of the primary cause of increase of fibrine in the cases I have named, except by entering upon the whole question of hypercausis and its origin; which task I would rather avoid, or at least retain. It will be more instructive, at this time, to pass to the subject of the symptoms of concretion, concerning which all is demonstrative.

CHAPTER III.

SYMPTOMS OF FIBRINOUS DEPOSITION:—THE ACUTE TYPE.

THE symptoms indicating the presence of a fibrinous concretion in the heart are modified or obscured by many circumstances, as by the position of the concretion, the rapidity with which it forms, and by the nature of the case in which it occurs. Sometimes the symptoms of concretion stand forward alone; sometimes they are marked by other symptoms; in their course they may be presented in one of three phases:

(*a*) They may be slowly developed throughout.

(*b*) They may be developed slowly in the first instance, and rapidly at last.

(*c*) They may be developed rapidly, appear in a marked degree, and run an almost independent course.

I would further note that the symptoms differ according to the side of the heart on which they are laid down; they naturally divide themselves into two classes—the general and the physical.

I shall pursue, in the narration of symptoms, an order the reverse of that followed in narrating the conditions under which fibrine is deposited; for now we must have the broad symptoms brought out first—those symptoms which are least obscured, purest in their manifestation. We shall find symptoms of this kind well marked in all that class of cases where the fibrine is absolutely increased, the diseases of the acute inflammatory group; we shall find them best marked of all in those examples where the local inflammatory changes are slightly developed, or are not developed at all.

If my own observations are correct, the fibrinous concretions which form during acute inflammatory disorders are, as a general rule, laid down on the right side of the heart. It is true that, in the *post mortem* examinations of such cases, small concretions will generally be found on the left side, the cavities of which are empty of all else; but the large concretion which has given rise to the symptoms will be found either in the right auricle, the right ventricle, or the pulmonary artery. The general symptoms arising from a concretion in these right cavities of the heart are such as might be anticipated by the physiologist who could devise means for checking the course of the blood through the inferior and superior cavæ. The supply of blood through the lungs toward the systemic side of the circulation is impeded; the chemical changes of respiration are reduced; and all the functions which depend for their manifestation on a due supply of oxygenated blood are disturbed, and in a greater or lesser degree suspended. The symptoms, however, are those of syncope, not asphyxia; *i. e.*, the leading processes of life are checked from mere absence of arterial blood, not from the presence of blood incapable of building up the tissues, and of endowing them and the brain with the essentials of the living forces. The symptoms, indeed, are analogous to those which arise from the introduction into the veins of air, oil, or other material capable of so modifying the course of the blood as to prevent it from making the pulmonic circuit. The symptoms are, that the body gradually becomes cold, and of livid paleness, the coldness commencing first in the extreme parts; the beat of the arteries is irregular and subdued; the brain, being deprived of its blood, the mind wanders; the muscles, robbed of the same support, sink into restless prostration; sensation is in a great measure lost; the respiratory movements are irregular, though often persistent in action for a few moments after the heart has actually stopped; and the veins of the body, being charged with blood, are sometimes markedly prominent; while a tinge of blueness, arising from their congestion, is given to the more vascular parts, such as the lips or central part of the cheeks.

But there is one other symptom which beyond all these is striking, and I must refer to it particularly, because it is not only striking, but misleading. This symptom is a peculiar *dyspnœa*. The dyspnœa occurs not because the movements of the thorax are sensibly impeded; not because the entrance

of air to the lungs is sensibly obstructed, for the murmur of respiration is audible enough, but for the reason that the supply of blood through the pulmonary artery is diminished. This form of dyspnœa is one common indeed to other modes of obstruction commencing in the heart; but in the cases I now name, it is most thoroughly apparent. It is the most distressing of all the forms of dyspnœa with which I am conversant. The asthmatic patient can find some relief in a certain position of the thorax; the phthisical patient can obtain relief on lying down in perfect repose; but the patient with this obstruction gains no moment of tolerance. He feels that in breathing, heaving, and sighing, there is no result; none of that steady continuous support which marks the natural life. You ask him where the oppression he feels is concentrated, and if he is conscious, he points to the heart. If he can speak, he tells you that he can fill his chest, that he has no pain in the strict sense of that term, but an indescribable restless, faint, sickening, deathly oppression. The very approach of your own body bending over the sufferer is insupportable. Whoever has suffered from loss of blood, whoever has carefully observed the symptoms incident to slow hæmorrhage, has felt or observed in a minor degree the symptoms I would describe.

The dyspnœa too, to the external observation, is special. The acts of respiration are rapid and yet deep. The breathing is mainly by the diaphragm, and the alæ of the nose undergo that "working" which all practitioners recognise as an unfavourable sign in acute diseases. The dyspnœa lasts to the end; after the mind ceases its function; after the body has become as marble; after the restlessness of body has ceased from sheer exhaustion; even some seconds after the heart has ceased to beat, the chest will heave, and the nostril play.

Such are the general symptoms, I say, of well marked concretions occurring on the right side, in the right auricle or ventricle, or both. But what are the special or physical signs? I would state at once, that in relation to the physics of the heart, there is no definite sign indicating the presence of concretion on the right side. The fibrinous mass may certainly interfere with the valvular machinery of the heart; the curtains of the tricuspid valves may be bound down to the cardiac wall, or looped up together by a transverse intertwining of fibrine threads with the tendinous cords of the valves; or the pulmonary semilunar valves may be overlaid with fibrinous deposit; but the conditions here are not such as to produce murmur; they are only such as reduce or destroy the normal sounds. And, as the valvular mechanism on the left side is free, and is capable of producing the two sounds in their natural time and place, so the sounds are at most but modified in tone. I have in a few cases observed an irregular action of the heart; but this has been so rare, that I do not attach to it any special importance. Turning from the heart to the lungs, we find, however, in some instances, some physical facts on which to confirm a diagnosis of concretion based on general symptoms. In children, especially where during the existence of an acute inflammatory attack fibrine is laid down, there are presented the most striking indications of emphysema of the lung. In infants, this emphysema is sufficient to raise up the thoracic wall anteriorly into a rounded prominent projection. If you examine this chest with the stethoscope, you may find at no part sign of congestion nor of condensation. When this state obtains (and it often does obtain), the evidence is almost demonstrative as to the presence of concretion. The emphysema is a consequence indeed, of the concretion. It is due, in this case, not to obstruction in some part of the respiratory tract, but to the deficiency of blood in the pulmonic circulation.

The presence of blood in the pulmonary tissue in the capillaries covering the air-vesicles, is essential, in fact, to the integrity of the vesicles. We may take a narcotised dog, lay open his chest, keep up his breathing artificially by the double acting bellows, and see the heart beat and the blood make its circuit for an hour or more; there will be no emphysema of lung under moderate and measured force with the bellows, so long as the blood continues to make way. But if we put a check on the blood-current going to the lungs, although the same force be employed with the bellows, the air will infiltrate the pulmonary tissue, there will be emphysema. I make this observation from direct experiment; it is one of value to remember in disease in general, and of great value in this particular form of disease. At the same time, it is not invariably the case that emphysema accompanies fibrinous deposit. I am inclined, indeed, to the opinion that the emphysematous condition is most frequent in children. In adults the heart has a greater propelling power; and the lungs, charged with a small current of blood, give, on physical examination, indications of congestion rather than of emphysema; in persons of adult life, this is the more common sequence.

In coming to a diagnosis, then, regarding concretion on the right side of the heart, we must carefully consider together the general and physical indications. The general indications are coldness of surface, pallor, prostration, restlessness, enfeebled pulse, and the peculiar dyspnœa, of which I have spoken at length. The physical symptoms, as regards the heart, are irregular or weak tumultuous action, with feeble sounds; as regards the lungs, emphysema or congestion. In the end, in the cases presenting these symptoms, the death is

usually very easy; a syncopic anæsthesia comes on, and the watching friends tell you that they were scarcely conscious when the sleep of life became the sleep of death.

The class of symptoms I have thus described marks peculiarly that condition of body in which the fibrine is in absolute excess—hyperinosis. In any one of the diseases which may be ranged under that head, we may have these signs manifested. They may occur before any decided local inflammation has been discovered, or at any time during the period of a local inflammation; in a word, there can never occur that systemic condition on which idiopathic inflammation is based, without some danger of deposition. It is true that in the majority of cases such risk is small, but it is always present; and what is more, the risk or tendency to deposition cannot be measured by the local signs of inflammatory mischief in any given case, nor yet by the symptoms of excitement which accompany the local.

I have stated, in reference to these symptoms, that they may last for different lengths of time. This is the case. I have seen them developed almost in an instant, and run their irresistible course to the death in the period of a few minutes. I have seen them develope slowly, and extend over two or three days; once over as many weeks. These are the extremes on both sides.

The causes of these differences are rather accidental than otherwise. They depend on the manner in which the concretion is laid down, the extent of the obstruction which it throws in the way of the blood, and its position in the heart. When the symptoms have been ushered in suddenly, and have caused death in a few minutes, the event has usually transpired as follows:—The concretion has been laid down at the root or in the body of the pulmonary artery, either in the form of a hollow cylinder, shaped like the tube whose walls it surrounds, of which, indeed, it is a plastic cast; or as a plug or solid cylinder. The deposit may retain for a time a hold on the cardiac wall, so that the blood can float either through it, or around it, at first with but slight impediment. But, unfortunately, three evils are by this time set up: first, the blood-current is impeded; secondly, the surface of the concretion, the inner surface if the fibrine is laid down as a tube—the outer surface if as a solid cylinder—form favourable points for further deposit; and thirdly, as the size of the channel by which the blood is transmitted diminishes, the column of blood passing onwards is rendered proportionately slower. All these events lead to two grand results—narrowing of the blood-channel, and resistance to the blood-current. Occasionally, this process goes on until the passage for the blood is gradually reduced to small dimensions, and the blood circulating through it is cut off slowly, so that symptoms prolonged in character are presented. But, in other examples, the deposit, as the pressure upon it from behind increases, breaks from its attachment at its base, is carried upwards by the force of the blood-current into the narrower part of the vessel, and, completely obstructing the life-stream, leads to inevitable and almost immediate death.

The symptoms of concretion on the *right* side of the heart, in their unmixed character, are sufficiently clear. I know of none more submissive to calm and considerate diagnostic skill. It is to be admitted, at the same time, that when complicated with local symptoms, these symptoms being developed in an important organ, as the lung, the diagnosis is much more obscure. In such a disease as croup, this embarrassment is markedly brought out; for the mind has to balance between those symptoms which are due to the withholding of air from the lungs, and those which are incident to suppression of blood. In such cases, physical examination best avails. If, then, such examination shows that there is distinct and persistent obstruction to the passage of air along the windpipe; if the lungs give evidence of congestion; if the action of the heart be free and regular,—the inference to be drawn is that, whatever may be in the heart, the leading indications point to the windpipe as the seat of the most important change: and this inference will be doubly strengthened if, in addition to the physical demonstrations above suggested, there be spasmodic dyspnœa instead of gasping dyspnœa; blueness or bronzed state of the skin, instead of ashy paleness; and convulsion, violent convulsion, instead of restless agitation and tremor.

Symptoms of the class here named may entirely mask those of concretion; and such masking may be present in any form of inflammatory affection of the organs of respiration; or the two series of symptoms—those, I mean, of inflammatory lesion of the air-passages, and those of concretion—may be blended, the one or the other prevailing, by the accident I had almost said, of prior development. I have thus found concretion present in cases of pulmonary inflammation, where I have not been perfectly certain of the existence of the concretion during the life of the patient.

But in examples where there is a blending of local symptoms with those of concretion, there occurs occasionally another state well deserving of note in all inflammatory affections of the lungs or of other organs. It sometimes happens that symptoms of local inflammation of the most serious kind are delusively relieved by the occurrence of fibrinous deposition, to be replaced by the symptoms of concretion themselves, in all their dangerous significance. The reason of this reduction of the acute inflammatory mischief, as

the result of the deposition, is simple enough; the deposited fibrine is cutting off the blood from all parts of the body, and therefore from the inflamed part. The active local mischief is therewith subdued: the general inflammatory fever, so called, is therewith subdued. The ancients, without knowing the cause, knew about these signs and changes, and symbolised them in their notions of critical days and sudden recoveries, and sudden deaths following upon sudden recoveries. There is much truth unexplained in old physic. Every practitioner in large practice will recall these cases, and may now connect them with a cause. He has had sometime a patient suffering from an acute inflammatory attack. The local mischief may have been pneumonia, bronchitis, erysipelas, or others of the inflammatory group. This local change may have been slight, or it may have been severe; but suddenly it has ceased; there has been, perchance, a temporary relief of acute symptoms, too sudden in its appearance to be satisfactory in its promises: it has been succeeded by all those signs which I have portrayed, and which are epitomised in one sentence, "the sinking state".

I will sketch out two examples. On the 6th September of the year 1853, I was asked to visit a child three years of age, who was supposed to be suffering from croup. I found, on examination, that this supposition was correct, and that there existed laryngeal obstruction in a marked degree; there was croupy cough; the pulse was rapid and full. On the 7th, the child was not worse; the breathing, indeed, was rather more free. On the morning of the 8th, the father of the patient came for me early, saying that his child had become much worse; the croup had ceased; but he thought the heart must be affected, as the throbbing quite raised the bedclothes. I went immediately; and there the fatal symptoms certainly were presented. Consciousness was lost; the pupil was dilated; the body was universally pale and very cold; the breathing was short and gasping, but free; the heart was beating violently against the chest; the face was extremely anxious; the limbs were restless to the last degree. At one in the day, the little patient was lying flat on his back, much more quiet, with the circulation irregular. At two, life was extinct. There was no difficulty of diagnosis here. The inflammation had been supplanted by deposition, and threatened asphyxia had given place to inevitable syncope. At the *post mortem* examination, the laryngeal, tracheal, and upper bronchial membranes were found injected and coated with a thin semitransparent secretion; but patent, and narrowed by no fibrinous layer. The lungs were inflated, perfectly pale and exsanguineous; they floated readily in water. The pleural membranes were normal. The right auricle was distended, and had a dark, deeply congested appearance. On opening it, it was found to be completely engorged with a firm fibrinous concretion, extending by a prolongation through the ventricle into the pulmonary artery.

