Remarks on the fibrinous element of the blood in relation to disease : read before the Medical Society of London, Nov. 15, 1851 / by Benjamin W. Richardson.

REMARKS

ON THE

FIBRINOUS ELEMENT OF THE BLOOD

IN

RELATION TO DISEASE.

READ BEFORE THE MEDICAL SOCIETY OF LONDON, NOV. 15, 1851.

BY

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ON FIBRINE,

&c. &c.

Physiological facts which bear upon the subject — Changes of fibrine during disease—Increase, *relative and real —Influence of respiration on the fibrinous element—Extraordinary increase of the fibrinous element in pulmonary inflammations – Cause of the increase of fibrine in inflammation, and of the excitement called inflammatory fever— Experiment—Diseases in which fibrine is relatively increased—Venesections – effects of, on the fibrinous element— Scurvy*—Decrease of fibrine—*causes of—from excessive fatigue—Effects of remedies—of mercury, of saline medicines, of alcohol—Fevers—divisions of —typhus, treatment in*—Change of character of the fibrinous element— *Coagulation during life—causes of— cases — Sudden formation of solid masses of fibrine in the heart—Valvular disease during rheumatism - Epidemics of fibrinous polypi—Chronic fibrinous concretions—effects of, on life.*

The subject of the paper which I have the honour to present to the Society this evening is of considerable importance; it is, moreover of great extent, and one to do justice to which would require a lengthened series of papers. While, therefore, a want of time will prevent me from bestowing upon it the due consideration it deserves, I shall, I trust, be enabled to bring forward sufficient matter to excite discussion for the evening.

As the subject I propose to discuss involves the consideration of fibrine in a pathological point of view only, I shall, as a matter of course, avoid those inquiries which pertain purely to the physiology of this important constituent of the blood—such as the questions whether it be an element for nutrition or an excrementitious substance? whether produced from albumen or blood-corpuscles? whether it is a product first formed in the vegetable world, and is afterwards transferred wholesale, if I may be allowed the term, into the animal body, according to the Liebig school; or a substance elaborated and perfected in the living body, as others assume? These questions, I repeat, I do not intend now to touch upon. At the same time I must intimate that I shall not fail to make use of those physiological facts which directly bear upon the pathology of fibrine, whenever I find that the recital of such facts will at all assist in elucidating obscure points.

To proceed, then, it is necessary for me, in the first place, to assume the presence of fibrine in healthy blood, and to fix its amount at a certain standard; for which purpose I shall select the figure derived from the mean analysis of Lecanu—namely, three parts in a thousand, as being at once the most simple and correct estimate. I must also state the fact that animal fibrine, however formed originally, is indisputably brought to perfection in the living body. Thirdly, I must refer to the circumstance that in health the fibrine is held in solution by the serum, and perhaps by the salts of the blood, and that, in all its course through the body, it is associated with a pretty constant quantity of albumen. And lastly, I would direct particular attention to the fact that, even in health, the proportion of fibrine in the body is continually undergoing slight changes; sometimes a real change, by virtue of a decrease or an increase in itself; and at other times an apparent or relative

change only, arising from corresponding alterations in the other constituents of the blood.*

The fibrinous element of the blood during disease may undergo distinct changes in quantity and in quality.

1stly. It may be increased.
2dly. It may be decreased.
3dly. It may be altered in character.

On each of these changes I wish briefly to treat; and I proceed by taking up *increase of fibrine*.

In the first place, then, it is interesting to observe that on no occasion does an increased quantity of the fibrinous element appear in the blood without being attended with a decrease in some other important constituent. Thus, as Simon shews, in almost all diseases of an inflammatory kind, where there is an increase of fibrine, there is at the same time a decrease of blood-corpuscle.† In other diseases, again, the increase of fibrine is attended with a decrease in the albumen, and so on. In all cases there is rarely, if ever, increase of fibrine with a coincident normal proportion of the other constituents of the blood.

Our knowledge of the metamorphoses which the blood undergoes is not, I regret to say, sufficiently clear to show us why this relative change in the different proportions of the blood should ensue. But the fact is not the less worthy of note, inasmuch as it testifies how very dependent each element of the blood is upon the others, and how careful analytical observers ought to be before they decidedly conclude that a separate constituent of the blood is either lessened or increased in quantity.

But admitting the full force of this remark, it must at the same time be allowed that the fibrinous element of the blood is capable of undergoing increase; and it becomes interesting, therefore, to inquire what are the circumstances which lead to such an increase.

