ON

PHLEGMASIA DOLENS: ITS ORIGIN & ITS CONNEXION WITH ERYSIPELAS AND OTHER INFECTIOUS DISEASES.

BY ROBERT J. LEE, A.M., M.D.

IT is important that the pathology of phlegmasia dolens, so far as its practical treatment is concerned, as well as its probable connexion with certain infectious disorders, should be clearly understood by those who are required to give attention to patients attacked with it. Some recent writers on this subject leave the reader in doubt of the origin of the affection, and the mode in which the condition observed after death is produced; so that a confusion of ideas exists in the minds of those who have not had much clinical experience of this disease, and have enjoyed no opportunity of pathologically investigating it. To some extent, the introduction of new terms imperfectly defined contributes to this result, as will be pointed out in the following remarks.

In the first place, the difference should be clearly established between coagulation of blood in veins and in arteries, and a more definite meaning ought to be attached to the terms "embolism" and "thrombosis" than is conceded them by some pathologists. In the second place, it is proper to distinguish between cause and effect when considering the origin of coagula in the vessels of the circulatory system—that is to say, whether the formation of coagula be the primary disorder or simply arises from morbid changes of tissue, productive, among other results, of mechanical interference with the free circulation of blood through the vessels of the part affected.

With regard to the relation which has been suspected to exist between the causes which give rise to erysipelas, puerperal fever, hospital gangrene, phlegmasia dolens, and other forms of disease of similar nature, it must be allowed that we are unable to demonstrate beyond all dispute the identity of their origin. But we may fairly inquire whether we ought not to be satisfied with something less than material proof when we reflect upon the obscure and mysterious character of this class of infectious diseases, and, in the interests of humanity, whether we ought to reject presumptive evidence when it is all that the most careful investigation furnishes as our guide to practical conclusions.

These remarks are introduced as a prelude to a case of phlegmasia dolens to which the attention of the reader is invited, as it is one of great interest from the circumstances connected with its origin, and from the rare and serious form which it assumed. The unfortunate lady who was the subject of it was engaged in the anxious and arduous duties of matron to the Westminster Hospital. With more zeal than prudence she neglected the first warnings of constitutional disturbance which preceded the attack of phlegmasia, and which was occasioned by long and frequent visits to wards in which several patients were suffering from erysipelas of serious character, among whom were two of her own nurses, the especial objects of her attention. It may be mentioned that both in the medical and surgical wards there were cases under treatment; those in the former had been admitted for erysipelas, those in the latter were attacked by it during their stay in the hospital. It was in the late part of last year that this tendency to the occurrence of erysipelas showed itself, outside the hospital as well as in the wards. It was clearly noticeable that the slighter cases were not productive of ill effects upon the nurses or adjoining patients, but that it was from the severe cases, and particularly from one that was fatal, that the infection was spread.

The details of the matron's illness combine to produce one of those rare and serious instances of phlegmasia of the lower extremities in which the favourable progress of the patient is suddenly interrupted by the occurrence of the fatal symptoms of plugging of the pulmonary artery and coagulation of blood in the right side of the heart, which are referred to the detachment of a portion of clot and its impaction in the pulmonary vessels. The disease showed itself first in the right leg, in a few days subsided, and then attacked the left leg, and later again the right; but at the end of three weeks the patient was making favourable progress when the symptoms occurred which in a few hours terminated in her death. It was immediately after changing her position in bed that the palpitation of the heart and distress in the respiration were experienced. The very first sensation which preceded these symptoms was a peculiar movement, as though something had rapidly passed from the left inguinal region directly to the heart; and so distinct was the account which the patient gave of the occurrence that the question arose in the minds of Dr. Fincham and Mr. Holthouse whether it could not fairly be referred to the detachment of a portion of clot and its arrest either in the right side of the heart or pulmonary artery. To what extent we are justified in directly connecting the symptoms with the change of position in the body upon which they followed is deserving of consideration, as the same cause has produced the same result in other cases, and indicates the necessity of making a patient observe the greatest tranquillity, as well as of preparing the friends of a patient for so sudden a catastrophe. It may also be remembered that in this form of phlegmasia dolens the origin of the disease is not to be referred to the uterus, as is the case in uterine phlebitis, but is probably the same as in those cases of phlegmasia which occur occasionally in the male sex.

The lectures of Trousseau contain a few of the most apparent reasons for supposing that there is some connexion between erysipelas and phlegmasia, but no individual instance is recorded of the latter disease having followed exposure to the influence of the former, and although a single case may contribute but imperfect evidence that such is possible, it is sufficient to direct attention to a careful investigation of the subject.