Here, then, is a case exemplifying what I have said. Previously to the visible development of the sinking symptoms, the pulmonic derangement seemed to be relieved. The pulmonic symptoms *were* relieved; for, at the time when the breathing became easier, a gentle check was being put on the pulmonic circulation by the concretion: the local mischief progressing in the lungs was thus arrested as by a venesection, to be replaced, however, by symptoms virtually analogous to the symptoms of hæmorrhage.

There is another modification of relationship between the symptoms of local inflammation and those of concretion, in which the local inflammatory symptoms again become subdued, to be followed by the signs of concretion, but by these in a prolonged and less acute form. The following history shows better than a bare summary of facts the kind of case to which reference is now made.

A child, fourteen months old, came under my care on December 6th, 1852, suffering from bronchitis. He was treated with antimony. On the third day after my first visit, the substance of both lungs became affected, and pneumonic crepitus could be heard at the base of each, especially on the posterior part. On the 10th, the breathing was relieved; and the crepitus was less distinct, and had not extended. On the following day, the body of the child became universally pale; the limbs were restless, and the breathing short; the signs of exhaustion were more manifest; antiphlogistic treatment was stopped, and was replaced by doses of the decoction of cinchona, with citrate of ammonia. On the 12th, there was no improvement, and the bowels were inclined to be disturbed. The bark and ammonia were continued. On the 13th, the bowel derangement continued; the pulse was irregular; the feet were œdematous; and the motion of the heart was tumultuous. Chalk powder was added, with mucilage, to the bark mixture. On the 14th, the symptoms were nearly the same; but the hands were now also œdematous. The same treatment, and a full supply of nutritious food, were continued. On the 15th and 16th, the exhaustion became still more marked, and the whole body was œdematous. The urine, however, showed no evidence of albumen, but was loaded with lithates. The surface of the body was peculiarly cold; consciousness failed; and the restlessness continued. During the whole period, the lungs were carefully examined; and, although the respiratory murmur was feeble in parts, there was evidently sufficient lung in play to support life, and there was not in the general symptoms the slightest evidence of imperfect

respiration or asphyxia. On the 17th, potassio-tartrate of iron was ordered; but, in a few hours, death put an end to all treatment. At the examination, twenty-four hours after death, the body was unusually rigid. The brain and its membranes were carefully examined, and no sign of disease was found in them; but in the longitudinal sinuses two threads of fibrine were detected. There was no effusion into the pleural cavity. The lungs in parts presented a white blotchy appearance; the bronchial membrane was healthy; the lung-substance was œdematous. There was no purulent deposit in either lung. The pericardium was natural. The right auricle of the heart was dark and distended. It contained two distinct fibrinous concretions; the one was firmly adherent to the cardiac walls at the upper and right corner of the auricle; the other was attached to the left wall; but they were both so placed that they did not altogether arrest the current of blood passing through the heart, though they must have impeded it materially. On the left wall of the pulmonary artery, at its root, there was spread out a thin layer of fibrine; so thin, indeed, and so beautifully adapted to the cardiac structures, that it would have escaped attention altogether, had it not been accidentally raised with the forceps. The concretions in the auricle were firm, and completely freed from blood-corpuscles; they filled up three-fourths of the distended cavity. A very small concretion adhered to the upper part of the left auricle, and a thin filament ran along the aorta. The spleen was very much congested, as were also the other abdominal organs; but no trace of organic change could be detected.

This case, which I watched with intense interest, exhibited symptoms which were to me very peculiar during life, though sufficiently obvious after death. On the day when the first symptoms of exhaustion came on, the pallor of the body and the tumultuous action of the heart led me at once to diagnose a concretion in the right cavities of the heart; but whether it was possible in any case for a concretion to form in such a manner as to obstruct partially the blood passing through the heart, and so lead to congestion of the abdominal viscera, to serous purging, and to general œdema of the body, was a question I could not answer, from want of a precedent. The inspection of this case offers that precedent, and confirms in every way the view that, when fibrinous deposits are formed in the acute stage of the inflammatory process, the local and general inflammatory action may be suspended; and, further, that fibrinous deposits in the heart, not sufficiently large to obstruct the circulation immediately and cause rapid death by sinking, may obstruct the blood-current to such an extent as to give rise to œdema, and in the end to induce death in a manner similar to that of death from valvular obstruction.

In the preceding observations on symptoms, we have observed the nature of the symptoms of concretion in cases either where there has been no manifestation of local inflammation, or where the specific signs of local inflammation have been superseded by those which mark deposit. To these observations I would add, that there are a class of cases in which local inflammatory changes precede the processes of deposition, but are themselves so obscure or so slight that they are passed over, or are treated as of too light a character to receive particular attention. I will narrate as briefly as possible, from observed cases, two illustrations.

A man of forty years, a healthy man previously, was taken with what he called "a cold". He next complained of a dull heavy pain in the back, and of a general languor. He had a pulse at 80, and a dry tongue. This condition lasted fourteen or fifteen days; it did not altogether confine the man to his house. Suddenly he became worse. He felt no acute pain; but his countenance was expressive of great anxiety; his breathing was hurried; his pulse was so quick and so irregular, that its time and force could not be estimated; the action of his heart was tumultuous; the secretions remained natural. These signs lasted forty-eight hours; then the lungs began to show evidence of congestion; and this congestion, gradually increasing, was sustained for a succeeding day and a half. The patient had occasion to rise from bed: in returning, he sank on the bed; he was dead.

The *post mortem* examination here revealed the immediate cause of death, in the presence of a large, firm, adherent, fibrinous concretion in the right ventricle, extending into the pulmonary artery. It also revealed more: it explained that there had been long pre-existent inflammatory change; that there had been splenitis, for the spleen was entirely disorganised from purulent exudation.

A lady was seized with cold; she had slight shivering, succeeded by febrile excitement. The following day there was the merest blush of erysipelas on the ear. The redness progressed a little that day, but was considered too slight to cause alarm. Some fifty hours after the appearance of the local change, this lady felt an uneasiness, oppression, faintness, and dyspnœa, "creeping"—as she expressed it—over her. A stimulant was given, but it was of no avail; from sitting up in her room, she retired to bed, and in six hours she was sinking. She was sinking, according to an explanation which I readily excuse, from suppressed erysipelas; and, indeed, the disappearance of the redness from the ear, as the supply of blood was cut off, favoured so crude a notion strongly. She was sinking, according to fact, from the formation of concretion in the right side of her heart. To relieve the dyspnœa, opium was given, which

served truly to soften the agony, but to clench the result. Death occurred within twelve hours from the date of the sinking indications, and the *post mortem* examination revealed the concretion on the right side: a large concretion, filling the auricle, prolonged into the ventricle, and curved upwards into the pulmonary current.

Am I not writing the experience of all practitioners in the above cases? Except that the local mischief commenced on the mucous surface of the air-passages, instead of the skin, the case of the Emperor Nicholas, late czar of all the Russias, was of the kind I have now told. The armies of two great nations, at a great crisis, rolled against him harmlessly; so simple a physical act as the deposit of a poor scruple or so of fibrine from his own royal blood, dethroned him. We who comprehend these truths of nature are the kings of the earth after all. We take into our considerations the destinies of everything that lives.

When a fibrinous concretion is deposited on the *left* side of the heart, the ventricle, the infundibulum, and the ascending portion of the aorta, are the most common positions. The general symptoms which characterise the presence of concretion here situated are different in many respects from the preceding. There is suffocative dyspnœa, with expectoration of mucus sometimes mixed with blood; the surface of the body is of a leaden colour, and the body is cold. The muscular perturbation lapses into powerful convulsions, and coma precedes dissolution. These symptoms may extend over many hours; or, as in the preceding class of cases, they may also occur in a sudden manner. The patient, in moving or making a straining effort, may suddenly fall back, may be seized with a violent convulsive fit, and so expire.

I once saw these symptoms and this sudden form of death in an old lady who had previously suffered from no other ailment than a slight attack of cold. In rising from bed, she fell back, and died convulsed, before medical assistance could be obtained. In this case, the concretion had formed as a hollow cylinder in the infundibulum of the left ventricle, had become dislodged, and had been carried into the aorta, which it entirely occluded.

The physical signs of concretion on the left side, in so far as the heart is concerned, are not materially different in reference to the systolic and diastolic sounds from those which attend deposition on the right side. For, if the right side of the heart is free, the two sets of valves on that side are all sufficient to produce the two sounds; so that a mere reduction in the intensity, or rather the fulness, of the normal sounds, is the only probable modification. There is, nevertheless, a distinction in the action of the heart. When the concretion is on the left side, the action of the heart is much more violent, irregular, and tumultuous. There is also a difference in regard to the physical signs of disease in the lungs; the lungs are never emphysematous; they are always congested, the congestion being most decided and extensive. Corresponding with this condition, the dyspnœa is not the syncopic dyspnœa which we observed in the previous cases, but a dyspnœa rather of the pneumonic type, laboured, but free from gasping; the oppression veritably is in the lungs, and is so expressed by the patient.

Death is less easy than in the cases where the concretion lies on the right side, or, at all events, is not so easy to appearance. There is coma, but therewith there is struggling, violent convulsive struggling, to the end. Death takes place often in the convulsion occurring by a mixture of syncope and asphyxia.

There are yet another series of cases in which concretion occurs on *both sides* of the heart at the same time. In these examples, it is to be observed that the signs are, generally speaking, those of concretion on the right side. If there is any distinctive sign of double clot, it is in cases where the valvular mechanism is interfered with by deposit on the two corresponding sets of valves, the ventricular or aortic. When this is the case, the systolic or the diastolic sound may be lost altogether, according to the sets of valves obstructed in their play by the deposition upon them.

CHAPTER IV.

SYMPTOMS OF FIBRINOUS DEPOSITION:—THE ACUTE TYPE (CONTINUED).

IN addition to those cases of fibrinous deposition in the heart, noticed in the preceding chapter, where the fact of deposition is apparently the result of inflammatory hyperinosis, there are other cases in which the hyperinosis exists, and in which death occurs from the deposit of fibrine in the central organ of the circulation without the exhibition of true inflammatory manifestations. The pregnant and puerperal states are of this nature; and various and comparatively frequent instances occur in which the puerperal woman, who has passed, perchance, through the most natural labour, and has had no subsequent sign of mischief nor of bad omen, is stricken in a few minutes to the death by a sudden arrest of the circulation, commencing at the heart. It would seem indeed that in the puerperal state the fibrine of the blood is normally increased; or, perhaps it were more correct to say, is physiologically increased. This increase after labour may be due to the changes taking place in the uterus; to the removal by absorption of the hypertrophied uterine muscles, or it may be connected with the lactating process: any way, it exists, and it must be received as a fact of great consequence, not so much from its physiological as from its pathological meaning.

In the puerperal state, deposition may take place before or at a somewhat advanced period after confinement. Taking it all in all, however, I am inclined to think that it is rare as an occurrence before the third day after labour. The symptoms are usually insidious in kind, and may in different cases be of the right or the left side of the heart. The puerperal state stands indeed in the category of a pure sthenic inflammation, *minus* the signs of local inflammation. In this state, the symptoms of concretion are often as insidious as they are sudden. I do not here speak of puerperal phlebitis, with deposits in veins; but of cases where the deposition is directly in the heart and elsewhere not; where, before or after the parturient act, there has been no untoward sign, but where the patient suddenly and unexpectedly succumbs, without any preliminary indication of acute disorder.

