and two conditions most favourable for the formation of clots in the heart are present; viz., a rough surface, furnished by the trabecular work of that organ, and retardation of the blood-current. Of all parts of the heart, the appendages of the auricles are the most frequent seats of thrombi, owing to the blood being with great difficulty completely expelled from them; and in the notes of the necropsy of the case, it is stated that the left auricular appendage contained an *ante mortem* clot, which must have been of some age, as it had undergone softening in the centre. In consequence of the thrombus in the left auricle breaking down into minute puriform particles, the latter became mixed with the blood which flowed into the left ventricle, and were sent during its systole into the systemic circulation. When these particles passed into vessels which were too minute to allow them to escape, they more or less completely plugged those vessels, and in this manner it is extremely probable that the splenic and renal infarctions were produced, as also the gangrenous spots on the back of the right hand. The peculiar wedge-shape of the splenic infarct, with the apex of the wedge towards the centre of the viscus, was due to the mode of distribution of the vessels, and its yellowish-white appearance to the colouring matter of the blood having been withdrawn from it, or to the decomposition of the hæmatine, and to the fibrine having undergone fatty degeneration.

The kidneys were larger than those generally met with in granular degeneration of these organs; but whether this condition, and the large quantity of albumen which the urine contained during life, were partly produced by the renal infarctions, I am unable to say.

I have now endeavoured to account for the hypertrophy of the left ventricle, the *ante mortem* coagula in the heart, and the hæmorrhagic infarctions of the spleen and kidney. I now proceed to consider the mode in which the pulmonary apoplexy was produced.

It has been shown by Virchow and others that, in all cases of pulmonary apoplexy, whether secondary to valvular disease of the heart or not, the first step in the genesis of the morbid change is embolism of a minute branch or branches of the pulmonary artery. Virchow has proved this experimentally, by introducing particles of fibrine, muscle, and elder-pith into the jugular veins of dogs, and examining the lungs after death, when he found that these foreign bodies had blocked up branches of the pulmonary artery, and produced, among other results, hæmorrhagic infarctions beyond the points obstructed. You will remember that, on *post mortem* examination, *ante mortem* coagula were stated to have been found both in the right auricle and ventricle; in fact, the thrombus from the auricle extended through the auriculo-ventricular opening into the ventricle. Well, it is my opinion that minute portions of these clots were washed into the pulmonary circulation, just as the granular matter found its way into the systemic circulation from the left side of the heart; and I would here remark that the circulation through the right side of the heart was sufficiently vigorous for the blood to be sent to the minutest ramifications of the pulmonary artery. When, therefore, minute portions of these clots arrived at pulmonary arterioles too minute to allow them an exit, they produced embolism and subsequent extravasation of blood.

The mode in which the hæmorrhage is produced subsequently to the plugging of the vessel is variously interpreted; but the theory of Ludwig, founded on the "influence which the contraction of an artery has upon its capillaries", appears to me to be by far the most feasible explanation. I will quote his words from the American translation of the seventh edition of Niemeyer's *Text Book of Practical Medicine*, vol. i, p. 156: "Tension within the artery below the point of constriction is diminished, since a liquid flowing through a narrow tube loses more of its impetus than in flowing through a wide one. But we must not infer from this that, when an artery is constricted, the contents of its capillaries are lessened, and that the parts which they traverse grow paler. The sluggishness of the stream thus produced in the capillaries rather has the effect of allowing the heavy blood-corpuscles to collect and become crowded together; now, as two or more blood-corpuscles, if brought into contact, are apt to become permanently adherent, the blood itself can form a plug capable of closing the capillaries. Such an occurrence, which converts the capillaries into blind appendices to the artery, must cause an increase in its internal pressure." "Let us add," says Niemeyer, "that, in consequence of the pressure, which, after the stoppage, is as severe in the capillaries with their delicate walls, as in their afferent vessels, a rupture of the distended walls and an escape of blood ensue..... The utmost that can be advanced against the accuracy of this explanation is, that the artery leading to an infarction not only is narrowed, but entirely closed. Such an objection, however, is untenable. The infarction of an embolus, which we find usually takes place at the bifurcation of an arteriole, very rarely produces absolute closure of it at first, but merely causes in it more or less obstruction. Afterwards when the infarction

has become established—an event requiring but little time—fibrine is deposited upon the embolus, and closure of the vessel becomes complete."

There is only one other point on which I will say a few words, and that is connected with the treatment of the case. A day or two after the patient was admitted, and when I was going round the wards with my pupils, I found the man's face of a purplish hue and bathed with perspiration; he was gasping for breath, and the pulse at the wrist could scarcely be felt. His extremities were cold. He was also expectorating bloody sputa. The heart's sounds were scarcely audible. What was I to do under these circumstances? One thing was quite certain; viz., that the man would die unless I could speedily relieve him. It occurred to me that the immediate cause of his distress was a surcharging of the right cavities of the heart, producing a state approaching to paralysis of that organ, and that it was just possible that cupping might relieve him. On the other hand, I had to contend with this difficulty; viz., that if I cupped him I should increase the coagulating power of the blood, and thereby the tendency to the formation of clots. I, however, asked the House-Physician to cup him, and the result was most satisfactory, for, after the operation, he was able to lie down in bed, and breathed more easily. The patient expressed himself as feeling decidedly relieved. He lived four days longer.