Between cases of simple phlegmasia and "idiopathic" erysipelas there is a strong resemblance in the sequence of symptoms and the particular tissues affected. There is this difference, however, that in the former the superficial cellular tissue, in the latter the deeper structures, are the seat of disease. With respect to the coagulation of blood in the veins, it is not difficult to demonstrate that it is the consequence of inflammatory changes in the connective tissue surrounding the vessels. If in a case like the one under notice a careful examination be made of the condition of the cellular tissue in the vicinity of the termination of the coagulum, it will be found that generally for a distance of from one to two inches above that point there is evidence of the change to which the term "cellulitis" is applied. The best method of demonstrating this is to dissect the artery from the vein with a scalpel till a slight induration of the tissue is observed and consequent adhesion of the vessels, so that greater force is required to separate them. The earliest evidence of the change referred to is always found above the point where the coagulum in the vein terminates; and, as I have frequently investigated the subject, I believe I am justified in supporting the view that the change of external tissue precedes and is the cause of the coagulation of blood in the veins as it advances in the direction of the heart; that it may likewise spread down the veins is highly probable; and that the effect of the inflammation of the cellular tissue, as it follows the course described, is gradually to arrest the venous circulation, occluding vessel after vessel in its progress more or less completely, the coagulum forming first in the minute veins and subsequently in the larger. If we take that part of the principal vein where the coagulum terminates, and, passing upwards, examine the orifices of the vessels which open into it for some distance above, we shall find that a coagulum has already formed in them, and that the terminations of the clots they contain project into the larger vessel, although the coagulum in the latter has not yet reached the same point.

In the case of phlegmasia which forms the chief subject of these remarks, the aorta and the vena cava were easily separable as far as their division into the common iliac branches. In this situation the vessels were surrounded by tissue, in which the earliest changes of cellulitis were observable. On the right side the artery and vein were firmly adherent where the division of the common iliac vessels takes place, and where the internal joined the external vein was found the termination of the coagulum, which extended down the latter vessel to those of the whole lower extremity. On the left side the vessels were affected in the same way as high as Poupart's ligament, but above were quite per-

vious, and the cellular tissue in the pelvis presented a perfectly natural condition.

When I stated in the early part of my remarks that certain terms were used inappropriately by some pathologists, I referred more particularly to the use of the term "thrombosis" in the case of all kinds of clot as the cause, and not the consequence, of previous change of tissue. It may be desirable to retain the word, but with the necessary predicate defining the cause of the clot; for "thrombosis" means simply "a clot," and nothing more. I need not point out the necessity of such addition to the term, as it may be applied to the coagulum in an aneurismal sac, in the termination of a ligatured artery, to the consequences of an embolism, and to other mechanical causes; and when I say that by the introduction of such terms as "embolism" and "thrombosis" a confusion of ideas has arisen, I wish to imply that the opinion entertained by some that the clotting of blood in veins is the cause of phlegmasia is erroneous, and that they overlook the real origin of it, thus mistaking cause for effect.

Maddox-street, March, 1872.

CARDIAC INTERMITTENCY.*

BY J. MILNER FOTHERGILL, M.D.,
SENIOR RESIDENT MEDICAL OFFICER, LEEDS PUBLIC DISPENSARY.

CARDIAC INTERMITTENCY is the last and most serious rhythmical disturbance of the heart, and is a symptom ordinarily of very grave import. It is evidence of great disturbance in the balance of forces by which the heart is regulated. This disturbance may occur in any of the three modes previously mentioned.† It is unnecessary to go over them again, and at present we will regard the symptom rather as it manifests itself clinically. Before thus leaving the question of causation it may be desirable to consider the question of the immediate mode of its production. Dr. B. W. Richardson, in his paper in the St. Andrews Graduates' Transactions of 1870, states that the arrest of ventricular action lasts over the period of a second auricular contraction; and further states that the distension produced by the second auricular contraction excites the ventricle to contract. When first read, this statement appeared to the writer as ingenious but totally unproven. That it was probable was unquestionable, and that the labouring ventricle should need an unusual stimulus to excite it to contract was quite in unison with views expressed elsewhere. It is the case however. But even a second auricular contraction is not always sufficient, and three, or may be more, may be required in cases where the intermittency is marked; for intermittency is merely an aggravated form of irregularity, differing indeed only in degree. The proof of this the writer got quite unexpectedly in pursuing some experiments on the rat to test the time of auricular and ventricular contraction in the mammal. The rat having been carefully chloroformed and the chest opened, at first the contractions went on normally, the auricular contraction immediately preceding the ventricular contraction; but as life flagged, two auricular contractions were requisite to excite the less irritable ventricle, and finally three auricular contractions were necessary. The ventricle needed goading; and firstly a second auricleful of blood became necessary, and finally a third, to produce such distension as should excite contraction. As to the method of immediate production of intermittency, Richardson's views are sound. As to the conditions under which it occurs, the writer finds it impossible to agree with him. Having abandoned the view of its cerebral origin advocated by him in 1867, Dr. Richardson has taken up the view of its being solely connected with exhaustion of the sympathetic. This view is a great improvement on the first; and while admitting that cardiac intermittency is due frequently to exhaustion of the sympathetic, the writer feels compelled to express his dissent from the view of this being its sole, or even its usual, causation. The chart‡ already given, which was drawn up before the writer became acquainted with Dr. Richardson's later views, gives the modes of disturbance of balance, and the various methods in which it may arise. At present the question will be regarded with reference to its appearance clinically, and the conditions under which it is found. Giving them in the order of relative frequency, commencing with the most frequent condition, cardiac intermittency is found with—1, fatty degeneration; 2, muscular exhaustion; 3, exhaustion of the sympathetic; 4, influences through or in the vagi.