Dr. Edward Smith has given the following graphic history of a puerperal case in which the deposit was in the right ventricle, the death being due to sudden occlusion of the pulmonary artery:—

"On Sunday evening, April 24," writes Dr. Smith, "I was urgently summoned to see a lady who was reported to be delirious. On arriving at the house, I found that she was dead, and had been so fully twenty minutes. She was a patient of Mr. Bartlett and Dr. Jackson, of Notting-hill, and was altogether unknown to me. I found that she was about 20 years of age, a little above the middle size, well developed, and in good condition, and within a few days of the term of utero-gestation of a second child. She had been perfectly well until within ten minutes of her death, except that she had complained of some pain and tenderness on the inner side of the left thigh, and, to relieve this, had been directed to lie in a recumbent position. She had eaten a very hearty dinner at three P.M., and tea at six P.M., and was full of spirits throughout the day, and up to nearly eight P.M. She had worn the stays used by pregnant ladies, even when lying upon the bed, contrary to the directions of her medical adviser; and it is probable that they were well laced. The child was known to be alive on Saturday evening, but nothing could be learned as to its vitality on Sunday. While lying upon the bed, dressed, and with her stays on, and in excellent spirits, she suddenly uttered a shriek, and flung her arms about wildly, and cried, 'Oh, my head! I cannot breathe! I am going mad!' and also, 'Give me my breath!' This continued for about five minutes, during which time her hand was placed upon her chest; and then she became calm for a moment, and said to her husband, 'There, Charles, I am better,' and expired. The face was deeply livid, and the body bent, so that the chin approached her knees, When I saw her, the face was blanched, and she lay stretched

on the bed. Having learned several of these particulars within a few minutes after my arrival, I became anxious as to the propriety of performing the Cæsarean section, to save the child; but, since so long a period had already elapsed after the death of the mother, since I had neither stethoscope nor scalpel with me, having been summoned from church; since, moreover, I knew nothing of the case previously, and could not fully persuade the husband and friends of the reality of their loss, I determined not to perform it. By the kindness of Mr. Bartlett, I had the opportunity of assisting Dr. Jackson and himself at the *post mortem* examination, forty hours after death, and of making the requisite microscopic investigation of the tissues. The features had lost somewhat of their pallor, and a fluid, very slightly sanious, was exuding from the mouth and nostrils. The under part of the body, as it lay on the table, was not only greatly congested, but presented many well-marked, purplish-black petechiæ. The left leg was not swollen or inflamed. The blood was black and fluid universally, except in the pulmonary veins, where the whole tube was filled by a cylinder of coagulum, having a central clot of blood, enclosed by two layers of condensed fibrine, the outer one of which was colourless, and the whole so firm in texture, that it could be handled and pressed with impunity. It was not strongly adherent to the lining membrane of the vein. The number of white corpuscles was considerably beyond the normal standard. The heart was flaccid, and rather enlarged on the right side. The tissue was undergoing the process of granular degeneration, or the first step of the process of fatty degeneration, and more particularly on the right side. The left side was empty—without coagula even. The right ventricle contained, and the right auricle was distended with, fluid, black blood. The valves were healthy. The arteries were preternaturally small, so much so that the aorta at its bifurcation could not admit the end of a small little-finger, and the capacity of the external iliac was not greater than that of a swan's quill. Neither blood nor coagula were found within any of them, nor were any of them ruptured. The veins were immensely and universally distended, and appeared to be as much larger as the arteries were smaller than the natural size. The inferior cava was fully an inch and a quarter in diameter. The most remarkable enlargement, however, was in the ovarian veins; but whether this enlargement was greater than is usual at the full term of utero-gestation, before labour has commenced, I cannot tell. They were about twelve inches in length, by three-quarters of an inch in breadth, and passed in a curved direction from the ovarian plexus in the broad ligaments, along the iliac fossæ, to the front of the vena cava on the right, and to the renal vein on the left side. The left was the larger of the two. The right one had thinner coats, so that the dark blood within it was more evident, and terminated by an opening so constricted, that a crow-quill could scarcely be introduced into the vessel from the vena cava. There was a bulging of the vessel directly on the side of the vena cava, viz., close to the constricted opening into the cava; and the trunk of both vessels was of even diameter throughout. A careful examination showed that the inner coat of these veins had not given way. The stomach and intestines were enormously distended with flatus, and contained fæcal and partially-digested matter. There was no odour of hydrocyanic acid. The uterus was normally developed and entire, but its parietes were flaccid. The placenta was very readily detached, and was bloodless, and had not undergone the degenerative process. The membranes were unbroken, and the os uteri perfectly closed. The child (a male) was somewhat small, and the cuticle peeled from the subjacent parts on very slight pressure; but there were no other signs of commencing decomposition. The ovaries were healthy. The diaphragm was pushed upwards to the level of the fourth or fifth rib, thus greatly diminishing the capacity of the thorax. The lungs were much collapsed, and crepitus on pressure was but slight. Numerous bubbles of extravasated air were scattered over the surface, directly under the visceral layer of the pleura, and more particularly on the left lung, towards the base. The discoloration on the posterior and inferior aspects was much greater than is usually met with as a *post mortem* occurrence. The tissue was somewhat readily broken up on pressure, but no rupture of the structure was evident. It contained very many granular corpuscles; but, since the blood was fluid, with no appearance of pus, and contained, in other parts, an unusual quantity of white corpuscles, it is probable that these cells were not exudation cells, but the white corpuscles of the blood. The pleural cavity, on the left side, contained about three ounces of a deeply tinged sanious fluid, without coagula. On the right side, the quantity was smaller, and the fluid less discoloured. The sinuses and larger veins of the brain were very turgid. The substance of the brain was of normal consistence, and had not been lacerated; it was slightly congested. There was no effusion at the base, nor in the ventricles of the brain, neither any remarkable congestion of the choroid plexus. The tissues throughout the body indicated a somewhat unusual degree of flaccidity."

Another case, in which death was produced after delivery, by concretion on the right and left sides, is recorded by Professor Giordano, of Turin, in an excellent work recently published by him on *Puerperal Fever*:—"On December 8th (thus his narrative) I had a severe case of puerperal convulsions in my wards. The patient, who was attacked on the eighth

day after childbirth, had had an enormous dropsy of the amnion. I had predicted that, in consequence of the evident dyscrasia of the blood, and of the undoubted obstruction which the excessive size of the uterus must have produced in the venous circulation, aggravated by the recumbent posture being maintained during most of the last fortnight of pregnancy, we should have to fear the development of puerperal fever. On December 1, labour pains set in. The labour lasted sixteen hours; five quarts of amniotic fluid were evacuated. The fœtus, contrary to expectation, was well developed. On the second day, there was slight and diffuse pain in the abdomen; the pulse was frequent. On the fourth day, the abdomen was supple, and painless, after the application of a poultice. The pulse was rather quick; there was cardiac dyspnœa, and tumultuous action of the heart. Incipient puerperal fever was diagnosed. Acetate of ammonia was given. The condition of the patient was for some days alternately better and worse; but, on examining her on the morning of December 7, I found a remarkable extension of the heart beat, which was diffused upwards as far as under the clavicle, and downwards as far as the umbilicus; it appeared as if produced by pulsation of the aorta. On the evening of December 8, the patient was suddenly seized with convulsions, which recurred at intervals of two or three hours. From the first attack, a comatose state, as from apoplexy, set in, and gradually increased. I was now called, and found the patient moribund; she died before any remedies could be applied.

"*Post mortem Examination.* There was nothing remarkable externally. The stomach and intestines were healthy, and much distended with gas. There were slight adhesions between the cæcum and the abdominal walls. The liver, spleen, and kidneys, appeared healthy. The uterus was large; its walls were healthy. The right Fallopian tube contained a small abscess near its fimbriated end. One of the ovaries contained a large corpus luteum. On opening the meninges, about a dessert-spoonful of blood escaped. The venous sinuses of the cerebrum were distended with dark blood. The cerebrum and the cerebellum were of normal consistence; a horizontal section, at the level of the corpus callosum, presented a dense, dark, punctuated appearance, evidently venous. The right pleural cavity contained about three hundred grains of serum; and its walls, at the lower part, presented thin and limited adhesions of recent growth. The lungs were healthy and crepitant. The right heart was large, and contained a dense white coagulum, very resistant, of oblong form; it adhered to the wall of the auricular appendix, and thence extended through the auriculo-ventricular opening into the right ventricle. The cavæ did not contain coagula; the descending cava contained some uncoagulated grumous blood. The left auricle was empty; the ventricle contained a large, purely fibrinous coagulum, about three grammes in weight. It extended into the aorta, and there divided. The smaller portion turned to the left, and extended for about an inch and a half into the arch of the aorta; the larger division completely blocked up the left primitive carotid, from which was drawn a piece of coagulum nearly four inches long, hard and resistant, and of tendinous appearance. The carotid was free and empty throughout its course; its walls were healthy, and pale."

For the particulars of a third puerperal case in which the death was sudden, the concretion taking place only on the *left* side, I am indebted to my friend Mr. Edenborough. The patient was delivered of her sixth child after an easy labour, on May 1st, 1849. On the following day, lactation was established, and the lochia were natural and moderate. On the 6th, "I saw her," says the writer, "quite well, as I thought; she had not one bad symptom. On the following day, I was sent for in haste, and found her sitting up in bed, suffering from violent spasm in the region of the heart, with insufferable dyspnœa. She was evidently dying. I gave ether; but, within ten minutes after my arrival, she was dead. I examined the body thirty hours after death. It was well developed; all the abdominal viscera and vessels were healthy. The uterus was firmly contracted. The lungs and pleura were normal. The pericardium contained five ounces of a pale, greenish coloured serum. The heart, of normal size, was pale and somewhat flabby. The right auricle and ventricle were normal. The left auricle contained three small fibrinous bodies. In the left ventricle lay a large mass of the same kind. It was not adherent; but, stretching diagonally across the mitral valve, obstructed the blood-current, and was evidently the immediate cause of death."

It is probable that in these cases there is some disposition on the part of the patient favouring the deposition. The disposition may be systemic, that is to say, the patient may be subject to a diathesis of which hyperinosis is an attendant condition. In Mr. Edenborough's case, the woman had suffered three years before her death from a slight attack of rheumatism affecting the wrist and fingers of the right hand; the disease seems to have been trivial, but nevertheless it indicates the prevailing disposition. Again, the tendency to deposit may be favoured by local causes, as by a diseased condition of the valves of the heart, and the impeded circulation consequent thereupon; in the case recorded by Dr. Smith, there was contraction of the arteries; I do not dogmatically assert that such predispositions always exist, but rather suggest that in cases such as those referred to, inquiry should always be made touching these

points. An affirmative answer on the subject derived from the facts of several cases would form a basis to certain very important practical inferences, prophylactic and prognostic.

The idea has occurred to many minds, that in all puerperal cases terminating as I have described, there is an empoisoned blood, the poison being specific to the puerperal condition. This opinion rests on the fact of a frequent concurrence of uterine phlebitis, and fibrinous coagula in the heart. I have no doubt that such combination is common, but, as I have before said, there is a distinct class of cases, such as those noted above, where the deposition takes place independently of any pre-existent uterine, or peritoneal, or venous lesion.

Hyperinosis, again, may follow upon some operations and some accidents, and, without developing itself with any local inflammatory change, may supply the cause for a deposit of fibrine. The reactive fever following important surgical operations, in the performance of which great arterial trunks are tied, is an illustration of this form of hyperinosis. In other examples, where a large tract of circulation is suppressed by other causes, as by effusion of serum on the brain, or constriction of the vessels of the neck, this same condition may occur. In the following singular example, inflammatory reaction, succeeded by deposit of fibrine in the heart, attended a suicidal attempt at death by hanging. I was called to this case, in consultation with Mr. Brown of Mortlake, on November 30, 1853.

A man, who had long been hypochondriacal, endeavoured to commit suicide by hanging himself, but was cut down before life was extinct. From the fact that the man had on his neck a stiff stock, and that his toes rested on the ground, his throat was saved from the direct compression that must otherwise have been caused by the cord, so that he went on breathing while suspended for several minutes. When cut down, the venous system of the head and neck was congested to a remarkable degree, and there was total unconsciousness and total anæsthesia. He was laid down, and some blood was drawn from the arm, but without relief. He continued breathing for about half an hour, at the rate of 13 respirations per minute; and the heart-beat was audible, though irregular and embarrassed. The body at the same time was cold and quite insensible. At the end of this period, the breathing began to quicken, and the pulse to rise. In proportion as these acts went on increasing in rapidity, the bodily temperature rose in every part except the head and face to a point above the natural standard. The whole of the surface then became covered with a profuse sweat. Two hours later, the breathing had risen to 60 respirations per minute, which were made without difficulty or abnormal sound. The pulse was at 120, full and firm; the body still hot and perspiring; the head still cold; and consciousness, sensibility, and voluntary muscular motion, absent. We now abstracted about eight ounces of blood from the temporal artery, but without any marked result. The blood showed a thick buffy coat. Four hours later, the skin continued moist; and, about this time, the bronchial tubes became loaded with secretion, which rendered the breathing difficult, reduced the force of the heart's beat, and brought down the animal temperature. Dr. Willis now saw the case with us, and we all thought the man would die from asphyxia produced by the bronchial secretion. On returning to the man at 7 P.M., three hours later in the day, we found, to our surprise, that the bronchial secretion had entirely disappeared, that the temperature of the body (the head still excepted) had again risen. From this hour, throughout the whole of the night and the following day, the symptoms remained the same; the breathing from 30 to 40 per minute, and free; the pulse from 100 to 110, and full; the body like that of a cataleptic person as regards sensibility and movement, but very hot. The blood of the man was, in fact, undergoing an active hyperinosis; and the result was obvious. But the question was, how long this existent condition would continue. I watched the case unremittingly, that new symptoms might not escape me. About thirty-six hours after the suspension—the first sign of concretion of the heart appeared. The heart became embarrassed; its action tumultuous; its sounds obscure; its beats less frequent. The temperature of the body gradually went down; the feet and hands became cold; and the face, which had before been of a pale livid hue, became as that of a mulatto. All this while the respirations remained steady, and took the lead of the circulation, until at length there was one respiration to two strokes of the heart. In this way he continued throughout another night, and till one o'clock on the following day. Half an hour previous to absolute death, the whole body was dark, and of a stony coldness. The fingers were actually rigid. The heart-beat was imperceptible, except at long intervals, when it gave a kind of sullen stroke. But the respiration still went on from ten to sixteen times a minute, affording, in truth, the only evidence of life. At last, this function reluctantly obeyed the will of our invincible and common foe; and death, who had for some hours possessed half his victim, grasped the whole.