As our knowledge on the subject is extremely scanty, I shall not enter upon many speculations with reference to the effects which a disordered state of the eliminatory processes of the body may have in altering the conditions of the fibrinous element; but I pass on to observe that it is now fully demonstrable that the respiratory act is intimately concerned in the formation of fibrine, and that the proportion of fibrine in the body is increased or decreased in proportion to the amount of oxygen imbibed in respiration. Thus, by the beautiful series of experiments lately performed by Dr. Gairdner, it has been proved beyond doubt that the blood of animals which have been exposed to an oxygenised atmosphere soon becomes highly charged with fibrine;† and the fact is still further borne out by the experiments of Prevost and Dumas,* and by the more recent ones of Nasse and Poggiale, which show that in those animals whose respiration is most active—such as birds—the temperature is not only higher, but the amount of fibrine is very much greater, than in other creatures.‡ While, on the other hand, the observations of Dr. John Davy prove that in all cases where death is induced by any process which deprives the lungs of atmospheric air—as in death by hanging, by fumes of charcoal, or by effusion of blood into the bronchia and air-cells—the blood remains destitute of the property of coagulation, "either from deficiency of fibrine, or from an alteration in the quality of that substance."§ This being the fact, then, it is by no means difficult to discover how it is that in some diseases the fibrinous element shall be in excess. We may not be able to prove, perhaps, that a highly oxygenised atmosphere ever produces such a state; but we can prove the existence of a condition in which the respiratory acts are so abnormally increased as to cause a greater exposure of the blood to the atmosphere than is consistent with health; under which circumstance there requires but one condition—viz. sufficient rest, to prevent an equivalent waste of fibrine, and then we may naturally enough expect to find an increase of this element in the circulating fluid—and this, indeed, is just what is found to happen in practice. In no diseases have we the fibrinous element of the blood so increased as in the first stages

* For observations on this subject, see Simon's Animal Chemistry, Vol i. p. 247, Sydenham Soc. edit.

† Animal Chemistry, Vol. i. p. 247.

* Gairdner on Gout, 2d edit. p. 1

† Ann. de Chimie, tome xxiii.

‡ See also Metcalfe on Caloric, Vol. ii. pp. 649-651.

§ Davy's Researches, Anatomical and Physiological, Vol. ii. p. 192 and 193.

of inflammatory affections of the lungs. Thus in pneumonia, according to an analysis of Rindskopf, the fibrine may be increased to as high a figure as 12 parts in the 1000,* and in pleuritis to 5 to 6.

Bronchitis, again, affords both positive and negative evidence of the fact in question, since, from the analyses of Andral and Gavarret, it would appear that while in the first stages of this disease, when a large quantity of blood is exposed too frequently to atmospheric influences, the fibrine is increased to a high figure, to 6, 7, and even 9 parts in 1000. In the later or chronic stages of the disease, as mucous secretion is poured out upon the bronchial membrane, thus diminishing the directness of communication betwixt the blood and the atmosphere, the fibrine falls to its normal standard, or even below it

But the great increase of fibrine in the blood, thus accounted for in the inflammatory affections just named, is not so easily explained in other cases—viz., in those inflammations where the respiratory movements are not at all concerned.

The above explanation accounts for the *extraordinary* increase of fibrine in pulmonary inflammations, but it does not account for the ordinary increase of this element, which is found in, I may say, *all* inflammatory affections.

The questions, in fact, concerning the increase of fibrine in inflammation, and the origin of what is called the accompanying inflammatory fever, are well worthy of thoughtful discussion; for, as far as I am aware, they have been as yet satisfactorily answered by no one.

Dr. Frantz Simon has attempted the matter, it is true, and has opined that the reaction which follows the setting in of inflammation quickens the circulation, and then more blood being exposed to the respiratory process, an increase of fibrine is the result.† Now, with all due deference to this illustrious chemist (and no one admires his immense labours, or deplores his untimely death, more than myself), I must say, that to me the hypothesis appears quite untenable,—1st. Because there may be inflammation and increase of fibrine without any preceding depression or reaction consequent on it. 2d. Because it is more than probable that the quickened circulation, which marks what is called reaction in inflammatory states, is the effect of an increase of fibrine, and that consequently such increase does not follow, but precedes the process of reaction. And, indeed, this supposition is quite in accordance with the views of Simon himself, expressed in another part of his great work.

After all, however, it is impossible to say positively that such is the case; but it can be stated as a thoroughly ascertained fact, that an increase of fibrine is always to be found simultaneously with the appearance of symptoms of inflammatory excitement, if not previously.

I find the question now before us very fairly put forward by our justly distinguished countryman, Mr. Wharton Jones. Does the inflammation, asks he, cause the fever, and the fever the increase of fibrine? or does the inflammation cause the increase of fibrine, and this increase the fever?*

I state my own opinion of this matter by observing that to me it appears that the inflammatory process gives rise to the increase of fibrine, and this to the attendant excitement which we call fever.

I am the more inclined to this opinion, because, in repeating some of the experiments of Dr. Gairdner, to which I have before alluded, not only have I found the amount of fibrine remarkably to increase, but I have also observed, that in proportion as the blood becomes fibrinized, symptoms precisely similar to those which characterize inflammatory fever present themselves. This in one experiment was peculiarly marked. A rabbit was put under the influence of oxygen; at the end of an hour the circulation had become greatly quickened, and the external temperature of the body was much higher than before. At the end of two hours the vessels of the ear became much enlarged, and could be seen most distinctly on raising

* It must be admitted that the increase of fibrine here noted is an exceptional example, inasmuch as in ordinary cases of pneumonia the fibrine only rises to 6, 7, 8, and rarely to 10, parts in the 1000. The person from whose blood Dr. Rindskopf obtained this analysis was a man 60 years of age, who was suffering from broncho-pneumonia. The blood was taken shortly before death. The full analysis is given in Simon's Animal Chemistry, Vol. i. p. 262.

