I have long known that early infancy is accessible to asthma, and that many of the best marked and purest spasmodic cases start from this early date; but, until I examined my cases, I was not aware so large a proportion, nearly one in five, occurred within the first year of life. In some of these the disease appeared so early that it would be difficult to say that it was not truly congenital. Within a few days of birth the breathing had the true asthmatic character. The constitutional tendency must be strongly marked indeed that would develop itself so early. And it is worthy of remark that, in all those cases in which the disease made such an early appearance, there was a history of inheritance, and this is quite consistent with what one would expect; its inheritance would imply its constitutional character; its being constitutional would make it probable that it would early declare itself.

The following is a table showing, in thirty-eight cases in which I have noted this point, the relative frequency in which asthma develops itself in the successive decades of life:—

During the first year	- - - - - 7	} 14
From one to ten	- - - - - 7	
From ten to twenty	- - - - - 8	} 5
From twenty to thirty	- - - - - 5	
From thirty to forty	- - - - - 5	} 2
From forty to fifty	- - - - - 2	
From fifty to sixty	- - - - - 4	} 38
	- - - - - 4	

It would appear from this, that the first appearance of asthma is less and less likely to occur as life advances up to old age, when there is an increase. And this is exactly what one might expect from the constitutional nature of asthma. All constitutional diseases are, *ipso facto*, disposed to declare themselves early; the earlier, the stronger the constitutional tendency; and the longer they pass without appearing, the less likely are they to appear. This table, it must be remembered, is not a table of asthmatics at different ages, but of those who have become asthmatic at those respective ages. It is strictly a table of the relative frequency of the *first access* of asthma.

Almost all the cases of which I have taken notes are uncomplicated cases. Of a large number of cases of senile bronchitic asthma I have preserved no record; if I had, the number of cases commencing between fifty and sixty, and upwards, would have been much larger. The number four in the table by no means indicates the proportionate frequency of the commencement of organic asthma in old age.

VI.—INFLUENCE OF SEX.

In fifty-four cases in which I have noted the circumstance of sex, thirty-six have been in males, and eighteen in females, or exactly twice as many in the former as in the latter. To what does this fact point? It unmistakably asserts that the causes of asthma are circumstances to which men are more exposed than women; such as the inclemencies of the weather, the wear and tear and hardship of life, the stress of violent and sustained respiratory efforts as in heavy labour, and intemperance. Unless this is the cause of the difference, we must suppose it to depend upon some greater proclivity to asthma on the part of the male nervous system than the female. Now this I do not believe. Indeed, I believe the reverse. An asthmatic nervous system is a mobile, sensitive, nervous system, and certainly the female nervous system is more mobile and sensitive than the male.

Consistently with this, I believe it will be found that idiopathic asthma—the pure neurosis—is as common in women as in men, and that the cases by which so large a preponderance is given to men, are cases starting from bronchitis. Senile asthma (chronic-bronchitic and cardiac) is, I should say, ten times commoner in men than in women.

VII.—IS ASTHMA HEREDITARY?

I think there can be no doubt that it is. Not that I would take this fact for granted, or on the common assertion that it is so; for I think this a point on which error is apt to arise. In all diseases a certain number of cases will be found in which, on the mere doctrine of chances, the parents, or other members of the family, have been similarly affected. But I think the number of cases in which there is a family history of asthma is greater than will admit of this explanation. Out of thirty-five cases in which I have noted this circumstance, I find distinct traces of inheritance in fourteen; in twenty-one not. It appears, therefore, to be inherited (and my numbers are sufficient to give some evidence) in two cases out of every five; that is, in the proportion of two to three. The kind of inheritance differs very much, sometimes it is direct, sometimes lateral;

sometimes immediate, sometimes remote; as will be seen in the following specification of the inheritance in these fourteen cases:—

1. Inherited from the father.
2. Father a confirmed asthmatic.
3. Inherited from the father.
4. Father a confirmed asthmatic, brother suffers from hay asthma.
5. Grandfather a confirmed asthmatic.
6. Brother and paternal grandmother asthmatic.
7. Father, two paternal uncles, and paternal grandfather asthmatic.
8. Inherited from father; paternal sister died of it.
9. Father died asthmatic at forty-seven.
10. Several indirect branches of the family asthmatic, but neither parent.
11. Mother slightly asthmatic, maternal grandmother severely.
12. Grandfather now suffers from asthma.
13. Grandfather and uncle both asthmatic.
14. Sister and paternal grandmother asthmatic, brother with hay asthma.

With regard to the inheritance of asthma, I have observed one curious fact, which suggests an interesting general pathological question. It is, that several brothers and sisters in a family may be asthmatic without the parents having been so. This would seem to suggest, in respect to disease, a principle with which breeders are familiar—that certain combinations produce certain results, and lead to the creation of certain peculiarities, and that the qualities of the progeny are not the mere resultant of the combined qualities of the parents—just as we sometimes see a family of red-haired children, both the parents of which have black hair.

