Pinching in bowels with pain in forehead *at the same time*, off and on; when bowels got *better* the pain in head *ceased*, for half hour (in ten minutes).

Sore throat, chiefly at back, with feeling of contraction when swallowing saliva; the soreness is worse by talking or swallowing solid food, for three-fourths hour (in forty-five minutes).

Cough excited by tickling in throat; causing raw feeling in middle of chest (in forty-five minutes).

Roof of mouth feels sore when the bread touches it; afterwards sore even when not eating, for two and a half hours (after one hour).

At tea time, more appetite than usual (after one hour).

A spot on left chest close to sternum, about the middle, is sore to the touch (in two and a half hours).

(5.) Mrs. —— took a drop in water at 4.30 P. M.

Tingling in both arms and hands (especially in hands), chiefly in the right, as if galvanized; arms feel as if paralyzed, for two hours (in five minutes).

Dull aching pain in forehead over eyes (in ten minutes).

THE "FIBRINOUS CRASIS,"—ITS CAUSE A LOSS OF ALBUMEN FROM THE BLOOD.

In discussing the cause of the so-called "*fibrinous crasis*," or what we prefer to call, simply, an excess of fibrin in the blood, it should be understood at the outset, that this is one of the NATURAL constituents of the blood, furnished to the latter from the chyle, in its proper relative proportion to the other constituents, through entirely *healthy* action, and as fast as the completion of the digestive process introduces into the lacteals the proper materials for its organization; just the same that each and all the other constituents are introduced more or less directly into the blood, either through the lacteals or the walls of the intestinal capillaries, in their proper relative proportion, by entirely healthy digestion and absorption.

And for these reasons, among others, soon to be noted, we must take issue with the claim so universally made by pathologists, physiologists and chemists, that *inflammation* causes the increase of fibrin in the blood, which is so commonly found existing in connection with that diseased action. That there may be no doubts, or misunderstanding, upon these two points, we introduce the subject with the following quotations to elucidate the former assertion, and will then give proper attention to the second proposition.

Kirkes and Paget, in their "Manual of Physiology," say on page 67:

"The development of fibrin appears to proceed commensurately with that of the second set of corpuscles. In the earliest state of the chyle no fibrin exists; but when chyle-corpuscles are formed, the fluid in which they float is spontaneously coagulable; and the fibrin, whose existence is thus proved, appears to increase as the chyle proceeds onwards to the blood, and passes through the lacteal glands. Yet, in the most perfect chyle and lymph the fibrin is less abundant, and coagulates less firmly than in the blood: we may therefore assume that its development, like that of the corpuscles, is perfected in the blood itself."

Carpenter also says on this point, pages 452 and 453, of his "Physiology":

"The chyle drawn from the lacteals that traverse the intestinal walls, contains albumen in a state of complete solution; but it is generally destitute of the power of coagulation, no fibrin being present in it. * * * * * * * * During the passage of the chyle through the absorbents on the intestinal edge of the mesentery, towards the mesenteric glands, its character changes in several important particulars. The presence of fibrin begins to manifest itself, by the slight coagulability of the fluid when withdrawn from the vessels; and while this ingredient increases, the albumen and the oil-globules gradually diminish in amount. * * * * * * * * During the passage of the chyle through the mesenteric glands, a further increase in the proportion of fibrin takes place; and the resemblance of the fluid to blood becomes more apparent. The chyle drawn from the vessels intermediate between these and the central duct possesses a pale, reddish yellow color; and when allowed to stand for a time, undergoes a regular coagulation, separating into *clot* and *serum*. * * * The chyle from the Receptaculum and Thoracic Duct coagulates quickly, often almost instantaneously"; thus showing, of course, that the fibrin has here still more nearly approached its maximum quantity, and also the perfection it attains in the blood.

These statements by Kirkes and Paget, and by Carpenter, are essentially confirmed by all other physiologists. From all this, then, it is clear that there is a special natural function

operating within the lacteal vessels, or mesenteric glands, or both, whose office it is to produce fibrin out of the normal materials furnished to the chyle by healthy digestion; and also that fibrin is an entirely *natural* product of this natural function; one constituent of the blood which is absolutely necessary to the continuance of health and even of life, or such great care would not have been taken to insure the constant repetition of its regular daily production, during the entire lifetime of the individual.

How unreasonable it seems then, to assume and assert, as all pathologists and physiologists do, that fibrin is increased, that is, actually *produced* by inflammation, in any part of the system in which this may arise: In other words, that a *healthy* constituent of animal life can be actually organized, or produced, indiscriminately, by *unhealthy* action; and this too in parts of the system, or in tissues, where we have no evidence that the fibrin-producing function exists. How can such a thing be possible? A healthy and therefore natural constituent of the blood, or a normal ingredient in any department of organic creation, produced by an *unhealthy* and *unnatural* process is an abnormity which Nature must abhor; and an absurdity, upon its face, as it seems to us, for science to pretend to teach.