† Animal Chemistry, Vol. i. p. 284 to 286.

* Essay on the State of the Blood-ves-els in Inflammation. Guy's Hospital Reports, 2d ser. Vol. vii. Part i.

the organ to the light, with all their anastamoses. The vessels were firmly injected with blood, so that very firm pressure on each side of an artery, made by the thumb and finger, was required to compress it, and to produce an arrest in the motion of the blood through the vessel. The temperature of the body was 102° by a Fahrenheit thermometer. Here, then, were almost all the symptoms which denote in the human subject an inflammatory fever—symptoms appearing most distinctly to follow an increase of fibrine; for, by this time, blood drawn from a vessel in the ear showed a very large increase of fibrinous element.

I have said above that the increase of fibrine is most probably dependent upon the inflammation. In this I have perhaps said more than may be considered allowable; since it may be supposed that in some forms of inflammation the increase of fibrine may be dependent on the same cause as the inflammation itself. Thus there is a decided increase of fibrine in erysipelas, a disease, as far as I can learn, depending entirely on atmospheric causes. I merely take one example; but I think that the analogy may perhaps be carried out more or less to all others.

I have before alluded to the fact, that there may be increase of the fibrinous element of the blood, not from any real increase in fibrine, but from a diminution in the other constituents, by which in health it is held in solution. This, I think, is shown in a very marked manner in phthisis pulmonalis, where the fibrine has been found increased to 6, 7, and even to 9. But, if we only consider how large a quantity of the watery portion of the blood passes off in this disease by the skin, and often by the bowels, and how at the same time the patient is disabled from taking muscular exercise, we need not feel much surprised at seeing the fibrinous element of the blood increased relatively to the other parts.

In Bright's disease of the kidneys, again, the fibrine often arises to a very high figure, but here there is an equal deficiency in some other constituents; for the blood does not lose its watery portion alone, but large quantities of its albumen also, in the discharges of pale urine, which characterize this disease in its progress. In these diseases, then, as in some others, the increase of fibrine may be relative, not real.*

With reference to the question whether venesection tends to increase the fibrinous element, I am in some doubt, since the analyses which have been conducted to settle the matter vary somewhat, and have not always been made on blood proper for the purpose. On the whole, however, I think the inference is pretty strong in favour of the supposition that it does lead to increase; but at most the increase is small, and by no means in proportion with the decided increase of water which follows the same cause.

I need not go on to enumerate in detail the other diseases in which fibrine is increased, since the causes of its increase are probably the same in all. There is, however, one disease on which I must for a moment dwell, inasmuch as there is apparently something anomalous about it: this disease is sea scurvy. In all the accounts which we have of scurvy, from persons well acquainted with it, we find that the causes which give rise to it, are, want of pure air and nutritious diet, insufficient clothing, great fatigue, and the like. Now, these are just the things which modern science declares, on the strength of careful experiment, to be most active in diminishing the quantity of fibrine in the body; while the symptoms of the disease also show a very marked degree of impoverishment and fluidity of the blood:† and yet, in the analyses of the blood of three patients suffering from decided survy on board the Dread-

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* In a case of albuminuria, with dropsy of a fortnight duration, Dr. Ayres found upwards of 11 parts of fibrine and tritoxide of proteine in 1000 parts of blood.—Lancet, Aug. 2d, 1845.

The same increase of fibrine in the blood is sometimes found during menstruation, owing, it is probable, to the same relative cause. The blood is at the time losing by the menstrual flow, water, albumen, and other blood constituents, but no fibrine; temporarily, therefore, the fibrine of the systemic blood seems increased. For analysis of the menstrual fluid, see Letheby, in the Lancet for Aug. 2d, 1845.

† Thus, in an excellent Essay on Scurvy, published in 1804, by Mr. W. Hunter, that author enumerates the following causes, as giving rise to a disease which he terms Cacotrophia, evidently scurvy with dropsy superadded:—

(*a*) "Food deficient in nourishment, or in stimulating power.
(*b*) "Respiring an atmosphere deficient in oxygen.
(*c*) "Preceding disease.
(*d*) "Intemperance.
(*e*) "Indolence, and want of exercise.
(*f*) "Excessive fatigue.
(*g*) "Suppressed perspiration."

rought, Mr. Busk found it buffed in two cases, and in all of them remarkably rich in fibrine. I repeat that these results greatly surprise me, and I should very much like to learn more upon this subject; for the impression forces itself strongly upon me, not that any error was committed in these analyses, but that at the time of their being made there existed in the blood another cause,—say an inflammatory affection of some kind, which led to what appears to be a remarkable deviation from expected results.*

I now come to consider the opposite condition to that last named—viz:

Decrease of Fibrine.—The circumstances which give rise to decrease in the fibrinous element, are those in which the process of fibrination is checked, or where the blood is overwhelmed with others of its constituents. Thus, as I have before hinted, in cases where the atmosphere is cut off from the lungs, this decrease instantly occurs; and so also, in those diseases which arise from improper food,† impure air, absence of evaporation from the cuticular surface: and suppression of the other secretions, as in cases of typhus, of continued fever, and particularly in cases of purpura hæmorrhagica. I allude strongly to purpura, because of its close affinity to scurvy; and I cannot but think that the analyses of Routier, which show that in this disease the fibrine is sometimes decreased to 1 part in 1000,‡ tends greatly to strengthen the view I have taken above with reference to scurvy.