No case that I have ever had illustrates with so much exactness the views I have been so long endeavouring to inculcate; and, as I may never again have such another, I shall enter a little into detail on the course and cause of the symptoms I have described. In doing so, I shall be at the same time describing my own train of thought as the symptoms were progressing before me. Commencing, then, at the beginning, the

theory I formed was this. In the first place, it appeared to me that the man, in suspending himself, had accidentally failed in making pressure on the trachea, but had succeeded in making sufficient pressure on the veins descending from the head to check the flow of blood through them. As a consequence of this, congestion of the cerebral sinuses, and of the cerebrum itself, had resulted; and therefore loss of consciousness and voluntary power. These were the primary results of the congestion. The next result was, that the congestion prevented almost entirely the course of the circulation through the brain; whilst, as the mechanical acts of respiration and of the heart's contraction continued, the circulation in the other parts of the body was persistent. But, as the whole of the circulating current had to make its journey through a circuit, shortened by all that the cerebral circulation represents, the heart was at first embarrassed with the increased pressure of blood thrown upon it, an embarrassment which it only got over by throwing so much the more of blood into the pulmonic and remaining systemic circuits. Upon this, in accordance with a law which I believe to be general, *that the force and activity of the respiratory movements are always in proportion with the amount of blood making the pulmonic and systemic circuits*, the respiratory movements were quickened; the blood was more freely oxidised; the heart was stimulated to greater exertion; and both respiratory and circulatory functions were raised to a pitch of action much above their natural standard. These conditions being thus set up, hyperinosis was a necessary consequence. The after results are easily understood. "General inflammatory fever", so called, was for some time manifested; the heart churned out the excess of fibrine; there was clogging up of the cardiac cavities by concretion; and, in the end, a complete removal of the inflammatory symptoms, obstruction of the whole circulation, typhoid or sinking symptoms, and death.

This is a brief sketch of the theory I formed and still retain as to the course of the symptoms and the death. However much of truth it may contain, one fact was clear, that the symptoms, for some hours preceding dissolution, were essentially due to fibrinous concretion on the right side of the heart; and so sure was I of this, that I took the opportunity of asking my friend and then neighbour, Dr. Cormack, to see the inspection, that he might, with Dr. Willis and Mr. Brown, attest the diagnosis. The body was opened (under the coroner's warrant) forty-six hours after death. The vessels of the head generally were congested to the fullest possible extent. The arachnoid was raised by a copious serous effusion. The whole substance of the cerebrum was charged with bloody points. All the sinuses and arteries were filled with clotted blood; and in the basilar artery there were white concretions. The brain weighed fifty-six ounces, imperial weight. The right auricle of the heart was distended with blood much beyond its normal dimensions. On opening the cavity, it was found filled with clotted blood, coated on the upper surface with *post cadaveric* buffy coat. In the right ventricle, there existed a fibrinous concretion, which was attached by its base to the mitral valve, and ran into the pulmonary artery; it was firm in structure, and was a perfect hollow cylinder. The walls of the cylinder were from three to four lines in thickness, and enclosed a column of coagulated blood. Smaller ramifications, all tubular, and enclosing columns of blood, extended into the branches of the pulmonary artery. The lungs were congested; but the larynx and trachea were quite patent, and showed no sign of having been injured. From one of the pulmonary veins another tubular concretion extended into the left auricle. The left ventricle contained no blood; but a small concretion was attached to it near the base of the aorta, and a thin thread of a similar description ran into the vessel. There was general congestion of the abdominal organs, but no other evidence of disease.

In some forms of apoplexy, this same condition of system may occur; *i.e.*, after the interruption to the cerebral system, there are excited a heated condition of body, tumultuous action of the heart, sweating, and death ultimately by slow syncope. In these cases, the last cause of death is not the coma, but the deposit of the fibrine.

Lastly, there is a form of hyperinosis occurring in old people, which destroys by deposition at the heart, with symptoms not easily mistaken when once understood. I believe that, in the period of "second childishness and mere oblivion", the natural mode of death—the mode, I mean, by which Nature means men and women naturally to die—is by the process of fibrinous separation. In other words, there is a tendency to deposition, which tendency is carried into fact on the occurrence of a very slight cause. We shall see an aged person, able to walk about, able more or less to converse, and one day, in the common acceptation of the term, well. Such an one shall take a so-called cold, or rather shall suffer from malaise, attended perhaps by very slight feverish symptoms and irritability. Speedily, and yet by gradation, these apparently passing symptoms lapse into those of concretion in the right or the left side of the circulation; and the death is ushered in with signs of syncope or convulsion, according to the point of the deposit. One illustration will suffice.

An aged man came under my care in August 1851. He was in his seventy-ninth year, a hale old man to look at, but with faculties dimming; an irritable man now; a sleepy man; a fidgetty man; loquacious about nothing, and transitional at a

breath from an angry to an amused mood; altogether like a spoiled child that can just talk, I should say. He had taken cold, he said; but he had no catarrh. He had a hot skin, a dry skin, a dry tongue, and prostration. He was treated with diaphoretic salines, followed by tonics; and he recovered. In January 1852, he was seized again with the same symptoms, these symptoms continuing for three days without much change, feverishness, restlessness, sleeplessness, without pain. The fourth day, his pulse, which before had been weak, was now irregular; and the action of his heart was tumultuous; the lungs congested. He was also inclined to somnolency. Next day, he became convulsed, and his hands and his feet were dark and cold; but his mind, except at intervals, was clear. So he progressed, dying step by step, and convulsion by convulsion. He died at midnight, on the seventh night after the commencement of the illness.

I had diagnosed here concretion on the left side of the heart; and the diagnosis was correct. The aorta, at its commencement and along its arch, was filled with a firm cylinder of fibrine, which sent up long branches into the innominata, and into the left carotid and subclavian vessels. The left ventricle was engorged with blood; the lungs were congested; and the right ventricle was congested. All the organs of the body were softened, from fatty change; but the condition was uniform; and, one organ being balanced against another, there is no reason but that life would have continued, had not the obstruction to the circulation closed the scene.

In these observations, I have included those cases in which the fibrine of the blood is absolutely increased. We may pass now to cases where the increase is relative rather than absolute.

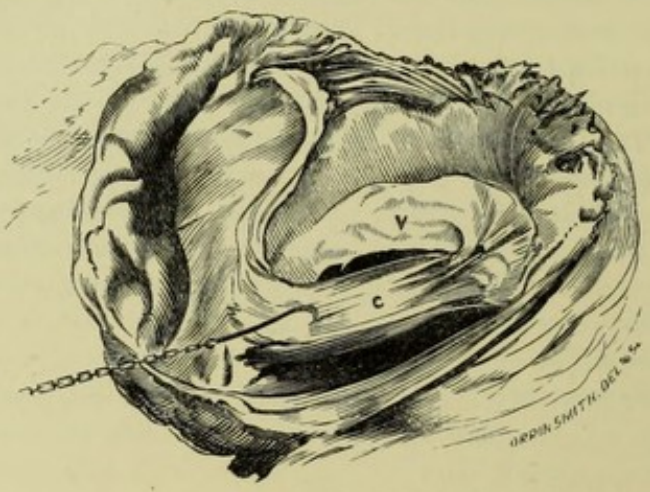


Fig 7.—V. Portion of **Tricuspid Valve**. C. **Concretion.**

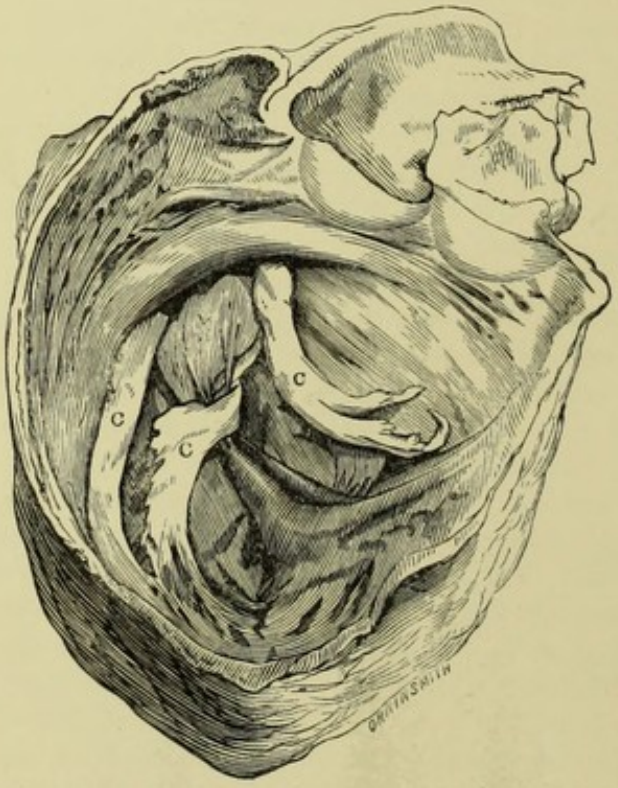


Fig. 8.—C C C. Concretions; Centre Concretion looping up the Cords of Tricuspid Valve.

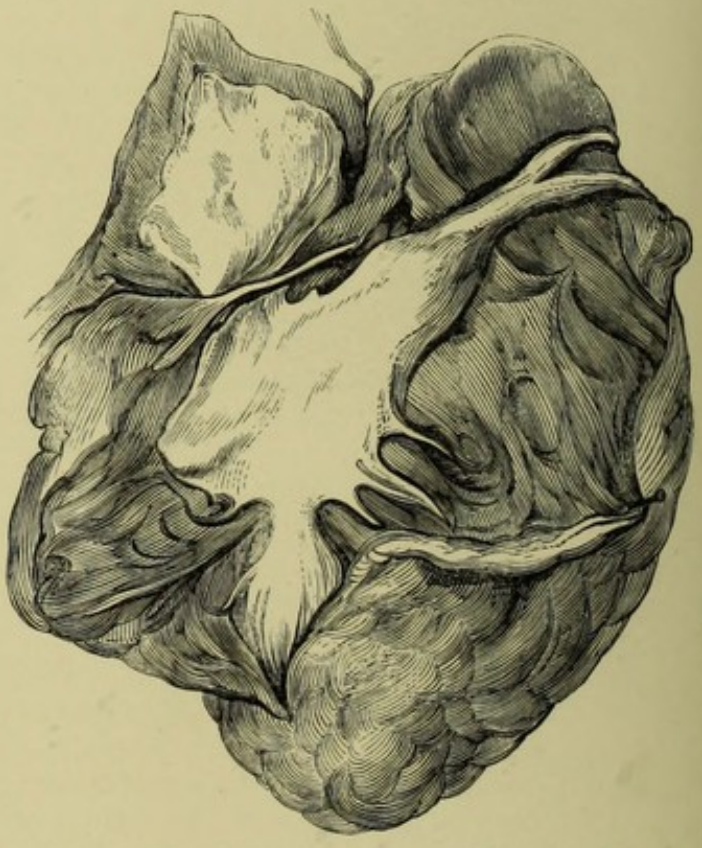


Fig. 9.—Concretion filling Right Auricle and Ventricle, and fixing Tricuspid Valve. (See Note III.)

CHAPTER V.

SYMPTOMS OF FIBRINOUS DEPOSITION:—RELATIVE HYPERINOSIS.

EXAMPLES in which there is a relative increase of fibrine, leading to deposition, are of different kinds. As I have already explained, the increase may relate to deficiency of water or menstruum, or to deficiency or over-distribution of the alkaline solvent of the fibrine. These cases divide themselves naturally, therefore, into two classes.

Cases of the first class are met with most strikingly in cholera. In fatal *cholera*, if the symptoms are much prolonged and the blood is drained of its fluid to the last extreme, the blood is found after death, as might be expected, condensed to a treacly mass everywhere, with, as a general rule, a fibrinous separation in the heart, usually, in these instances, I believe, on the left side. The symptoms of concretion here are not clearly marked; for those pertaining specially to the disease mask the specific signs of deposit. In *phthisis* attended with profuse colliquative discharge either from the skin or the alimentary mucous surface, the same tendency to deposition may develope itself. From this cause, patients sometimes die in consumption at an early stage, before there is set up any sufficient disorganisation to destroy life by the arrest of respiration, or by discharge from the lungs. A woman aged 44, two of whose sisters had died phthisical, was under my care in 1850. She had symptoms of tubercle at the apices of both lungs; but there was little cough, and actually no expectoration. Three months before her death, her life was threatened by profuse discharge from the bowels; but, under astringents, this flux was stopped, and no further bowel derangement followed. Soon afterwards, she was seized with violent heats, followed by the most profuse perspirations. The symptoms were almost of the ague type, and were amenable to no treatment. Meanwhile, the disease of the lung remained latent; but the appetite failed, and the tissues melted. Suddenly she commenced to sink, and died with all the signs of obstruction on the right side of the heart. I found, after death, tubercular deposit at the apices of both lungs; and a heart large, soft, and thinned. The right cavities of the organ were literally filled with separated fibrine, the concretion extending into the pulmonary artery; and in the left cavities there was also a deposit, which extended into the aorta. The concretions on both sides had firm hold upon the cardiac wall. The blood was coagulated in the vessels.

The examples in which the fibrine, from loss of its solvent, becomes deposited in the heart, are very numerous. We may, indeed, in reference to these cases, lay down a general proposition to this effect—*that whatever cause slowly enfeebles the circulation sufficiently, without producing a fluid condition of blood, leads to a deposit of fibrine.* By virtue of this law, we may have a deposition of fibrine during a prolonged syncope, after injuries which have produced shock, and after the administration of some poisons which slowly paralyse the heart, such as opium.

I am indebted to my friend and colleague, Dr. Cockle, for the history of a case in which concretion on the right side of the heart followed a severe injury. The case has a special value, from the circumstance that the mode of deposition was peculiar, as the sequel will show. A man was crushed by the fall upon him of brickwork. He was conveyed to the Royal Free Hospital, where it was found that there was fracture of the pelvis and thigh. The shock was extreme; but he lived on for four days, rallying a little, when he was suddenly seized with intolerable dyspnœa, followed by congestion of the lung, and death. At the *post mortem* examination, it was found that a band of concretion (Fig. 7), taking a firm hold on the right auricular wall, passed downwards into the ventricle, where, making a spiral turn around the tendinous cords of the tricuspid valve (Fig. 8), it looped up the curtains of the valves, and entirely occluded the auriculo-ventricular opening. By the side of this central concretion lay two others beneath the curtains of the valves, and less adherent.