In saying this, we fully understand that there is great unanimity, as already stated, among all the authorities upon the subject, in asserting that fibrin really is found increased in the blood, or in other words in excess of its natural relative proportion to the other constituents, in inflammation and inflammatory diseases. And let it be borne in mind, also, that it is the *cause* which has been assigned for this result, upon which an issue is here raised, not upon the fact itself, as this must generally, if not always, be as claimed, for the exact conditions exist, *independently* of inflammation as such cause, to *fully* account for its increase on perfectly rational grounds, as we shall now endeavor to prove by some of the best among these very same authors themselves. The essential condition, and one that appears sufficient to establish the whole truth in this matter, is, that there is a *loss of albumen* from the blood, at such times, which simply leaves the fibrin in a relative excess in the serum.

Lehmann tells us on page 618, Vol. I., "Physiological Chemistry": that albumen is *diminished* in the blood "in severe inflammations," and we know from what has already been so fully demonstrated several times, in this Journal, in regard to diseases of the mucous membranes, that when these are the seat of the inflammatory action, and excite any catarrhal secretions, albumen is lost from the blood; hence the former, or albumen, would be deficient in the serum, and necessarily leave *all* the other constituents, *fibrin* included, in a relative excess in the circulation, as compared with the albumen remaining, after the loss of any portion of this has been sustained, as has also, already, been so many times shown in these pages, during the last two years. Besides, if the increase of fibrin, under such circumstances, must be referred to inflammation as the cause, in consequence of its actual production by inflammation, there is no escape from referring the increase of all the other constituents of the blood, found in excess therein at the same time, to the same cause, which would certainly be a great absurdity, in so far as some of those constituents are concerned, to say the least. Let us consider the following in this light:

"The quantity of water in the blood is always proportional to its quantity of fibrin." Lehmann, Vol. 1st, page 616.

And this:

"Almost from the beginning of every acute disease" (inflammatory diseases, of course, included,) "there is an augmentation of the fats in the blood." Lehmann, Vol. 1st, page 620.

Now, we repeat, if the increase of fibrin is properly to be ascribed to inflammatory action as *producing* it, we must also attribute the increase of water, and that of the fats, as shown in these quotations, or the augmentation of the blood-corpuscles, or any of the other constituents, which might be found existing in excess in the blood, in inflammation, (as they all are, except albumen, when this is lost,) to the same cause, and it would only be carrying the idea to its legitimate conclusion to do so. This brings us to speak of pneumonia, that inflammation, or inflammatory affection, which, according to Virchow, Carpenter, Lehmann and Wood, is one of the very few diseases, that shows the most striking increase, or excess, of

fibrin in the blood, of any kind of diseased action. Well, now, what about the loss of albumen in this disease by which to account for such excess, instead of attributing it to inflammation? We will let the following, from "Copland's Medical Dictionary," Vol. I., page 983, answer:

"Viscid, thick, and adhesive sputa, containing *much* albumen, characterize acute inflammation of the lungs."

Here, then, in this simple fact, of a loss of much albumen in the expectoration, we have the most rational and complete explanation of the augmentation of fibrin in the blood in pneumonia, without resorting to any bald assumptions, or speculations, to account for it; and does not this point directly to the only key, namely, loss of albumen, through some one or more of the mucous membranes, for the solution of the long and much-talked-of increase of fibrin in all inflammatory diseased action? especially when Lehmann tells us, as has already been stated, that *albumen* is always *deficient* in the blood in all cases of severe inflammation.

We are, however, by no means limited to inflammatory diseases for cases wherein fibrin is found in excess in the serum. Lehmann, Vol. I., page 319, after speaking of its augmentation in the blood in such diseases, says:

"It is moreover worthy of remark that inflammation in which no fever is present, and likewise mere fevers without inflammation, augment the quantity of fibrin in the blood."

Besides, as is well known, fibrin is found in excess in the serum in albuminuria, during *all* its stages, whether acute or chronic, and this, too, independently of inflammation as its real cause, as what precedes would appear to go a long way toward proving. Though this point, even, we are not compelled to leave to doubt, or to rest upon probabilities, notwithstanding so much of the accumulated testimony, upon the subject, appears, upon its face, to be against us—and one of the most marked instances of which we will now give. Lehmann again tells us, in the paragraph next succeeding the one just given from him, that:

"In other diseases, as for instance in chlorosis, typhus, tuberculosis, Bright's disease, and carcinoma, there seems only to be an augmentation of the fibrin when an inflammatory complication supervenes; in carcinoma, however, certain observations of Popp and Haller appear to indicate that there is a decided augmentation of the fibrin, independently of any inflammatory fever."