It has been long known also that a decrease of fibrine is exceedingly common after great bodily fatigue. Thus, in instances where animals have been over-driven, the blood is sometimes found nearly destitute of fibrine. I dwell upon this remarkable fact, because it is very corroborative of the view that fibrine is dependent for its existence on the respiratory process. In an experiment by Dr. Gairdner, in which he caused a horse to be galloped, and afterwards bled, he found the fibrine, though apparently increased, of normal quantity in reality; and this result, he says, is what after-thought showed to him should happen, since it was evident that any increase of fibrine that might be engendered by the increased respiration would be at once made use of by the rapidly acting muscles.* Now, in the case of an over-driven animal, the circumstances are a little changed from this. The waste in the muscular substance is kept up for a long time, and the demand for fibrine is very great in consequence; but, the continued exertion leads also to failure in the respiratory process: the animal does not obtain the proper supply of air; the blood, consequently, is not fibrinized in proportion to the waste of fibrine implied, and hence, on examining such blood, we find a decided decrease in the fibrinous element.

On the effects of mercury, and of saline medicines, in lessening the amount of fibrine, I need not dwell,—they are so well known; but I must just make a passing observation with reference to the effects of alcohol. We are indebted to three American physicians—Drs. Peters, Goldsmith, and Moses—for an able report, embodying the history of 70 cases of death by ardent drinking, in all of which the blood seemed very deficient in fibrine, being fluid and dark, resembling cherry juice in appearance, and showing no tendency to coagulate.†

And, here, before I pass on to the third division of my paper, I take occasion to offer one or two observations on the different forms of fever, inasmuch as the past part of my subject leads me directly to the matter. And, first, I cannot too strongly object to the habit

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* The coming on of inflammation during typhus fever, a disease of the same type, will, it is well known, lead to an increase of fibrine. It is but right for me to state, however, that since the above was written, I have learned through the kindness of Dr. Hare, that by some analyses of scorbutic blood, lately made in University College Hospital, Mr. Busk's results have been fully confirmed.

† Lehman has found that the amount of fibrine is greater during the time that a person lives on animal food, than it is during a vegetable diet. And in this he is borne out by the experiments of Nasse on other carnivorous animals, such as dogs and cats. At the same time it is curious to observe, that the blood of herbivorous animals is richer in fibrine than is that of carnivorous.—Lehman's Physiological Chemistry, Vol. i. Cavendish Soc. edit. p. 358.

‡ Gazette des Hôpitaux, Vol. vi. No. 90.

* Essay on Gout, 2d. edit. p. 151.

† New York Journal of Medicine, 1845. Willan also narrates a case of purpura brought on by the drinking of undiluted spirits. I have alluded above to the great absence of fibrine in purpura. See Reports of Diseases of London, by Willan, p. 169.

we have of designating at least two most dissimilar diseases by one name. We call the excitement which attends an inflammation, fever. We call the prostration of all the bodily powers, which arises from a polluted atmosphere, or from contagion, fever also; and yet, were ever two diseases more widely different? We have seen that as regards the pathology of the blood they differ entirely; and I am sure we have but to look at the symptoms of each to see an equal difference. Induration and softening, anemia and plethora, atrophy and hypertrophy, are, in fact, not more distinct from each other than are the two states, which I would call inflammatory excitement, and true fever. I am sure that the distinction I here indicate is required; it would prevent many grievous mistakes; and, a strict regard for scientific accuracy seems to me imperatively to demand its being made.

There are one or two other matters with reference to fever,—of course I mean true fever, *typhus*—which I cannot let escape.

And, first, I would observe on the custom of giving large quantities of saline medicines, and especially of nitrate of potass, in true fever.

On theoretical grounds, it is evident that nothing can be more prejudicial than such treatment. The plastic element of the blood in such cases is already deficient in consistency, and, as some salines, like mercury, are known to have the effect of diminishing the consistency of this element, they should surely be as much avoided as mercury is avoided in such cases. And so also, for the same reasons, there is an objection to the profuse use of alcoholic and watery drinks.