I should but waste time to deliver more examples of this class of cases. Whoever chooses to remember the simple fact that, whenever the circulation is slow and the blood charged with no new solvent, deposition is an imminent occurrence, will understand the signs of concretion whenever they occur, and their origin. If I have to add anything to this observation, it is this—that whatever conditions exist favourable to fibrinous separation, such as anæmia, or its opposite where the blood is condensed, in such conditions very trivial depressing influences will turn the scale from imperfect solution of fibrine to absolute separation.

The mention of those conditions of system favourable to deposit from a change in the relative quantities of the blood constituents, leads me naturally to those examples in which there exists what I have designated as *spontaneous separation* of fibrine. We have seen that this state, curiously enough, may occur in diseases approaching to those in which the blood is made permanently fluid. In some forms of low fever, for instance, we have at the earlier stages deposit, and thereupon the exhibition of symptoms which run throughout parallel with those of the typhoid itself. I select an excellent illustration of this form of disease from the *Dublin Hospital Gazette* for December 15th, 1841. The report is by Mr. Rambant; the physician was Dr. Corrigan.

"Matthew Walsh, aged 70, was admitted into the Hardwicke Fever Hospital on November 20th, 1845, fourteen days ill. For the last one or two years, he had been much exposed to cold, and suffered much from poverty. Fourteen days before admission, he was seized with rigors, headache, and vomiting. He became, as he said, worse from day to day, lay in a small close room, and received but little attendance. The symptoms on admission were, shivering and cold extremities; the pulse very weak; the tongue dry, hard, and dark brown; all the secretions and excretions scanty; and the intelligence imperfect. He rested on his back, and made no complaint. The treatment was ether, ammonia, and brandy. On the 21st, 22nd, and 23rd, his sinking continued. He lay in bed on his back, still complaintless, with a pulse hardly perceptible. There was no tympanitis of the abdomen. On the 23rd, the tongue was moist, with slight mucous rattle in the throat. On the 24th, he showed more restlessness, with an inclination to attempt getting out of bed; on which day he died.

"At the *post mortem* examination, the lungs were found healthy in texture (with some effusion, not considerable in quantity, into the bronchi), but moderately collapsed. The tissue of the heart was healthy. The left ventricle contained grumous dark coloured blood. On slitting up the right ventricle, the pulmonary artery, and its branches, a singular appearance was presented. A bulbous shaped polypus (concretion) lay in the right ventricle, but was not sufficient to fill it. This polypus, narrowing gradually as it passed into the pulmonary artery, extended along this vessel, being of about the thickness of a quill; it divided at the bifurcation of the pulmonary artery, and continued its ramifications along the arterial branches. This extensive polypus of fibrine was so firm that, taking it by the root which lay in the right ventricle, it was drawn out, with all its attached branches, unbroken from the pulmonary artery. It was not attached anywhere to the sides of the arteries, but lay like a cord along their centre."

The following observations by Dr. Corrigan, in relation to this case, are, like all observations that have proceeded from his clear and unprejudiced mind, of great practical value.

"Two questions naturally suggest themselves here. 1. Was this polypus only formed in the last dying moments? 2. Had it any effect as a cause of death? As to the first question, I think the structure of the polypus itself shows that it must have been a considerable time in the process of formation, probably for some days. It was very nearly destitute of the colouring matter of the blood, except in the smaller ramifications of it. It was not of great size, such as we generally see in polypi of recent formation in the cavities of the heart, but was firm, comparatively hard, and small compared with the size of the cavities in which it lay. But the most satisfactory proof, probably, of its formation having occupied some time, is to be found in its structure. It consisted of distinct separate layers of fibrine. The outer layer or layers were comparatively soft, and, when laid hold of and pulled on, could be most readily drawn down off the middle fibrinous cord, which constituted the middle of this long-branched, tree-like polypus. This centre fibrinous cord was very firm and pale coloured. Those separate layers into which the polypus easily separated, resembled exactly the separate layers of lymph which are found in successive deposits of lymph in the trachea, in croup. On carefully examining this construction of the polypus, its position in the centre of the vessels, its extent in length, and its small diameter as compared with the size of the ventricle and vessels, it seems quite evident that this singular formation has been the growth of probably some days, and has been either the cause or the consequence of the sinking process of the circulation. But whether the cause or the consequence, it is plain that once formed, even in small extent, it would prove a serious hindrance to the circulation, and would most probably, even necessarily, cause death by its constantly increasing size and proportionately increasing obstruction to the function of the lungs and to the circulation."

Conditions of blood favourable to deposition may occur also in scurvy, and in purpura. I have already stated briefly the history of a case of this latter kind occurring in a child. The body of this child, during life, was covered with purpuric blotches, and such was the tendency to effusion, that even firm pressure on a clear spot of skin would give rise to a temporary blotch. One would hardly have conceived such a condition favourable to fibrinous deposit; but, after some weeks of great anxiety and languor, death occurred suddenly. And in the heart, in the right auricle, a concretion of fibrine filled the cavity, and sent a long prolongation into the inferior cava. The concretion was laminated, was clearly formed before death, and was the immediate cause of death.

As I have said once before, there may be deposition of fibrine in the heart as the result of paralysis of the circulatory organs by the action of poisons which depress, without adding to the circulating fluid any solvent of fibrine. There is much room for investigation in relation to this subject. In an animal struck down by the respiration of sulphuretted hydrogen, and left afterwards for some hours in pure air, there may be fibrinous separation. In a dog killed by a large dose of morphia, the death being prolonged over twenty hours, I once found also such separation, the concretion being on the right side, and filling the cavity, but being soft, and only slightly adherent. In the human subject, I have had no personal experience as to the production of concretion from the absorption of poisons; but the following case, reported in the *Lancet* for 1837-8, vol. ii, p. 626, by Dr. Frederick Page of Beccles (now of Landport), is of extreme interest, as indicating the possibility of deposition after the administration of *arsenic*. It may be a question truly whether, in this case, the deposit, which was clearly the immediate cause of death, was the result of the arsenical poisoning or of preexistent disease, independently of the arsenical dose; but, whether or not, the narrative deserves quotation in full.

"A fine healthy boy, aged 3½ years, swallowed a small piece of bread which was covered with butter, sugar, and arsenious acid (about eight grains), which his mother had prepared to poison vermin. It appears she had just turned her back to him for a moment, during which time the child ate it. I was immediately summoned to attend, and exhibited an emetic of sulphate of zinc, which produced powerful vomiting; and, in about twenty minutes from the time at which the poison was taken, the stomach appeared to be quite free from the drug. During the following three days, the child appeared to be as well as usual; but, on the morning of the fourth day, he was attacked with slight difficulty of breathing, quick pulse, hot skin, loss of appetite, and anxious expression of countenance, without sickness, purging, or any indication of pain. In the evening, he was evidently sinking, and died on the following morning, at about five o'clock, continuing perfectly sensible up to the time of his death.

"On viewing the body, ten hours after death, some few dark spots were visible on the surface. Nothing particular offered itself to notice on examining the stomach and the organs of digestion, with the exception of one or two slight traces of inflammation in the former viscus; the liver was enlarged, hardened, and of a pale yellow colour; the bladder was full, distended; the lungs and cavities of the pleura were healthy; the pericardium was filled with a pale straw-coloured transparent fluid. On dividing the venæ cavæ close to the auricle, a considerable lump of semitransparent, perfectly organised, coagulable lymph, fell from this cavity, in which was discovered much more. On opening the ventricles, large strings of the same substance were found to occupy their cavities, interwoven with the carneæ columnæ, and extending from the left cavity into the aorta, nearly filling up the ventriculo-aortic opening. The walls of the heart were pale, flabby, and soft."

It is probable that, in some forms of poisoning by the mineral acids or by inhalation of chlorine, where life is prolonged for some hours, separation of fibrine might also occur. But these probabilities we must wait to see solved.

CHAPTER VI.

SYMPTOMS OF FIBRINOUS DEPOSITION:—CHRONIC AND ANOMALOUS SYMPTOMS.

BEFORE leaving the subject of symptoms of fibrinous deposition in the heart, I must not forget to mention certain ones of an anomalous character, and sometimes prolonged over a considerable period.

It may happen that, in the course of the acute symptoms of concretion, relief may occur suddenly to the labouring heart, with the appearance of obstruction in a remote part of the circulation. It is worth while to recall the fact that Dr. Chisholm, in giving the history of his cases in Granada, speaks of the symptoms as showing a tendency to terminate in what, using the language of his time, he calls a "metastasis". One patient was relieved all at once, and without any evident cause; but perceived at the same instant an excruciating pain a little below the elbow, and nearly about the middle of the thigh. He continued free of the symptoms of cardiac polypus, but suffered from large abscesses at the points of the extremities where the pain was developed. Through the kindness of my friend Dr. Sibson, I was once enabled to see a case very similar to the one just noted. A man came under Dr. Sibson's care, suffering from terrible embarrassment of the heart. The embarrassment continued for many days, when suddenly it was removed, but was followed at the same instant by numbness and coldness of the left arm, which continued for a week after the event. The man had valvular disease of the heart; but, after this sudden relief, the anxiety about the heart did not return.

These cases are rare; but, as they occur, I must describe them, and their nature. The peculiar modification of symptoms is easily read off. It is due to the sudden dislodgment of a concretion from the left side of the heart into the arterial stream. The symptoms of major obstruction are removed, and new symptoms of minor obstruction are set up. The minor symptoms are recoverable without necessary loss of life or even of limb; for obstruction of a vessel will not cause death of a limb when there is free anastomosis.

There is a second class of cases in which concretion is produced in the heart, and does not at once destroy life, but gives rise *occasionally* to symptoms of the most distressing character, and which, often renewed, are at last fatal. These symptoms are so peculiar in different cases, that it would be difficult to subject them to any given descriptive rule. I shall prefer to illustrate what I know of them from histories of clinical facts. The following case was reported to me by Dr. Sayer.

A widow lady, aged 49, who had borne four children, had been the subject of much grief and anxiety; the extravagant and intemperate conduct of her husband (who died in 1819) having reduced her from a state of affluence to the verge of indigence. She had suffered for several years from uneasiness about the chest, accompanied with deep and frequent sighing. In March 1825, there was increased uneasiness about the chest, a sense of oppressive weight, frequent deep and prolonged inspirations, a weak, slow, tremulous and unequal pulse, a loaded condition of the superficial veins, a muddy hue of skin, an œdematous state of the feet and ankles, and withal a tendency to somnolency.

At the end of June, she had improved so much, that she was able to leave town by coach, and to repair to the sea-side, whence she returned home, early in August. She was now in that degree of health, to which she had been habituated for the last few years, being able to attend to her household duties, and to the education of her younger children.

At the end of September, she became suddenly afflicted with a return of all the former symptoms; for which little or no relief could be afforded. She died on October 30th, presenting during the last week of life symptoms of hydrothorax, hydrops pericardii, and œdema of the lower extremities, extending from the feet to the upper part of the thighs.

At the *autopsy*, 21 hours after death, the brain was found perfectly healthy. Extensive serous effusion existed in both pleural cavities. The lungs, quite free from adhesions, were

engorged with dark blood. The pericardium contained a small quantity of effused fluid. The heart seemed considerably enlarged, and was surrounded at its base by a large quantity of fat. A circular ulcerated spot, covered with a thin layer of purulent matter, and measuring an inch and one-eighth of an inch in diameter, was discovered on the upper surface of the heart immediately beneath the junction of the right auricle and ventricle. Both the cardiac cavities on the right side were thicker and firmer than natural. The cavity of the auricle was enlarged in size, and the musculi pectinati were unusually developed. Within the auricula of this auricle there existed an abnormal formation, a fibrinous concretion, firmly attached to the muscular wall. When pulled with considerable force, it broke off close to the place of attachment, leaving the base behind firmly adherent. The concretion measured two inches in length, and an inch in breadth at the base. It weighed three drachms and five grains (troy weight), and tapered to half an inch in diameter at its extremity, from the anterior edge of which issued an appendix, which measured an inch and a half in length, and terminated abruptly. The walls of the right ventricle were slightly thickened, and the columnæ carneæ, chordæ tendineæ, and tricuspid valves, were increased in size and firmness. In this ventricle, and amongst the columnæ carneæ, near the septum ventriculorum, there was found another similar abnormal formation, extending into the pulmonary artery and over the semilunar valves. The concretion measured eight inches in length, the last two being thread-like or filamentous. The width at the base was an inch. When pulled with much force, it broke off like the one in the auricle close to the point of adhesion, leaving the base firmly attached. It weighed two drachms and thirty-seven grains (troy). The pulmonary artery and semilunar valves were natural, and so also were the pulmonary veins. The foramen ovale was perfectly closed, but its margin was more distinct than usual. The left auricle was in its normal condition; the left ventricle was thicker in substance than is normal; and the mitral valves, the chordæ tendineæ, and columnæ carneæ, had acquired a corresponding development: this cavity also contained a small free coagulum, in which the separation of fibrine had commenced. The arch of the aorta and the semilunar valves were healthy and sound. The heart weighed one pound, seven ounces, seven drachms, and forty-two grains (troy weight). The abdominal viscera were all normal. The venous system generally was loaded with dark blood.

In the succeeding case, kindly forwarded to me by Mr. Duncan R. McNab of Epping, the symptoms are peculiarly interesting, in that one modification of them—viz., an alleviation dependent on change of position of the body—proved the existence of concretion in a way singularly convincing.