[To be continued.]

THE FORMATION OF CLOTS IN THE VENOUS SYSTEM DURING LIFE.

(A THESIS FOR A MEDICAL ACT IN THE UNIVERSITY OF CAMBRIDGE.)

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III.—ON THE CLOTING OF THE BLOOD IN THE CAVITIES OF THE HEART.

In the following case the evidences of the formation of clots in the cavities of the heart and in the blood-vessels were unusually clear, and there can be little doubt that death was due to this cause.

A delicate girl, aged 11, was received into the hospital, March 27th, 1859, with a burn upon the outer surface of the right thigh, and on the palmar aspect of the right forearm. The skin was destroyed over a considerable extent; but it was hoped that she would recover, and, for several days, the wounds proceeded favourably, under the application of flour. About April 8th, without any apparent cause, she became rather feverish and refused her food. The slight feverishness subsided, but left her very feeble, with small pulse, dryish tongue, and shrivelled, harsh, scaling skin, the finger-ends feeling as after scarlet fever. Wine and nutritious diet were administered, and some improvement took place. On the morning of the 17th she was very low, but enjoyed her meals more than on previous days; and the nurse thought her better. About four in the afternoon she was somewhat uneasy, and asked the nurse to sit by her; but nothing remarkable was observed till about two hours afterwards, when she asked to be raised up in the bed. As soon as this request had been complied with, it was perceived that she was dying, and in a few minutes she was dead. No marked difficulty of breathing or other especial symptom was noticed.

Fifteen hours after death we found the right cavities of the heart, the left ventricle, and all the vessels connected with these cavities, filled with fibrinous clots. The left auricle also contained a clot, but was not filled by it. The central and greater parts of the clots presented the ordinary characters of the fibrinous masses often found in the heart, though they were tougher and firmer than usual. They were not laminated. Under the microscope they exhibited very fine linear fibres, disposed in an irregular network. The exterior of the clots presented, however, in many places, quite a different appear-

ance. It had a more opaque white, or cream colour, something like lymph in the neighbourhood of an abscess, and was composed, apparently throughout, of well formed cells, larger, clearer, and less granulated, than pus cells, with distinct nuclei. This cream-coloured portion of the clot was, in the right auricle, separated by a defined line from the remaining buff-coloured part, and could easily be peeled off from it. It had not quite a smooth external surface, and adhered slightly to the interior of the cavity, particularly in the appendix; nevertheless, it remained with the clot when the latter was turned out of the auricle. In the ventricles it adhered to the lining membrane of the heart more closely than in the auricles, especially about the edges of the valves, and near the apex. Where the clots extended into the pulmonary artery and aorta, they were deeply impressed by the sigmoid valves; but were not adherent to them.

Near the bifurcation of the pulmonary artery the whole thickness of the clot had a dull cream colour, and this was the case in the primary divisions of the vessel. In no place were the clots adherent to the walls of the pulmonary artery. Fibrinous strings, presenting the usual appearance, extended into the smaller branches of the artery.

In the inferior cava and the iliac veins was some fluid blood, which coagulated on exposure to the air; and mingled with it were numerous small clots, coloured in different degrees, and some of them partly coloured. They appeared to be free in the fluid blood, or, if adherent to the walls of the vessels, their connexion was very slight. In the great veins which converge to the superior cava, the clots were more numerous, larger, and more fibrinous, but still loose in the tubes, or very slightly connected with their walls. The left jugular and subclavian veins, at and near their junction, were almost filled by yellowish or cream-coloured clots, having a peculiar coiled or wrinkled exterior, which must have been caused by their being subject to some movements during the flow of the blood, or to their having been formed or increased by smaller clots carried in the current from other parts, and intercepted there. None of the vessels were distended by the clots.

In all the arteries which I examined, except the aorta and pulmonary arteries, the blood was fluid. The heart itself was quite sound, and there was no disease of the internal organs.