And, what theory thus suggests, is, as far as my observations go, fully borne out at the bedside. The number of cases of fever which I have seen has been by no means few, nor have I been inattentive in watching them; and this I can say, that while I have seen much harm done by salines and alcoholic drinks, I have never yet seen a sinking patient from fever permanently rallied by the administration of any preparation of alcohol. I have spoken of the free use of watery drinks in fever; and these carried very far, as is often done, are likewise highly injurious. There seems to come on a state, indeed, when such drinks are not received into the circulation at all; when the specific gravity of the blood would appear to be reduced to that of the fluids given, in which state there is consequently no absorption; and the results are, a highly diluted state of blood, with an accumulation of fluid in the stomach and intestines, giving rise to tympanitis, uneasiness, weight, and other distressing symptoms.*

At the same time, I am bound to admit that the view I here take concerning the effects of saline substances in reducing the consistency of the fibrinous element of the blood, does not coincide with the views which certain very eminent continental physiologists take on the same subject

Thus I find Lehman, in the last edition of his work on Physiological Chemistry, directly combating this view, and urging, in support of his argument, some experiments by Scherer, in which that experimentalist tries to prove that inflammatory fibrine is not soluble in solutions of nitrate of potash. "How, then (asks Lehman), can a solution of nitre prevent the augmentation of fibrine, through a solvent power which in this instance it actually does not possess?" Now, unfortunately for this reasoning of Lehman, it is not only in opposition to the experiments of Zimmerman, who asserts that inflammatory fibrine is soluble in a solution of nitrate of potash, but it is actually in opposition to his own conclusions; since he, in a previous page, writes in contradiction to Sherer, by stating that, in all his own experiments on the blood of man, he has found the fibrinous element soluble in solutions of nitre, whether it be from venous blood, from arterial, or from that drawn during inflammation (at least, with two exceptions). Other contrary arguments, having reference to the amount of nitre necessary to dissolve the fibrine, and to the inadequacy of other alkaline salts in effecting such a solution, are also named by this chemist; but, as these

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* Adair, in his Thesis on Purpura (Edin. 1781), records a case in which that disease was self-induced in a farmer, by living on bread and water. And it is well known how much the same impoverished diet tends to bring on fever, an allied disease. It has also been shown by Albert, a German physician, that the water cure tends to induce symptoms similar to purpura.

are not so important, I need not relate them.* Returning, therefore, I now pass on to the third part of my subject —viz. change in the characters of fibrine.

Change in the characters of fibrine.— I have not time to dwell on all the changes of character which fibrine may undergo during life. Passing, therefore, those cases in which it has exhibited signs of putridity and of softening, and which have been so well described by Andral, Babington, Gulliver, and many other authors, I shall proceed at once to consider it as undergoing *coagulation* in the vessels of the living body. The doctrine that the fibrine of the blood may, during life, separate from the other constituents, and assume the solid form, though objected to by some writers, is, I think, capable of being fully established.† The circumstances which lead to such a state being:—1st, super-fibrination of the blood; 2d, reduction of the ordinary diluents of the fibrine; and 3dly, slowness of motion in the vessels.

Dr. Burrows, in his admirable Croonian Lectures‡ on the Pathology of the Blood, discusses this subject at great length, and points out particularly cases of fibrinous coagulation in the blood-vessels during life, arising from retardation in vascular motion. He narrates one case which was under the care of Dr. Bright. A weak, emaciated, waxy-looking girl, was suffering 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 mass of yellow laminated coagula was found to fill the inferior vena cava and the iliac veins; thus fully accounting for the impeded circulation.* Dr. Burrows also relates the particulars of another case, in which gangrene followed a somewhat similar cause; but I prefer to substitute for it a case of my own, which is, I believe, of the same kind.

The patient to whom I allude is a tall, emaciated, pale woman. She first became ill in 1848, complaining chiefly of lowness of spirits, with fits of excitement, and palpitations at the heart. One evening, on getting into bed, she felt a sensation of great heat in the feet, with pain, and, on looking at them, she saw that they were swollen. She now underwent some treatment, she cannot explain what, and soon recovered. After this she suffered from numbness in the feet, and sometimes in the hands, and in 1850 she became much worse. I saw her first on the 28th of February, 1851, and found her in a most painful and deplorable state. An enfeebled heart was acting with a rapidity rarely seen; the feet were dark and swollen, and the tips of all the toes were gangrenous. The superficial veins were very much enlarged, and she complained of intense pain. The heat of the rest of the body was low, and all the secretions were then natural. On examining into the cause of this gangrenous state, my attention was naturally enough turned to the heart, as having most to do with the matter, But in what state could that organ be to cause such results? Was there a constricted aortic orifice from induration of the valves? Of this there was no sign. Were there warty excrescences on the valves, and were minute portions of these being carried round the circulation, thus arresting the currents of blood in the extreme parts of the circulation; or, lastly, was there a fibrous concretion in the heart undergoing the same process, as I have once seen? I must confess that at first I inclined to the latter supposition; and, without anticipating more than temporary relief, I

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* Lehman's Physiological Chemistry, vol. i. pp. 349 to 363; Cavendish Soc. edit. Lehman affirms, however, that, in all cases, the solubility of fibrine by nitrate of potash is very imperfect. See an excellent paper by Dr. Basham, in the Med. Chir. Trans., Vol. xxxii. p. 1, in which the power of nitrate of potash in altering the blood during life is most satisfactorily shown.