This, certainly, is quite direct evidence that an inflammatory complication does augment the fibrin in Bright's disease, as well as in all the other diseases named, except the last; but, if there is anything in scientific truth, the one exception destroys the entire weight of testimony of all the rest. Still as the main assertion therein is so fully supported by all other authors upon the subject, it necessarily must have become in the minds of many, if not all, a formidable point to carry by assault; but mark how easily it is *turned*, and that too by a fact communicated by one of the most prominent advocates of the view we are contesting, namely, by Watson himself. He says, on page 883, of his "Practice of Physic," after speaking very fully of the qualities of the *urine* in all stages and conditions of Bright's disease that:

"In general the albumen is plentiful and almost constant in the outset of the malady. * * * * * * And another fact, which it is essential for you to know and to remember, is, that, in any stage of the disease, the supervention of febrile disturbance, from local inflammation, or whatever other cause, tends to renew for the time, those qualities of urine which belong to the early period."

If this be true, then, and there seems to be no doubt of Watson's entire conviction of its truth, from the earnest manner in which he calls attention to it by saying it is essential "to know and to remember," we have the fact established, that there is a *renewal* of the loss of albumen in the urine, in amount corresponding with the first stage of albuminuria, whenever in *any* of its stages there arises febrile disturbance from inflammatory action. How perfectly and fully this corroborates all that we have before claimed upon this subject. Here we find a marked increase in the loss of albumen *from* the blood, at the very time in the disease, when all observers say there is an increase of fibrin *in* the blood, and assert that to inflammation such increase is due. While we reassert that this augmentation of fibrin is solely due to the increased loss of albumen, which Watson, as we see, tells us occurs in this disease whenever inflammation arises, thereby simply leaving the former, no

less than all the remaining constituents of the blood, in so much excess of what the system can use for nutritious purposes; and this fact which this author furnishes us, is one of the most convincing evidences of the truth of our position that we have anywhere met.

If it is a fact, also, that fibrin is augmented in the serum whenever inflammation supervenes in phthisis, as many authors assert, we find it to be explained upon the same basis as the foregoing, that is, that there is an increased loss of albumen, in this case, through the mucous membrane of some portion of the air passages, by catarrhal secretions, in consequence of inflammatory action arising therein. For have we not already given proof from one of the best authorities, that viscid, thick expectoration, containing much albumen, characterizes inflammation of the lungs? And if so, then is there not an increased loss of albumen in this very identical kind of expectoration, which so commonly arises whenever inflammation lights up in consumption, and at a time, too, when the surplus fibrin is found in the blood? Again we ask, do not these facts fully account for the excess of the latter, upon perfectly rational grounds, in all the cases named belonging to this class, and without resorting to any hypothesis whatever, just the same as the similar facts did the like condition, in connection with albuminuria?

In typhus, which Lehmann also says in the last quotation from him, and in dysentery, which he asserts on page 634, same Vol., show an augmentation of fibrin in the blood, we have the same explanation to offer, for this very author states on pages 537 and 538, Vol. I., that "large" quantities of albumen are discharged from the bowels, in both of these diseases; while on page 618 he gives a list of diseases in which albumen is found *deficient* in the serum, and in this list both typhus and dysentery have a prominent place.

Other diseases in which fibrin has been, or may be, found in excess in the circulation, we leave to the same rational method of solution, fully convinced that future investigation will confirm what we here claim; and show that this, like all other facts in the natural world, has a fundamental principle in nature for its basis, and is not the result of indiscriminate causes.

This brings us to a consideration of the *effects* produced by

an excess of fibrin in the blood. If this constituent is allowed to go on augmenting within the blood vessels, from day to day, and week to week, when albumen is lost in considerable quantity daily, through any of the mucous membranes which may be suffering from catarrhal irritation, the amount of it, beyond what could be used for nutritious purposes, would soon become so great that it would begin to coagulate, or fibrillate, and form clots, or *thrombi*,* within the blood vessels. When this effect occurs, the clots are, of course, generally of the small size, at first, that would only clog the smaller vessels; but they may grow to larger masses, as the fibrin increases in quantity, and soon attain such dimensions that some of the larger arteries, or possibly one or more of the cavities of the heart would be blocked up entirely by them, and thus suddenly produce death, a result which sometimes actually occurs from this cause. But it is seldom, however, that so sudden a termination of life is permitted, for under the protecting care of the vital force, the excess of this agent is expelled from the circulation, evidently to avoid, among other things, such an immediately fatal result. Through the successful efforts of the system in expelling this surplus, we have exceedingly various conditions and diseases produced, which we will now proceed to notice.