I have seen other cases more or less similar. In none of them, however, did the symptoms and the appearances after death so clearly indicate that death had been caused by the clotting of the blood in the cavities of the heart and in the adjacent great vessels. They form a highly important class of cases, to which the attention of pathologists and practical men is being more and more closely directed, and for the clear appreciation and discrimination of which much information as to the causes influencing and regulating the coagulation of the blood within the body, before and after death, has yet to be sought. At present, I think we are unable to explain many of the appearances we are in the habit of observing, and of deciding with certainty whether they are to be referred to *post mortem* changes, or whether they commenced during the life of the patients. For instance, we often find the cavities of the heart (the right auricle and ventricle more particularly, the left ventricle not unfrequently, the left auricle rarely) filled with fibrinous clots, and the prolongations of the clots extending into the adjacent vessels and descending into the most dependent branches of the pulmonary arteries; so that the absence of colour in them does not admit of explanation upon the ordinarily received hypothesis of the subsidence of the red corpuscles during the slow coagulation which goes on in the recently dead body, because the lowermost portions of the clots in the several cavities, and the most dependent portions in the pulmonary arteries, are as devoid of colour as the uppermost parts. Nevertheless, it seems pretty certain that clots of this kind are, for the most part, formed after death. Coincident with these fibrinous clots is sometimes a variable amount of fluid blood, which may be in the same cavities as the clots; commonly there is some fluid blood in the left auricle. Not unfrequently the clots extending from the ventricles into the pulmonary artery and the aorta exhibit well marked impressions of the semilunar valves, showing clearly that they were moulded upon the valves; and this is regarded as evidence of their having been formed before death (Richardson *On the Coagulation of the Blood*, pp. 407 and 420). Yet these clots are almost invariably found not adherent to the valves, but quite free from them and smooth. Moreover, each one commonly bears the impression of each of the three valves in an equal degree; and this often happens on both sides of the heart in the same body. If, therefore, these clots formed, as is sup-

posed, or commenced, before death, upon the valves, they must have lined the whole of the interior of the vessels at and near their connexion with the heart, and must have completely covered and impeded the action of all the semilunar valves in such a manner and to such an extent that it would scarcely have been possible for the circulation of the blood to have been continued at all.

I think that additional observations upon the coagulation of the blood in the dead body, under varying circumstances and after various diseases, must be made before we can agree with the author of the excellent work *On the Coagulation of the Blood*, that either the tubular or the laminated structure of a clot, or the fact of a clot consisting of a fibrinous tube containing red blood in its interior, is conclusive evidence of the coagulation having begun before death. Lamination is a singularly prevalent and remarkable feature in the inorganic, as well as in the organic world; and the circumstances under which it occurs and the causes which induce it would prove a fertile subject for study and experiment. It is doubtless more commonly observed in those clots which, there is good reason to believe, have formed before death, than in those which have commenced subsequently. It would seem to depend upon the deposition of the fibrine in successive layers; though that is not certain, inasmuch as it may depend upon some peculiarity in the manner of the solidification of the fibrine, the particles becoming disposed in plates and layers, just as we know they have a disposition to be arranged in fibres; and it is no unreasonable supposition that this may, under favourable circumstances, take place after death. With regard to the tubular form of a clot, it is by no means improbable that, in certain peculiar conditions of the blood, the tendency of the fibrine to separate itself and settle upon the walls of the vessels, on the valves, and on other structures, may be manifested after death, in the same way that it often is before death; and may thus lead to peculiarities in the appearance and structure of the clot which render it very difficult, or impossible, to distinguish whether they are *ante* or *post mortem* formations.

The mode in which the *fibrinous* and the *corpuscular* varieties of the fibrine are combined in the same heart, or even in different parts of the same clot, are difficult of explanation. Thus in a lad, who died with cerebral symptoms after amputation, I found in the right auricle and ventricle a firm white clot, which presented, under the microscope, a distinctly fibrous structure, the fibres being clear and delicate, but strong; and in the left ventricle were small reddish clots, containing an unusual quantity of pale nucleated corpuscles. When the two varieties coexist in the same clot, the fibres generally preponderate at the circumference, and the corpuscles nearer the centre: the reverse, however, was found in the clots occupying the heart in the case above related.

CASE OF POISONING BY SIR WM. BURNETT'S DISINFECTING FLUID: RECOVERY.

By HENRY PORTER, M.D., Peterborough.

I was summoned at six o'clock on the morning of February 26th, to go five miles into the country, by a message to the effect that "Mr. F. had swallowed something by mistake, which had made him very sick and ill," but with no more definite statement. I therefore at once proceeded to visit my patient; and, on inquiry, found that he had returned home late on the previous evening from a dinner party, when, according to custom, he had gone to a cupboard where a bottle of Dinneford's Fluid Magnesia was usually kept, for the purpose of taking a dose, as a corrective to his port-wine. Unfortunately, however, on the present occasion, a bottle of Sir Wm. Burnett's Disinfecting Fluid had been placed by the side of the fluid magnesia, resembling it so much in size, shape, and colour of the label, that he at once put it to his lips, threw his head back, and, taking a full inspiration, swallowed a draught, amounting at least to two ounces, before perceiving the mistake. "At once," says my patient, "I felt a burning sensation all down my throat, with great pain and a sense of tightness in my stomach. My wife directly prepared me an emetic of mustard and water; and in less than four minutes I was violently sick, ejecting all the food I had taken, as well as the fluid I had swallowed; and I have been in excessive pain up to the present time": which was about seven o'clock A.M. when I first saw him.

He then had that particularly anxious expression of countenance indicative of irritant poisoning, with a small, quick,