† The belief that fibrine during life may undergo coagulation from arrest of circulation in the blood, seems quite in accordance with the views of Lehman.—Physiological Chemistry, vol. i. p. 349.

‡ Med. Gaz. Aug. 15, 1835.

* The coagulation of blood in the body from languid circulation was quite well known to the old writers. Thus Dr. Browne, the writer of a book entitled "Institutions in Physic," published in 1714, in a chapter devoted to Symptomatological Pathology (page 238), in speaking of the reception and expulsion of blood from the heart, remarks:—"But being much slower, and the expulsion still more languid, then follows a weakness, or decay of strength, *polypous concretions*, cold, *leucophlegmatic dropsies*, and several other diseases proceeding from hence."

prescribed mercury combined with morphia. To my surprise, as the woman came under the influence of mercury, the symptoms passed off, the sloughs disappeared, nourishing diet was well borne, and she became enabled to resume her ordinary occupations; coldness of the feet only remaining.

After some three or four months of respite, this woman again suffered from the same symptoms; and, on this occasion, they seemed plainly to have been brought on by an attack of diarrhœa, which had much reduced her. The right hand was now also affected. I employed on this occasion a liberal use of saline medicines only, with light nutritious diet, and she recovered. Since then I have seen her suffering from two other similar attacks, and I have therefore been enabled to watch the matter closely. The chief causes of these attacks are anything that lowers the body, and robs the blood of its fluid parts, especially profuse purging. Rest also has a great effect in bringing on the attacks; and she can sometimes ward them off altogether by taking a sharp walk when feeling premonitory symptoms. I have never seen the gangrene proceed very far; and it always passes off dry, leaving beneath a delicate epithelium. During the attacks the superficial veins are always enlarged, and, about the flexures of the joints, distinct, moveable, little pultaceous bodies, about the size of a pea, may be distinctly felt, and pushed on in the course of the venous current.

On examining the heart, I find no trace of any organic disease; and in this opinion I am happy to be strengthened by the opinions of my excellent friends, Drs. Arnott, Willis, and Crisp; all of whom have seen and carefully examined the case with me.

Reverting, then, I set this case down as one of the same kind as those described by Dr. Burrows, only in a less severe form. I believe that the gangrene is the result of a soft coagulation of the fibrinous portion of the blood in the extreme venous trunks, produced by enfeebled power of the heart, and especially so when this is attended with a diminution in the fluid constituents of the blood. Three things lead me to form this opinion:—

1st. The appearance of the gangrene after any cause which gives rise to an enfeebled state of the heart, or robs the blood of fluidity.

2d. The enlargement of the veins, and the existence in them of the soft masses to which I before alluded.

3d. The very good results which invariably follow the effects of mercury, saline medicines, and mild nutritious food,* and especially the prophylactic power of active exercise.

The formation of *solid* masses of fibrine in the heart and bloodvessels during life is another subject of extreme interest, and has at various times excited violent discussion. Of the existence of such depositions during life I am thoroughly convinced, and I am inclined to the opinion that they are of far greater importance than some pathologists of this day are given to admit. In another place† I have not only shown that, when the blood is surcharged with fibrine, these deposits may be left upon the valves, but that, in the event of a valve being indurated, there may be left upon it suddenly a deposit of fibrine of sufficient size to bring on instant death by syncope; and I have detailed a case from the essay of Dr. Crisp, in which a poor woman, on exerting herself after her confinement, died suddenly, as I opine, from a rapid deposit of fibrine on an indurated aortic valve, by which the grand exit for the arterial blood was closed, and the circulation was at once completely arrested.‡

I might here greatly enlarge on the question, whether that peculiar state of the valves, which occurs by virtue of some

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* This conclusion is quite in accordance with the experiments of Zimmerman, which show that the amount of fibrine in the extreme venous trunks is greater than in those near to the heart. It is also in most perfect accordance with the opinions of Bouchet, expressed in his paper "On the Spontaneous Coagulation of Blood in the Veins in Cachectic Diseases." See Dr. Ranking's Retrospective Address, Prov. Med. and Surg. Trans., 1846.

† Medical Gazette, August 8th, 1850, "On the Different Ways in which Death Occurs."

‡ Diseases of Blood-vessels, p. 88. The experiments that have been made by Magendie, and which show that foreign substances, on being introduced into arteries and veins, become loaded with a fibrinous concretion, are remarkably corroborative of the position I have here taken.

See some remarks on this subject in Carswell's Pathological Anatomy, Fasciculus 11th, Analogous Tissues; and in the very excellent Lectures on Pathology lately published by Mr. Simon, of St. Thomas's Hospital—Lancet, 1850.