1. *Fatty degeneration.*—With degeneration of the muscular structure of the heart itself is intermittency most usually associated. It is under these circumstances found accompanied by a feeble first sound and subjective symptoms of cardiac failure. Thus in the generally degenerate condition of the circulatory organs known as atheroma, where the fatty condition is a necessary result of imperfect blood-supply and consequent tissue-degradation, intermittency is very frequent. And in the aged, with rigid arteries, fatty cornea, &c., when, amidst a number of feeble heart strokes, the long halt of intermittency is detected, occurring almost rhythmically, a most grave prognosis will be found best suited to the case, and a too early verification of it will often be met. The wearied muscular fibres, constantly becoming more scanty and unequal to the distension of the rigid and inelastic aorta, and thus constantly tending to a less and less sufficient blood-supply for themselves, become more and more tolerant of distension till, finally, a slumber too prolonged for the continuance of the existence of the organism results in cessation permanently. Thus, when in serious dilatation the imperfect contraction, and consequently imperfect aortic recoil, leads to degeneration of the muscular tissue in addition to its elongation, amidst the flutter of irregularity will be found the ominous halt of intermittency. This is a point of great practical importance. Except in great exhaustion, the muscular fibre is never tolerant enough of over-distension to allow of intermittency unless its own integrity is interfered with. When, then, intermittency is found along with other objective and subjective symptoms of failure of power, it is a symptom of the greatest gravity.

2. *Exhaustion of muscular walls.*—Next in frequency is muscular exhaustion as a cause of cardiac intermittency. Exhaustion of the muscular walls is often closely allied to exhaustion of the sympathetic. But in cases of aortic obstruction, &c., there is no exhaustion of the sympathetic unless after effort, and then the intermittency is more frequent: it is a simple disturbance of balance of power between the muscular walls and the obstruction offered to the flow of the blood. The two most marked cases of cardiac intermittency—i. e., the most prolonged halt—which have come under the writer's notice were both cases of aortic disease. In these cases the halt extended over a period of time equal apparently to three or four ordinary ventricular contractions. The exhausted muscular fibre claimed a long rest before it could be again goaded into activity. The sensations of the patients during this prolonged ventricular systole were of the most painful description. One is a man of great intelligence and of a nervous temperament. His account of his sensations is most affecting. On the most vivid sensations of instant death is implanted a sense of acute agony. A look of horror passes over his face when even mentioning his sufferings. All sensations which are connected with parts supplied by the sympathetic are exquisitely painful, and the over-distension and inability of the ventricle to contract seems to instantly pass all over the system. During periods of temporary irregularity of the heart's action the writer has had occasion to note the unpleasant sensation which accompanied the ventricular arrest. During the prolonged halt in these two cases this sensation amounts to agony. The frequent instantaneous halts in the walk of the aged, and the instinctive clutching or snatching at some one, or even anything which could afford support, which are usually found connected with some lesion of the heart, are now relegated in the writer's mind rather to the list of unpleasant sensations of sudden failure from ventricular inability than referred to sudden temporary cerebral anæmia. That this may be produced by sudden cerebral anæmia mediately as a result of ventricular failure it is not attempted to deny, but the cerebral anæmia and the sudden sensation of impending death, or other catastrophe, are alike results of ventricular inability. Relief by rest in bed, and the proper therapeutic measures by reducing the demand on the muscular walls (so many

* Palpitation, THE LANCET, Aug. 6th and 13th, 1870; Cardiac Irregularity, Dec. 10th and 17th, 1870

† See chart in THE LANCET for Dec. 17th, 1870.

‡ See THE LANCET for Dec. 17th, 1870.