"A labouring man," so Mr. McNab writes to me, "under our care, was suddenly seized in the night with a sense of suffocation. After struggling awhile for breath, he partially recovered, and lived on for weeks, but breathing with great difficulty. The only relief he could obtain was by placing himself in one position; viz., by lying with his face downwards, and resting on his elbows. This circumstance was so peculiar to me, that I was induced by it to examine the body after death. I found the lungs milk-white and bloodless. In the right ventricle of the heart was a white tape-like concretion, attached by its base to the anterior wall of the ventricle, and continuing upwards into the pulmonary artery.

"The position of this concretion explains the cause of the relief obtained by the act of lying with the face downwards. For, when the patient was reclining on his back, the concretion must have floated across the pulmonary artery, and have checked the current; but when he lay on his face, the concretion would lie flat against the anterior wall, and the passage for the blood would be comparatively easy. I should add, that this man had consulted us about strange sensations in his heart a year or two previously; but his complaints were then regarded by us as due to profuse smoking, and as hypochondriacal in character."

Once more, there are cases where a fibrinous deposit, without causing rapid death, produces much the same class of signs as valvular disease; viz., lividity of the body and œdema. With the existence of this class of cases, and of the cause producing them, the illustrious Morgagni seems to have been well conversant, as the following history by him indicates:—

"A woman, previous to her death, had shown symptoms of universal tumidity of the body, and that livid hue which the skin usually exhibits when a great number of the subjacent veins are turgid with blood. The respiratory movements were performed with difficulty, and the pulse was feeble. The body of this woman was opened by Joseph Stancari, who found the vena cava and emulgent veins dilated, their coats partly cartilaginous, partly bony, and they, as well as the iliac and other veins, were nearly filled with a hard polypous concretion."

Another case somewhat analogous to this has been given by Dr. Burrows, in his admirable Croonian Lectures (*Medical Gazette*, August 15, 1835):—"A weak, emaciated, waxy-looking girl, who was under the care of Dr. Bright, suffered from palpitations of the heart. These were followed by œdema of the legs, great pain in them, and extreme enlargement of the

superficial veins. In this state she died; the œdema gradually going off, and becoming succeeded by wasting. After death a yellow, laminated coagulum was found to fill the inferior vena cava and the iliac veins, thus fully accounting for the impeded circulation."

But the most remarkable example of this kind is one reported by Dr. Fuller, in the *London Medical Gazette* for the year 1847. I must refer the reader to Dr. Fuller's excellent report and observations for the full particulars of the case. Its leading points are these:—A thin and pallid woman was admitted into St. George's Hospital on June 11th, 1846, under the care of Dr. Page. She was suffering from extreme pain in the right foot and leg, and was passing albuminous urine, but had few constitutional symptoms. On the 22nd, an aggravation of her symptoms took place. The right foot became colder than the left, and assumed a mottled appearance, as if from incipient gangrene; the pain also became more severe, and febrile disturbance showed itself. On the 27th, the whole of the right foot was perfectly black, and the leg greatly discoloured up to the knee. On the 28th a patch, of the most vivid red colour, presented itself on the hitherto black foot; and by the 30th almost all the toes on this foot, and the greater part of the foot itself, were of a brilliant scarlet colour; nevertheless, the whole foot remained perfectly cold. The femoral artery could be felt pulsating as usual; but it was hard and tense to the touch, and its contractions were evidently imperfect, as if from the blocking up of the cylinder. The left foot now became painful and gangrenous, while the peculiar redness already alluded to continued to extend up the right foot and leg, until it displaced the blackness, and continued some distance above the knee. On the 5th of July, vesications appeared on the right leg, and the left, which had gone through the same series of changes as the right, was becoming of the same bright red colour. Her back also showed a disposition to slough, and the extremities, from the hips downwards, had become perfectly cold. She continued to get worse until the 12th of July, when she refused food, and, after lying in a comatose condition (from which she could be roused, and was then quite sensible), she sank on the 15th, without any alteration in her symptoms. During the last week of her life, some parts of the legs and feet, which had presented the vivid scarlet appearance, became perfectly black, and emitted an insufferable stench. The autopsy disclosed no peculiar lesion of the lungs, or of the structure of the heart; but the right cavities of the heart contained coagula of a mixed character, some dark, others fibrinous and recently coagulated, and one small one in the auricle, evidently of long standing—it was quite firm, for the greater part discoloured, and, when laid open, its centre was soft and cream-like. In the left ventricle was a coagulum presenting the same characters as the one last described; but it was much larger, and occupied a considerable portion of this cavity, being entangled and firmly fixed in the muscles of the fleshy columns, with which it appeared in some places to be connected by slender adhesions. The liver was healthy; the spleen congested, but otherwise healthy. The intestinal canal and organs of generation presented nothing remarkable. The right kidney, much reduced in size, presented a well-marked specimen of granular degeneration. Its artery and veins were healthy. The vessels of the left kidney were blocked up by firm coagula. The kidney itself, much increased in size, presented a singular appearance; both its structures were for the greater part filled with an extensive deposit of a variegated appearance. The abdominal aorta immediately after the giving off of the superior mesenteric, and the common internal and external iliac arteries, were all blocked up by firm, light-coloured coagula of long standing, and adhering slightly to the internal coat of these vessels. In some places, the centre of these coagula was softened and cream-like. The internal coat of the arteries was neither thickened nor discoloured. The vena cava inferior, at the entrance of the iliac veins, was also blocked up by similar coagula, which were traced downwards into the various larger branches of these veins; but the upper part of the cava was quite free from coagula.

The muscles of the inferior extremities were discoloured; all the larger arterial trunks which were examined were blocked up by coagula, similar to those observed in the iliacs. The arteries were traced down to the dorsum of the foot; the veins were also blocked up by similar coagula. No diseased appearance could be detected about the coats of any of these vessels. The blood, examined microscopically, showed no unusual appearance.

There are, again, examples in which fibrinous concretions are formed in the heart, exist there for a time, and, without exciting any chronic or paroxysmal symptoms, destroy life at last by a sudden syncope. The old writers were so well conversant with this occurrence, that one of them, Queye, referred specially to it in an essay on syncope ("De Syncope et causis eam producentibus", Haller, *Disp. Anat.*, vol. vii, Göttingen, 1735); and Cullen favoured the subject with a similar mention. In modern times, we have had clinical histories bearing in the same direction. In cases of the kind in question, the fatal result is usually preceded by some muscular exertion. I will give two examples.

In *Guy's Hospital Reports*, vol. iv, p. 157, Dr. Hughes has

given a good illustration of this kind. The patient, a boy eleven years old, under the care of Mr. Aston Key, died on passing a motion from the bowels. An old fibrinous clot, of the size of a pigeon's egg, blocked up three-fourths of the right auriculo-ventricular opening.

The case of the aged lady, whose death was noticed in the first section of these lectures, and in whom an organised concretion existed in the left heart, is the second case of this kind to which I would refer. This lady had suffered, certainly, from symptoms which had been referred to the heart, but not of an intensity sufficient, as it seemed, to endanger life. Suddenly, after some trifling exertion, she fell and died almost instantly, the globular concretion being carried by the blood-stream into the aorta, and occluding it as effectually as a tight ligature around the vessel.

Lastly, the deposit of fibrine in the heart, in cases of old standing heart-disease, may give rise to peculiarities of symptom reflecting on those of simple cardiac disorder on the one hand, and on those of concretion on the other. In the instance of a girl who had suffered for some years with hypertrophy of the heart and mitral induration, an acute but slight rheumatic attack led to deposition, and to the most extensive deposition I have perhaps ever witnessed. In the course of this case, about four days before death, the mitral murmur disappeared altogether; the pulse at the same time becoming most irregular, and so quick that it was barely to be counted. Without any marked general indications of obstruction from clot, this patient, while sitting up in bed taking drink, exclaimed "Oh dear, how weak I am!" and died instantly from syncope.

Now, in this case, the disappearance of the mitral murmur was accounted for fully at the *post mortem* examination. It was found that the indurated mitral valve was held back by a fibrinous band; while the absence of the special signs of concretion was accounted for by the great size and dilatation of the left cavity. I was not at the time when this case occurred prepared to account during life for the disappearance of the murmur; but in another similar case, I feel that any such difficulty of diagnosis would be greatly removed. There remains yet much to be done in relation to fibrinous deposition in hearts previously and chronically diseased. I have at this moment no further data on the subject; and with this observation I close the history of the symptoms of concretion.

CHAPTER VII.

PATHOLOGICAL CHANGES INCIDENT TO FIBRINOUS DEPOSITION IN THE HEART.

WHENEVER there is deposition of fibrine in the heart, leading to obstruction of the blood and death, there are produced in other parts certain changes which, though not necessarily organic, are deserving of consideration.

The condition of the *lungs* first calls for attention. The lungs are always changed; but the change varies from two of the widest extremes—*i. e.*, from the extreme of bloodlessness to the extreme of congestion. These distinctions depend upon the position and size of the obstructing body. When the concretion is on the right side, and is of such size and in such position as to check the circulation to the lungs effectually, the state of the lungs is then very uniformly the same. The organs are inflated, blanched almost to pure whiteness, and bloodless. In some instances, emphysema is added to these signs: then the pleural membrane is seen elevated in bead-like rows over a considerable tract of the external surface. In children dying from deposition during acute hyperinosis, this emphysematous condition is, according to my experience, always present, and, as a general rule, present in the most marked degree. If the concretion, still supposing it to be on the right side, be so small or so placed that it will allow a current of blood to pass to the lungs, the normal force of the current being nevertheless considerably reduced, then the lungs, instead of being blanched, are congested throughout, and even condensed in depending parts.

When the deposition is on the left side of the heart, whether it obstruct much or little, and whether death be sudden or prolonged, the lungs are always congested. But, in prolonged cases, there is often more than congestion; there is effusion, œdema, sometimes exudation of blood itself, profuse secretion into the bronchial passages, and condensation of structure in depending parts amounting to hepatisation.

The *pleural cavities*, in all forms of deposition, sometimes contain serum of a pale straw colour, which varies in amount, but does not extend to a sufficient amount to cause compression of the lung-structure. In one example, I observed that some serum exuded into the pleural cavity coagulated on exposure to the air, a little fibrine having been thrown out with the serous fluid.

The *pericardial bag* usually contains serum in excess; but, again, not in sufficient excess to stop by its pressure the action of the heart. When this fluid is cleared away, one is often struck, especially in examining the bodies of children who have died of concretion laid down in the right auricle, with the peculiar appearance of the heart: the auricle, from distension, is as large, or is even of larger size than the ventricle; the veins of the heart superficially are distended almost to rupture; and the whole appearance of the organ indicates the intensest congestion.

The condition of the *large veins*, and of the parts below the heart, the *liver*, *kidneys*, and *spleen*, in cases where the concretion is on the right side of the heart, is one also of extreme congestion. In chronic cases, such as those related in a previous page, where the obstruction extends over weeks or months, there may be œdema of the lower parts of the body, or even effusion of fluid into the peritoneum. In a case seen by Mr. McNab, in which a child died with the signs of concretion on the right side, and in which the *post mortem* examination showed a firm concretion in the right auricle, with pathological appearances in the lungs such as have been so often mentioned—viz., bloodlessness and whiteness of texture—there was found intussusception in five portions of the small intestine, in one portion to the extent of three inches. Mr. McNab suggests that this intussusception may have been due to spasm arising from the deficiency of arterial blood in the intestine—a very probable hypothesis. The symptoms of concretion in this infant were observed first on Monday; its mouth then felt cold to the nipple of its mother; and it continued cold and apparently insensible to everything till Wednesday, when death occurred. I have not in my dissections met with any such con-

dition of the intestine as is here described in cases of death from fibrinous deposition.

The concretion being on the left side of the heart, the congestion of the abdominal veins and organs is less determinate; for in fact the lungs receive a large supply of blood, and the congestion is shared by the two circulations, pulmonary and systemic. In very prolonged cases, nevertheless, the œdema and other indications of central obstruction may have an extensive manifestation in the parts below the heart.

The brain is always congested when there is concretion on the right side, and this congestion may extend to effusion beneath the arachnoid or into the ventricles; but again the fluid effused is always small in quantity. Not unfrequently the sinuses of the brain will be found containing separate tubular deposits of fibrine. When the concretion is on the left side, the brain is also congested, as a general rule, but to a less extent.

It must always be a matter of care, in summing up the pathological appearances incident to concretion, not to confound these with conditions pertaining to other coincident forms of disease, nor to mistake secondary for the primary causes of those changes of structure which are presented by the scalpel. In the brief history I have given, I have kept this caution in view, and have intentionally reduced the description to the narrowest bounds, that there may be no chance of stepping beyond the simple and the true.

CHAPTER VIII.

CUI BONO?

WE have been led by our previous studies to the last, but not the least important, part of our subject. We are brought to consider the *cui bono*. Some writers, who would agree with much that I have said, ask whether, supposing it all to be correct, there is any good in the knowledge of the facts? If, say these argumentatists, things are as you have described, the hopelessness of medicine as a practice is broached openly, where it had better be concealed; for treatment there is none, when the diagnosis of fibrinous deposit in the heart is complete.

I demur altogether to this argument; but, as it has met me these ten years, daily I had almost said, I must state it as it is. I urge, on my side, that what seems hopeless knowledge, is ever better than floundering ignorance. I hope now to show that the information I have tried to convey has a threefold value: that it may, in the first place, make prognosis more accurate; in the second place, guard the practitioner against bad practice; and, in the third place, that it may in time lead to suggestions bearing directly on curative treatment. But, as a primary step, let us consider how far, in special cases, the existence of a concretion in the heart determines the course of a disease. From this point of view, fibrinous depositions have three meanings.