Vogel and Wagner also believe in the direct deposition of fibrine from agitation against the valves of the heart.—Willis, Wagner's Physiology, p. 267.

change of fibrine during rheumatic fever, is caused by inflammation of the lining membrane of the heart, or by direct deposit. On this matter I shall only remark that, while I am fully convinced that fibrine may be directly deposited, and while I also admit that the internal lining membrane of the heart seems from its structure not to favour the exudation-theory, I must allow that, in this particular case, there does appear to be something more at work than simple deposit. Otherwise, I see no reason why similar valvular depositions should not occur in other cases where the blood is super-fibrinised, as in pleurisy, pregnancy, phthisis, pneumonia, and the like—diseases in which such results never happen, as far as I am aware.

The tendency to coagulation of fibrine during life has, it would seem from the old writers, sometimes occurred in a very striking form. Thus I find, in the Edinburgh Annals of Medicine for the year 1800, an account by the famous Dr. Chisholm of what he calls an epidemic polypus, occurring in Granada in 1790. He records that forty negroes, after being exposed to excessive heat, were seized with peculiar symptoms, the most important being vehement palpitations of the heart; the audibleness of which, he adds, was so great that the owner of the slaves could hear the palpitations in an adjoining room. Seven of these negroes died. In the first two cases the heart was not opened; but, as every other organ appeared healthy, the heart was inspected in the third case, and revealed what appeared to Dr. Chisholm to be the cause of the whole mischief. In the right ventricle there was found a polypus of great extent, two inches in breadth; while, in the fourth body, there were large, firm, polypous concretions in both ventricles, and in the right auricle. In the 5th, 6th, and 7th cases, were the same appearances. "After finding these signs (the Doctor adds) I changed my plan of treatment. On first detecting symptoms of the epidemic, blood was drawn, and afterwards the patient was freely salivated with mercury. The results were most happy—not one that was salivated died."

I wish I could go further into these cases, the symptoms are so interesting, especially in illustrating the influence of mercury on the fibrinous element of the blood. An account of another similar epidemic is also related by Dr. Huxham, in the Philosophical Transactions. This happened amongst some sailors. Palpitations here also were very prominent, and twenty died. On opening the hearts of two of them, monstrous polypi were found on both sides, weighing in one or two cases an ounce.* I do not dwell on these cases.

Amongst the old physcians, the existence of fibrinous concretions in the heart, in what may be called a *chronic* form, was likewise fully known, as well as the effects with which they are likely to produce. Thus I find one Dr. Wm. Gould† writing in 1684, describing these concretions and their effects with an accuracy as surprising as it is valuable. Remarkable, indeed, it is to find this simple observer, a man ignorant of course of all that pertains to a knowledge of the chemistry of the blood, naming those diseases as most apt to give rise to polypous concretions, which modern science has demonstrated to be very abnormally rich in the fibrinous element, and which modern pathology has proved to be commonly followed by fibrinous coagula. Pneumonia, pleurisy, and phthisis pulmonalis, enter particularly into this writer's category.‡

The effects of such concretions in inducing sudden death were also particularly noted by this writer, and by many others. Queye, the writer of a Latin essay on Syncope and its causes, published in Haller's Collection, names polypi of the heart as one common

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* Philosophical Transactions, 1740.

† Philosophical Transactions, 1684. The question relative to the cadaveric or anti-cadaveric origin of these concretions was one of great importance with the old writers. Thus, Dr. Gould, the author above noted, attacks the opinions of Kirckringius on this subject in the paper referred to, he himself being a firm believer in their existence during life. On this matter see also a letter by Pasta, published in 1737, entitled Epistola de Cordis Polypo, in Dubium Revocato.

‡ Burserius attributes to pneumonia the power of producing polypous concretions in the heart, and in this opinion he is followed by Grisolle, who in his Treatise on Pneumonia states, that in one-fifth of the cases inspected by him he found dense concretions intertwined among the columnæ carneæ. Burserius, Institutionum Medicinæ Practicæ, vol. iv. chap. x. p. 233; Grisolle, Traité Pratique de la Pneumonia, p. 70-76; see also Bouilland, Traité Clinique des Maladies du Cœur, tome ii. p. 710-726.

cause of death by syncope,* and I find Cullen,† and many others, making the same allusion. A very interesting symptomatic fact with reference to these concretions is, that no symptom has attracted so much attention during their existence in the heart, as the violent palpitations which they seem to induce; and it is still more curious that a late author at the Royal Med. Chir. Society, in describing a case of the kind occurring in a patient of his own, speaks of violent palpitations of the heart as forming the chief peculiarities of the case.‡

Did the occasion permit, I could lay before you many cases illustrative of the effects of these concretions in producing sudden death, and proving that we might not be far from right in falling back in this matter, on some of the observations of the old school. The subject, however, has not been entirely neglected in the present day. Dr. Hughes has produced an elaborate paper on the subject, in which he has divided these concretions into different varieties, and has given a table containing a great many cases, with intent to show that such concretions are more commonly found on the arterial, than on the venous side of the system. I have looked over this table, I may add, and in addition have taken the pains to collect evidence on the subject from many more cases, and the result is, that I can second Dr. Hughes' supposition, though at the same time I may remark, that the difference is not very striking.