Often, one of the first results which arises in albuminuria, from throwing off the excess of fibrin, is the excretion of this into the uriniferous tubules, where it coagulates and forms casts of the tubuli, and is then frequently discharged in that form in the urine. Sometimes, also, we find that a very similar action transpires in the lungs. The excess of fibrin, or a portion of it left by loss of albumen in the expectoration, in diseases of these organs, is occasionally excreted into the bronchial tubes, and there moulded in the ramifications of the bronchi, either as solid or tubular casts of these passages, and then expectorated, sometimes with various prolongations, corresponding with the branching of the tubes.

* See Virchow's very interesting account of thrombi in his work upon Cellular Pathology. Of course, the cause he assigns for them, is very different from that given by us above, except that they have their origin in fibrin, but this does not detract from the interest of his description of them and their effects.

The membrane in *Croup* arises from the same cause. And as this is a condition, or disease, of much more frequent occurrence than the last named, we will give more details in regard to it. Virchow, on page 434, "Cellular Pathology," says of fibrinous exudations and croup:

"If we confine ourselves to those parts, where inflammations with real unquestionable fibrinous exudations do occur, we have a category nearly as limited as that of the mucous inflammations. In such a category the first place is occupied by the serous membranes proper, which even upon slight inflammatory irritation generally produce fibrin; the second place is filled by certain mucous membranes, in which, in a great number of cases, fibrinous inflammations unmistakably arise, as an aggravation out of mucous ones. Ordinary croup does not generally at its very outset manifest itself in the form of fibrinous croup; at the commencement, at a time when the danger may already be very considerable, there is often nothing else found than a mucous or muco-purulent false membrane. Not until after a certain lapse of time does the fibrinous exudation set in, and then it does so in such a manner, that we can trace the transitions in the same false membrane, and see that a certain portion is manifestly mucous, another manifestly fibrin, whilst in a third part it can no longer be affirmed with certainty whether the one or the other is present. Here, therefore, both substances appear as substitutes for one another. Where the inflammatory irritation is more violent, we see fibrin, where slight, mucus, appear."

In Copland's Med. Dic., Vol. I., page 983, we find the following, in regard to the secretions from the larynx in croup:

"A membranous or tubular substance, with thin, viscid or puriform mucus, is often discharged in croup, and consists chiefly of albumen, sometimes approaching the fibrinous state."

These quotations, then, furnish us with the evidence of all the conditions existing in croup, requisite for the construction of the membrane out of an excess of fibrin in the blood, left there by the loss of another constituent. The mucus secretions, and even the muco-purulent false membrane, referred to by Virchow, as occurring in the early period of this disease, before the fibrinous membrane is created, and which we all know to take place as he describes, wastes albumen as its chief organic constituent, even if there was no pure albumen in it, as we have before proved to be the case with *all* mucus discharges. Besides which, we have the direct evidence in this very quotation from Copland, that such discharges "consist

chiefly of albumen" in the first instance, and then approach the fibrinous state. In other words, as we claim, there must necessarily first be a loss of albumen in a more or less pure state, by the mucus secretions, which occur in the early period of membranous croup, and not until this takes place can the fibrin be in excess, to be poured out in a fibrinous exudation. But when it is brought into such excess, then it would be secreted along with mucus, and be mingled in it, producing a membrane of both characters about equal, or predominating in the one or the other, or in a layer by itself, as the case might be, and as the quantity of fibrin thus disposed of might regulate, even to the creating of an entirely fibrinous membrane. How well this accords with Virchow's assertion that, "not until after a certain lapse of time does the fibrinous exudation set in;" that is, not until sufficient albumen has been lost, in the preceding albuminous discharges, to throw the fibrin into such excess that the system cannot dispose of it, in any other way, in each individual case, than by pouring it out as an exudation upon the inner surface of the larynx, where the vital force has already been partly overcome by disease, there to organize or fibrillate into a membrane, or be expectorated in a more fluid form, as circumstances favor. In this sense, and in this alone, as it appears to us, do fibrinous inflammations, or more properly exudations, "arise as an aggravation out of mucous ones," as Virchow claims. And, of course, in our explanation of the origin or cause of the membrane, the fact that, the more violent the inflammatory irritation, the more purely fibrinous it would be, is as fully and more naturally covered than it is by Virchow's theory that fibrin thus exuded is the *product* of the local inflammation, even provided there was any basis whatever in truth for this, but which we have already shown there cannot be.

There are some good reasons for believing, furthermore, that occasionally the excess of fibrin may be exuded upon the mucous membrane of the larynx, causing membranous croup, when albumen is lost from some of the other mucous membranes than that upon which the exudation forms, in a similar manner to its being expelled through or upon the serous membranes, or into fibrous tumors, and there organized,