I. There is, as we have seen, a class of cases in which the deposition is the sole cause of dissolution. I have given already various illustrations of this variety of disease, when dwelling on acute hyperinosis. Those examples connected with puerperal fever are, *par excellence*, examples of the kind.

II. In other cases, the deposition of fibrine occurring in the course of an acute disease adds new symptoms to such disease, and renders fatal that which before was dangerous, but not *of necessity* fatal. In acute pneumonia, in pleurisy, in peritonitis, and, in fine, in all the sthenic inflammations, this effect of deposition may obtain.

III. In a third set of cases, the deposition of the fibrinous constituent may be the consequence of conditions in themselves *necessarily* fatal. We have had before us several examples of this nature. We may place advanced phthisis pulmonalis, cholera, deadly injuries producing shock, and poisoning by depressants, in this category.

It is my wish strongly to impress these distinctions on the mind; and I would specially guard myself, as I always have guarded myself, from the assumption that fibrinous deposition is in every case where it occurs *the* cause of death. I may offer a law on this point, indeed, without a touch of dogma. It is this: Fibrinous deposition occurring in the course of a disease ensures a fatal termination as a general rule: but the disease might be fatal without the occurrent deposition. This premised, let us turn to matters of prognosis and practice.

In all cases of the acute inflammatory type, whether slightly or broadly defined, the practitioner who is conversant with the risks of fibrinous deposition will be on his watch tower. He will remember the symptoms by which the existence of deposit is recognised; he will be guarded in his promises, and careful in his predictions. Most of all, he will be protected from being misled by those rapidly developed changes from severe local disease to suppression of local symptoms, which so often give rise to illusory hopes, and usher in the death.

Again, as I have said, the knowledge of the existence of a fibrinous concretion in the heart may prevent the committal of meddlesome, and, because useless, mischievous practice. In days when blood-letting had its full swing, when everybody was bled once in every disease, and once a day in inflammatory diseases; when even the convulsion of hæmorrhage was met by a recall on the lancet; in those days there arose, amidst all the confusion, this persuasion, that to bleed late in an inflammatory disease was bad practice. If I remember rightly, immortal Mackintosh himself, on every page of whose *Practice of Physic* there is a gaping vein, admits this rule; and the reason

for the rule is obvious enough. In the later stages of inflammation, the tendency to fibrinous deposition is increased. The effect of hæmorrhage is to increase the volume of water in the blood, to distribute the fibrine solvent, and to encourage fibrinous deposition. When, therefore, late in the inflammatory state blood is drawn, the balance of the blood constituents, already disturbed, is disturbed the more, and all that is wanting to secure the deposition of fibrine is secured. The rule, therefore, as I say, originated that it was better in inflammatory disorders not to bleed late in such disorders; and this rule, expanded now into wider pretensions, goes pretty nearly to the length of telling us that it is well to avoid bleeding at all stages; not because bleeding is not a grand remedy, but because it is too grand a remedy to be used without a definite knowledge of the time when to use it. I have, nevertheless, seen the rule about late bleeding in an inflammatory disease disobeyed even in our own time; and therefore I enforce it, and give the reason for its enforcement. In a case of acute pneumonia, which came under my observation some years ago, I was overruled to take blood. After the bleeding, the strong man fell like a shot bird. He fell into sleep, and he slept into death. A large fibrinous concretion blocked up the right cavities of his heart. This clot had possibly commenced to form before the venesection; afterwards its rapid increase was determined, and the death was hastened by the means intended for its prevention.

The remarks against the general abstraction of blood in the later stages of inflammatory disease tell with little less force against local blood-letting. Indeed, there is not unfrequently a greater danger in the local than in the general remedy, inasmuch as, owing to its supposed harmlessness, it is more fearlessly resorted to. "A few leeches can do no harm," is the common sentiment: it is an incorrect sentiment, nevertheless, and to be guarded against always, and with special care in cases of acute hyperinosis in the young. Speaking, indeed, of hyperinosis in children, I would, if my voice had any influence, raise it against the abstraction of blood altogether in such examples. There is a form of acute pneumonia often met with in well fed fat children. We put on leeches; the bitten surfaces bleed furiously; but the disease is not stopped. From such a patient, the more the blood taken, the greater the risk incurred; for, whenever the local indications of inflammatory mischief subside, the general indications of deposition are in the ascendant. "Breathless, pale, and bloodless", the end of the patient is at hand.

The same rule which tells against venesection extends to the administration of two classes of medicaments—purgatives and opiates. To reduce extremely the amount of water in the blood, by draining it off through the alimentary canal, is but to increase relatively the amount of fibrine in a blood in which the fibrine is already absolutely increased. And so, once more, to paralyse the heart with opium, during the existence of acute hyperinosis, is to double the danger of deposition by bringing into play that feeble condition of the circulation which we have described as in itself immediately favourable to fibrinous separation.

There are, further, some points of practice to which I would direct attention, in relation to one special disease and one particular surgical operation. The disease is croup, the operation tracheotomy. I have shown that, in croup, death may result from two distinct causes—from obstruction in the trachea or larynx, from obstruction on the right side of the heart. Some years ago, led by the observation of these facts, I was forced to protest against what I considered an indiscriminate resort to the operation of tracheotomy as a remedial measure in this disease. My opinions had the misfortune at that time to be opposed with great vigour and ability by my friend Mr. Henry Smith, an English surgeon who has, I believe, performed tracheotomy in more cases of croup than any other operator in this kingdom. I saw, however, no occasion to desert the position I had taken; and late events have confirmed the correctness and safety of that position. Recently, with a candour which is most honourable to his character, Mr. Smith has publicly stated that he believes himself to have been wrong. He admits that there are cases in which death in croup is the result of fibrinous deposition in the heart; and the practical lesson he leaves with his readers is, that, in these cases, the operation were better left undone. With such an important admission before me, I need only plead the seriousness of the question in excuse for some repetition in laying down the following observations for the guidance of the medical man in doubtful cases of the disease.

The symptoms which mark the cases of croup about to terminate in syncope, the result of cardiac obstruction, are distinct from those arising from obstruction in the air-passages, in the following particulars:—

In the cases of fatal syncope from arrested circulation, the dyspnœa is not caused by obstruction in the larynx, but by the peculiar anxiety and gasping desire to breathe incident to the want of blood in the pulmonic circuit. In this case, therefore, if the stethoscope be carried from the upper part of the windpipe downwards, and over the whole chest, the respiratory murmur is audible, and, it may be, clear throughout, so that the observer is prepared to say that there is here no such deficiency of respiration as will account for the severity of the symptoms. Again, the most

common physical pulmonic sign in these cases, is that of emphysema, which is often accompanied, in very young children, by a peculiar prominence in the anterior part of the chest. This emphysema, when present, is strictly diagnostic of fibrinous obstruction, and is altogether subversive of the idea that the cause of the symptoms is an obstruction in the windpipe.

In addition, there are, in these cases, the definite signs which mark the cardiac obstruction. The body is cold, and generally pale, almost marbly, but mostly so at the extreme parts. The lips are slightly blue; the cheeks are occasionally the same. The jugular veins are distended. The pulse is irregular. The body is painfully restless. The heart-beats are feeble, quick, and irregular; the sounds muffled. No real convulsions of the limbs occur, but intense anxiety and constant movement.

In those cases, on the other hand, where the death is really due to apnœa —the effect of obstruction in the air-passages— the symptoms are widely different. In these cases there is some point in the respiratory canal where an obstruction can be detected. The lungs show signs of congestion, but never of emphysema. The difficulty of respiration arises from an absolute inability to fill the chest. From the fact of the obstruction being in the respiratory circuit, such blood as passes through it is not arterialised, and the surface of the body, instead of being pale, as in cases of cardiac obstruction, is generally of a dark hue, with the veins more decidedly turgid. The muscles are not simply restless, but actually convulsed violently, the patient being unconscious of the fact; the heart-sounds are clear, and the motion of the heart, though feeble, is rarely tumultuous.

Lastly, the breathing is the first to stop at death, while, in the cases ending by syncope, the circulation takes the precedence in this respect.

If then in any given case the practitioner shall find the symptoms referrible purely to obstruction in the trachea or larynx, and the circulation unembarrassed, he will perform tracheotomy with good chance of success, granting that the point of obstruction is not too low, and that no further inflammation succeeds. If, on the contrary, he should diagnose the symptoms of cardiac obstruction, whether or not complicated with constriction in the windpipe, the operation is worse than useless; it will, of necessity, fail, because there are other fatal influences at work which the knife cannot reach and the most perfect respiration cannot remove.

These same precautions in reference to croup extend to another disease; I mean to diphtheria. In this disorder, as my friend Dr. Milner Barry of Tunbridge Wells has pointed out, fibrinous deposition in the heart is a frequent cause of dissolution, as frequent, I opine, as the obstruction arising from exudative plasma in the windpipe. In considering the propriety of tracheotomy in diphtheria, the same diagnostic rules would apply as in the analogous disease croup.

So much with regard to the acute forms of hyperinosis. In those states where the increase of fibrine is relative, or where there is tendency to deposition from sheer feebleness of the circulation, the rule against depressant measures is the more strongly to be enforced. I can add nothing further in this direction.

We will move from what may be called negative treatment to our third point—the lesson taught by our knowledge, such as it is, regarding a positive course of proceeding for the prevention of deposition. And here, I think, the first great truth which appears is that, to treat disorders marked by hyperinosis successfully, we must treat them by depressants at their early stages only, and must be prepared to move from the depressant system at much shorter notice than is commonly considered necessary. Before there is positively established in the blood an absolute increase of fibrine, I have no doubt that the conditions leading to such increase may be struck down by the use of rational means for the reduction of the force of the circulation; and so sometimes an acute inflammatory disease may be averted by free venesection. But, as it is rare that the practitioner is called to see disease in this early stage, bloodletting is dangerous to recommend as a general remedy. Whither, then, shall we turn for a remedy? We must turn, certainly, to those remedies which, while they reduce the force of the circulation, possess also a solvent effect on the blood. Is mercury such a remedy? I see no reason, either of a physiological or practical kind, to lead me to an affirmative on this question. In its physiological effects on animal organisms, mercury, carried to salivation, produces a peculiar disintegration of blood, but not a fluidity: in many of the cases of acute hyperinosis, in which after death I found large concretions, I trusted throughout to mercury, and gave it boldly, but with no result. The old calomel and opium pill is, therefore, to my mind, a combination deserving its loss of caste. When such a pill does good, I suspect the opium should have the sole credit, if not a little more.

Is antimony deserving of more favour? I believe it is. In a large number of experiments, in which I placed animals under the influence of antimony, I found in every case that the effect of the substance was to maintain a fluid condition of the blood; while common experience confirms that the drug has a power of arresting the force of the circulation. The danger of antimony, indeed, is that it may kill by the very depression which it produces; and its value, to put the opposite,

is in proportion to the sthenicity of the disease. Its value, consequently, is limited: add to which, the remedy may be put into the body very much quicker than it can be carried out; so that its administration in excess is a pretty sure way of securing a prolonged convalescence, should the hyperinosis even be checked, and the patient recover.

Salines, what of them and their uses in hyperinosis? In these, I opine, we have not only the oldest, but the best remedies. I speak specially of the vegetable salines. As I have often pointed out, it is a curious fact that the common vegetable acids, citric and acetic especially, act on the blood exactly as the alkalies; they maintain, that is to say, the fluidity of blood. In combination, therefore, these bodies have a rational intention in the treatment of all disorders attended by excess of fibrine, particularly when they are given with a free quantity of water. Given in full and frequent doses (and it signifies, probably, but little which acetate or citrate is chosen) they are, after all, the safest, and, according to our present knowledge, the surest medicines in the diseases under discussion. It is not easy to push them too far; they do not accumulate; they put a gentle check on the circulation; and their transformation in the economy does not apparently diminish the solvent effect produced on the blood by their administration.

The narcotic series of remedies, dangerous as they become in late stages, are invaluable in the early stages of inflammatory disorder. I do not now speak of opium alone, but of the volatile as well as of the stable narcotics. The whole class have the physiological property of arresting the oxidation of the tissues, and, as a result, of calming that excitement which is the preliminary and necessary step to hyperinosis. The more rapidly acting narcotic is therefore often the best; and if chloroform, as the most rapid of ordinary narcotics, were a practical remedy—a remedy which could be entrusted to other than medical hands for its administration—it were, I believe, the best antiphlogistic known. I have treated croup at its onset with chloroform by inhalation, with a success such as I have seen from no other plan of treatment. But the effects of the drug must be fully carried out; and, unfortunately for practical utility, the effects must also be sustained, often for many hours, and must be watched with an attention which none but an experienced mind can give. Opium, from being more convenient in its application, has the advantage of chloroform as a presentable remedy; but, as it is so much the less rapid in its action, and so much the more permanent in its effects when these are elicited, it is in a physiological sense so much the less valuable as an active remedy against deposition.

External applications in the shape of counterirritants, blisters and sinapisms, whatever may be their influence on the local lesion, can have no direct influence in preventing deposition of fibrine in the heart. If cantharidin applied to the cutaneous surface be absorbed, it may have an effect on the blood; but whether that is for good or for evil it is impossible to say. I do not make these remarks on counterirritants in opposition to them as local remedies for local affections, but simply to indicate that they are not to be considered as of any avail in meeting the more general mischief present in the inflammatory condition.

Presuming that the practitioner is under suspicion, in a case of acute hyperinosis or in a case of an asthenic kind, that deposition of fibrine is occurrent, what course shall he pursue? I know of but one: it is to give up at once all idea of carrying depletion further, and to throw in ammonia freely but with discretion. The carbonate of ammonia is the remedy usually given; but the liquid ammonia is the most direct and most satisfactory remedy: well diluted, it may be given in doses of five, ten, and by increasing it, twenty drops; and it should be given until it shows itself demonstrably and easily in the breath on the application of the hydrochloric acid rod.