Dr. Hughes also affords further distinctive marks of cadaveric and anti-cadaveric polypi, and speaking of the effects of these concretions in the living heart, he supplies cases of sudden death from their presence.§

In a paper read before the Medico-Chirurgical Society, Dr. Barclay has also briefly alluded to this subject; and from the observation of 79 cases in which the heart was examined, he is led to conclude that fibrinous concretions are most common in the young.* With this opinion I fully concur, and I think it accords admirably with the fact pointed out by Wagner, and other physiologists, that the fibrinous element exists in all animals in greater proportions during the first periods of life

Lastly, it occurs to me that in some cases of death by asthenia, where these fibrinous concretions are found filling entirely the outlets and the cavities of the heart with an extension even into the bloodvessels, the very cessation of the act of life may be owing to their presence, the central organ of the circulation becoming literally choked by them.

In a case which I had an opportunity of inspecting in the month of August last, I was particularly struck with this occurrence:—A gentleman, who had been accustomed to business habits, was suddenly seized, while preparing for a continental tour, with considerable paralysis on the right side of the body. At the same time the lungs showed signs of tubercular deposit. He was kept quiet, and lived on for three months, being carefully supported throughout with good food, which he always took with appetite, and seemed to digest easily. He sank, however, and died by a gradual failure of the circulation. At the autopsy, the corpus striatum of the left side was found a little softened, and the lungs were filled with miliary tubercles; the heart was soft, and the aortic orifice and the ascending part of the aorta were completely filled with a round, dense fibrinous cord, which had evidently been for some time in process of formation. The same thing was also found in the pulmonary artery, but the concretion was not quite so large and firm.

A case differing somewhat from this in primary symptoms, but ending by asthenia in a similar manner, is recorded by Dr. Corrigan in the Dublin Hospital Gazette.† A man was admitted into the Richmond Hospital

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* Queye de Syncope et causis eam producentibus, Hall. Disp. Anat. vol. vii., Gottingen, 1735.

† Cullen's First Lines of Practice of Physic, edited by Gregory. Edin. 1829, vol. ii., Art. Syncope, p. 38.

‡ Dr. Fletcher, of Birmingham.

§ Guy's Hospital Reports, vol. ii., p. 147. The subject has also been ably discussed by M. F. Fredault, in an Essay on Polypiform Concretions. He looks upon these clots as pseudo-membraous transformations deposited from the plasma of the blood during life, and believes fully in the coagulation of blood in the body.—Archives Générales de Médecine, July, 1847.

* Med. Chir. Trans., vol. xxxii., p. 135. Dr. Barclay does not, however, altogether incline to the theory of the direct depositions of fibrine.

† Vol. ii., Dec. 15th, 1845.

supposed to be suffering from fever. "The case," says Dr. Corrigan, "presented no appearance of head symptoms. There was no affection of the abdominal viscera. The lungs presented no signs of engorgement except in the last day of life. There was no complaint of pain; the patient was merely feeble, lay on his back, with a cool skin, and a very feeble and daily sinking pulse. The patient was then dying of the failure merely of one function—that of the circulation." At the inspection, the lungs were healthy and the tissue of the heart soft. On slitting open the left ventricle it was found to contain grumous dark-coloured blood. In the right ventricle, pulmonary artery and branches, a singular appearance was presented: a bulbous-shaped polypus lay in the right ventricle not sufficient to fill it. The polypus passed also into the pulmonary artery, and continued its ramifications along the arterial branches." Dr. Corrigan, after giving reasons to show that this concretion was not formed in the dying moments of the patient, but was of previous formation, next traces its effects, and speaks of it as having been "either the cause or the consequence of the sinking powers of the circulation." "But whether the cause or the consequence," he adds, "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." * * * This case, I presume, adds powerful testimony to the correctness of the views which I have proposed above.

I have thus given a rapid sketch of portions of a very important subject. I have shown how I conceive the fibrinous element of the blood may be increased in certain diseases; I have dwelt on the manner of its decrease in other diseases. I have ventured to criticise our present habit of calling two dissimilar diseases by the one name, of fever. I have spoken of some changes of form which fibrine may undergo during life, and I have traced the effects of such changes as regards life.

As I said at first, the subject is too long and too important to have any justice done to it in one paper, and although I have kept as much as possible to general views and inductions, yet I still feel that even with reference to the matter now brought forward, many things have been omitted which might very properly be made subject of discussion in this place. At the same time I cannot forbear to express a hope, that my paper, as it stands, may not prove altogether devoid of interest. In its construction I have earnestly endeavoured to combine pathological and practical observation with physiological fact, and therefrom only to draw conclusions. That the attempt has been a rude one I am quite aware, but it occurs to me that this mode of procedure, however imperfect, is in the right direction, and that by eliciting the knowledge and criticism of others, it cannot fail, in the end, to be in some way serviceable. At all events it is far superior to the system of mere empirical medical observation and argument; a system which not only robs its advocates of much real pleasure, but forms the groundwork of every absurd medical schism, and mightily impedes the onward and truthful progress of a grand and useful science.

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