It may be given at the same time that quina or the decoction of bark is given; but in most cases I suspect it forms alone the natural remedy. It is a chemical remedy; that is to say, it is applied for definite intentions.

But, while I thus recommend ammonia as a remedial measure which has to me proved of most value in the cases under description, and as a remedy which I have often brought into use in the early stages of acute disease, and always with advantage, I warn that, in prescribing it, its effects must be carefully watched. Carried to an extreme, it produces the very opposite condition to hyperinosis; and then it is no longer a remedy—it is a poison. In one case, recorded in my essay on the *Coagulation of the Blood*, in which five grains of bicarbonate of ammonia were given every two hours, the remedy acted, I fear, as a poison to a certain extent. The patient was sinking, and in some measure comatose, when the administration began; all the excreting processes being much diminished. After death, the odour of ammonia in the blood was most distinct. The blood was dark and thick, but uncoagulated. I collected half an ounce from the right side of the heart, and exposed it to the air for three days: it underwent feeble coagulation, but its true plastic quality was clearly destroyed. The corpuscles, as is common in such blood, were disintegrated, and modified in character; there were some stellate, others oval, others many-sided, others collapsed, but not one was of the natural appearance. (See note II.)

Let me, then, renew this precaution relating to the action of

ammonia, and extend it to the other alkalies. Indeed, the effects of potassa and soda, carried to the extent of producing the same changes, are more serious than those produced by ammonia; for the volatile alkali, being volatile, can escape more quickly from the system than the fixed alkalies, after its withdrawal; it holds, in fact, as a physical agent administrable to man, the same relation to soda and potassa as chloroform holds to opium or belladonna.

In those extreme cases where the fact of absolute deposition is too clearly demonstrated, what, then, shall be done? Alas! I can at this moment point out no satisfactory solution of this question. The day will come when the veil shall fall from our or our successors' vision; but now we can but grope our way, and pray for the light. I for one know, however, the difficulties in the way; and this knowledge, perchance, may be useful to some one in the future. I will therefore sketch it forth as matter for thought, and aid to future work.

It will occur to all, that the proceeding to be first considered is the removal of the fibrinous deposit by causing its dissolution. This is a definite proposition; the difficulties in the way are equally definite. In the first place, we possess no agent having a sufficiently rapid solvent action on separated fibrine. When fibrine is as yet in solution in the blood, it is the easiest matter in the world to keep it fluid; the addition of a very small quantity of alkali, say two parts of alkali to a thousand of blood, is sufficient for this purpose. But, when once fibrine has separated, its resolution is a difficult matter. True, it is not less soluble than before; but time is destroyed in the process. If, for instance, a solution be made containing so much as four grains of alkali to the thousand grains of a blood containing two grains of fibrine, a period of at least six hours must elapse before such fibrine would be redissolved. But such an amount of alkali thrown into the mass of blood as would bring that fluid altogether to the alkalinity required, would be in itself an impracticable, because a fatal measure. Under such alkalinity, the blood-corpuscles would be entirely dissolved.

Again, when deposit is the fact, the administration of alkalies by the mouth is in no way a direct means of effecting solution of the obstructing body. Medicines introduced in this way, granting that they are taken up into the blood at all, which is, in truth, doubtful, are but slowly carried through the heart, and are brought into contact with the concreted mass at a single point. From such limited exposure we can expect no result.

The administration of ammonia by inhalation in combination with the vapour of water, seems at first a reasonable suggestion. I have tried it both in disease and in experimental work. It fails. Ammonia is not absorbed easily by the lungs; and, as absorbed, it is never present in the blood, passing stroke by stroke through the heart, so as to produce any effect on a deposit of fibrine in this organ.

The injection of alkaline solutions into the veins may seem a reasonable project. It is worthy of trial in extreme cases, but I fear it would not succeed. If solution of clot could be determined by this process, I am afraid there would also be determined universal dissolution of the walls of the blood-corpuscles; and an animal without corpuscles in his blood is no longer alive. The first microscopist of blood little knew upon what he had dropped when he beheld these little invisibles. If we could or should at any time light on a chemical body which will dissolve fibrine on immediate contact, without destroying the blood-cells or the coats of the vessels, then we should have a remedy on which we might set our hopes. The difficulties in which the question is involved will be readily understood by this antithesis of argument.

The observations I have made above are, however, intended to bear entirely on cases of a kind where death is threatened within minutes or hours: in that series of cases where the symptoms extend over a long period, such cases as those recorded by Dr. Sayer and Mr. McNab, a course of treatment by a fixed alkali might possibly be productive of real good. Such treatment is based on philosophical and sound bases.

Returning to extreme cases: would it be possible, when the concretion is on the right side of the heart, to remove it by operation? I will relate an experiment bearing on this point. In one case, where a cat died in oxygen gas, I took the animal out of the box immediately after she seemed to cease breathing, laid bare the right external jugular vein low down in the neck, pushed through it a crochet-needle, passed that into the heart, and brought up to the point of insertion a separated mass of fibrine, which had before obviously filled the auricular cavity. In cases of dying in the human subject, *where all is hopeless*, I see no harm in doing a similar operation; the diagnosis of concretion on the right side being clear.

Before I conclude, I have to touch for a moment on a special question which has been specially asked of me. It is this. What are the conditions leading to hyperinosis? To this question I give frankly the reply, that it is at present unanswerable. To approach it even requires a new and extended inquiry into the whole subject of the oxygenation of animals—a subject to which I may at some future day be enabled to call the attention of the profession, but with a knowledge yet to be learned, and aspirations yet to be realised! *Valete.*

NOTE I.

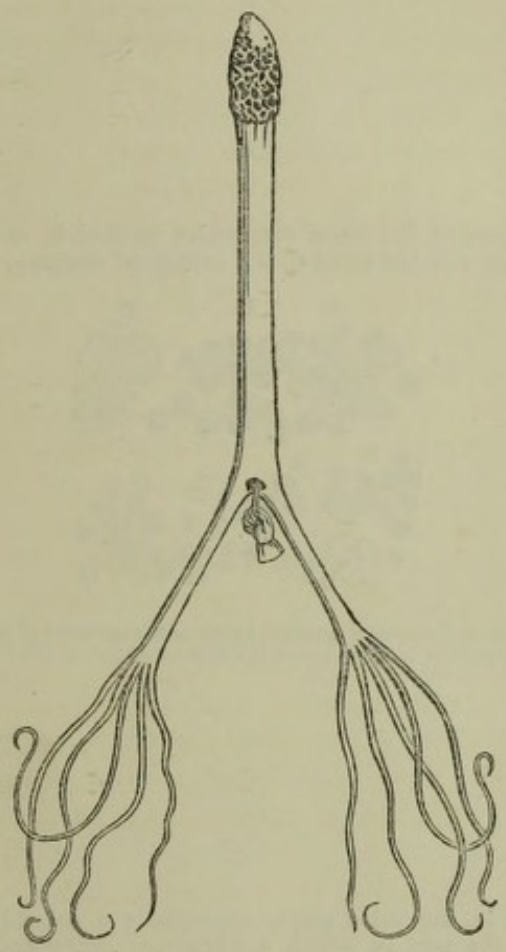
I APPEND in full the account of the "serpent in the heart" case, noticed in chapter I, page 3. Dr. May, the author of the case, was a man in good practice in London in his day. We must not laugh at him; for there may be physician-physiologists, two hundred years hence, whom we would have forgive our ignorance as we forgive.

"The seventh of *October* this yeare current, 1637, the Lady *Herris* wife unto Sir *Francis Herris* Knight, came unto me and desired that I would bring a *Surgeon* with me, to dissect the body of her Nephew *Iohn Pennant*, the night before deceased, to satisfie his friends concerning the causes of his long sicknesse and of his death: And that his mother, to whom my selfe had given helpe some yeares before concerning the Stone, might be ascertained whether her Sonne died of the Stone or no? Upon which intreaty I sent for Master *Jacob Heydon Surgeon*, dwelling against the *Castle Taverne* behind St. *Clements Church* in the *Strand*, who with his Man-Servant came unto me: And in a word we went to the house and Chamber where the dead man lay: We dissected the naturall Region and found the bladder of the young man full of purulent and ulcerous matter: The upper parts of it broken, and all of it rotten: The right kidney quite consumed, the left tumified as big as any two kidnies, and full of sanious matter: All the inward and carnose parts eaten away and nothing remaining but exteriour skins.

"No where did we find in his body either Stone or gravell. The Spleen and Liver not affected in any discernable degree, only part of the Liver was growne unto the Costall membranes, by reason of his writing profession.

"Wee ascending to the Vitall Region, found the Lungs reasonable good, the heart more globose and dilated, then long; the right Ventricle of an ashe colour shrivelled, and wrinkled like a leather purse without money, and not any thing at all in it: the *Pericardium*, and Nervous Membrane, which conteyneth that illustrious liquour of the Lungs, in which the heart doth bath its selfe, was quite dried also: The left Ventricle of the heart, being felt by the Surgions hand, appeared to him to be as hard as a stone, and much greater then the right: Wherefore I wished M. *Heydon* to make incision, upon which issued out a very great quantity of blood; and to speake the whole verity, all the blood that was in his body left, was gathered to the left Ventricle, and contayned in it.

"No sooner was that ventricle emptied, but M. *Heydon* still complaining of the greatnesse and hardnesse of the same, my selfe seeming to neglect his words, because the left Ventricle is thrice as thicke of flesh as the right is in sound men for conservation of Vitall Spirits; I directed him to another disquisition: but he keeping his hand still upon the heart, would not leave it, but said againe that it was of a strange greatnesse and hardnesse; whereupon I desired him to cut the Orifice wider: by which meanes we presently perceived a carnouse substance, as it seemed to us wreathed together in foldes like a worme or Serpent: at which we both much wondred, and I intreated him to seperate it from the heart, which he did, and we carryed it from the body to the window, and there layed it out, in those just dimensions which are here expressed in the figure.



Alongside the body of the "serpent", in the original drawing, is the following certificate:—

"That the 7. of October, this 1637 an Embrion of this forme and dimentions, as is here described was found in the Left Ventricle of the heart of *Iohn Pennant* Gentleman, of the age of 21. yeares, or thereabouts. Wee who saw it testifie under our hands:

Edward May Doctor of Physick.
Iacob Heydon Surgeon.
Elizabeth Herris Aunt unto the said *Iohn Pennant.*
Dorothy Pennant Mother to the said *Iohn Pennant.*
Ricard Berry
+Mrs *Gentlemans* marke.
This is my wives marke I testify *George Gentleman.*"

The figure has been here reduced to the scale of one linear inch in three.

"The body was white of the very colour of the whitest skin of mans body: but the skin was bright and shining, as if it had beene varnished over; the head all bloody, and so like the head of a Serpent, that the Lady *Herris* then shivered to see it, and since hath often spoken it, that she was inwardly troubled at it, because the head of it was so truely like the head of a Snake.

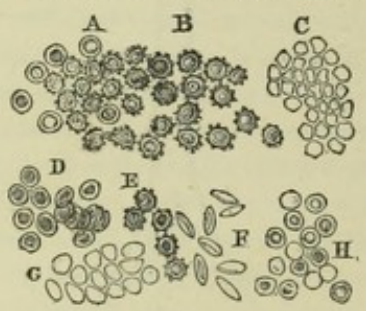
"The thighes and branches were of flesh colour, as were also all these fibraes, strings, nerves, or whatsoever else they were.

"After much contemplation and conjectures what strange thing that part of the heart had brought forth unto us, I resolved to try the certainty, and to make full exploration, both for mine owne experience and satisfaction, as also to give true testimony to others that should heare of it: And thereupon I searched all parts of it, to find whether it were a pituitose and bloody Collection, or the like: Or a true organicall body, and Conception: I first searched the head and found it of a thicke substance, bloody and glandulous about the necke, somewhat broken (as I conceived) by a sudden or violent separation of it from the heart, which yet seemed to me to come from it easily enough.

"The body I searched likewise with a bodkin betweene the Legs or Thighs, and I found it perforate, or hollow, and a solid body, to the very length of a silver bodkin, as is here described: At which the Spectators wondered. And as not crediting me, some of them tooke the bodkin after me, made triall themselves, and remained satisfied, that there was a gut, Veine or Artery, or some such Analogicall thing that was to serve that Monster for uses naturall: Amongst whom the Lady *Herris* and the Surgian made tryall after me with their own hands, and have given their hands that this Relation is true. This Lady dwelleth at the signe of the Sugar loafe in S. Iames street in the *Convent Garden*."

NOTE II.

THE changes of the blood corpuscles noticed in chapter VIII, page 40, are well indicated in the subjoined drawing.



A, B, C, E, F, G, Corpuscles modified by the administration of an alkali.
D, Corpuscles healthy, before the administration of the alkali.

The drawing was made from a specimen of blood taken from an animal under the influence of an alkali; but the modifications of corpuscle are identical with those which were presented by the blood of the patient noticed in the text. The oval corpuscle was specially well marked. This oval condition of the human blood corpuscle is sometimes present in other forms of disease. I have seen it in anæmic blood, and a similar observation has been made by Dr. Leared. The importance of this modification in medico-legal cases will be readily understood.

NOTE III.

FIGURE 9, in the third plate, exemplifies a second case not noted in the text, in which a fibrinous concretion, having a firm adhesion on the inner surface of the right auricular wall, descended into the ventricle, and entwining around the chordæ tendinæ entirely prevented the action of the tricuspid valve. The specimen was given to me by Mr. Spencer Wells, who, previous to death, had diagnosed concretion of the right side.