THE SPECIFICITY OF ACUTE DISEASES.*

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MORPHOLOGY and physiology are so intimately connected together in all organic beings, that it is doubtful (as Mr. H. Spencer has shown) whether any functional change can take place in an organism without a corresponding alteration of its structure; and modern science would lead us to assume that, whenever we find a change in the vital operations of an organ, there is, or has been, a corresponding change in its textures, whether this be apparent or not. Starting with such a doctrine, it is easy to perceive how the various phenomena accompanying the loss of health have been divided and subdivided according to the method of their serial development, and how the present system of classification of diseases has arisen. It appears to me, however, that there now reigns a large amount of confusion on the subject of the differentiation of diseases, from our knowledge being based rather upon hypotheses than on facts. What I mean by this statement, I now hope to explain by reference to the so-called specific diseases. Such diseases are supposed to originate in the system by the introduction of a "material principle, the product of a specific morbid process" (*Traité de la Contagion*, par Dr. Anglada, Paris, 1853, t. ii, p. 12), and many clever and able writers have upheld this doctrine. The latest investigations of Chauveau, Sanderson, Bastian, and others have failed to show that there exist any specific elements in the product of such lesions which differ in form and external characters from analogous elements met with in simple inflammatory lesions. Therefore, if there be a difference in the two (to quote M. Chauveau's words) "it must be in their intimate qualities and active properties." Our only means of investigating such "intimate qualities and active properties" is to observe the results of the introduction of such specific agents into the animal economy, and to see whether there be an agreement between the phenomena thus produced; and, if so, whether it be only general or differing with different agents. Such investigations can only be obtained by actual experiment; and, as we are mostly debarred from making such on the human subject, and experiments on the lower animals are open to many great objections, it will probably be a long time before this difficulty is solved. There is, however, one source of information open to us on this subject; and that is the process of vaccination. If it can be shown that on the introduction of vaccine into the human frame a progressive series of phenomena arises, definite and similar in all cases, and yet that this series invariably differs from all others induced by other agents, we shall have a strong argument in favour of the specificity of diseases in general. If, on the other hand, no changes take place after vaccination, which differ from such as may be induced by inoculating the system with another organic poison, then the hypothesis of specificity (as far as vaccination is concerned) is not yet proven. Before we consider the process of vaccination, however, it may be as well to review the subject of inflammatory skin-diseases generally, and to ascertain in what way, if in any, the typical vaccine vesicle differs from all other skin-eruptions. In tracing the course of hyperæmic or inflammatory skin-affections, we cannot

* Read before the Abernethian Society.

well overlook their general resemblance in one signal respect—namely, in the sequence of the phenomena accompanying their tissue-changes. Let us take, for instance, as an example, the pathological effects of a common burn on the cuticle. The first objective symptoms are redness and slight swelling of the integuments; in other words, an erythema is produced. These symptoms are of short duration, the epidermis is detached, and the inflammation ceases. This sequence of events is similar whatever be the cause of the erythema. If, however, the action of the heated substance be more violent, or the tissues be left to its influence for a prolonged period, a secondary group of phenomena begins. The epidermal layers of the integument are raised from the deeper portions by the intervention of a layer of serum. A bulla or vesicle is formed. This effusion of lymph (as it is called) is due to the transudation through the capillary walls of the liquid and (to some extent) corpuscular elements of the blood. As, in the first instance, it would be almost impossible to determine from the symptomatology of the erythema what had induced it, so, in the second case, were one ignorant of the cause, there would be nothing in the appearance of the bulla, nor in the manner of its local formation, to justify a guess at its etiology. A physician, however, would be in a position to assert that the bulla, if rapidly produced, had been preceded by a local hyperæmia of the skin, and he might further safely affirm that such a bulla had never contained pus; in so doing, he would (unconsciously, perhaps) admit that there was a definite sequence in such pathological tissue-changes.

If we pass on to the pustular form of eruption, we shall find that there are many reasons for believing this to be due to the reaction of the tissues after a still more prolonged or severe irritation than that necessary to induce the vesicular stage. The ordinary vesicular skin-eruption named eczema may be produced equally by the action of external irritants (Neligan's *Cutaneous Diseases*, Eczema) as by the so-called idiopathic causes. If this eruption be preceded and accompanied by an excessive hyperæmia, the vesicles become charged with a turbid greenish-yellow fluid (in other words, pus), and the sero-purulent eczema impetiginodes is the result. As the vesicular eczema may under certain conditions be transformed into an affection closely allied to, if not identical with, the pustular impetigo, we find other vesicular eruptions (*e.g.*, those of herpes, variola, varicella, etc.) pass under favourable circumstances into the pustular form. If the pustules be long in maturing and be situated on the hair-follicles, their apices become umbilicated (Hebra, *Skin-Diseases*, n. s. s., vol. i, p. 237), as is frequently seen in variola.

If we now consider the form of the typical vaccine-eruption, we shall perceive that it is characterised by the maturation of an umbilicated vesicle on a reddish base about the eighth day after the puncture; but in its appearance, and in the sequence of the pathological phenomena observable during its formation, there is nothing dissimilar to what may arise during the formation of any other vesicle. It is, therefore, clearly necessary for us to search elsewhere for the essential points of distinction between this and all other eruptions. The next necessary step in our investigations will be observations on the intimate constitution of these and other vesicles. M. Chauveau, Drs. Sanderson, Hallier, and Bastian have patiently and fully investigated the microscopical appearances of inflammatory products; and although they differ in the details of their work, I think we may now accept this much as proven—namely, that in specific inflammations we find no elements which differ in form, shape, or physical constitution from these found in ordinary inflammatory affections. Of course, it may be possible hereafter, by the aid of still minuter investigations or of hitherto undiscovered reagents, to discriminate between specific and simple inflammatory elements; but at present this is not the case, and I do not think we are more justified in forestalling this branch of knowledge by wild speculations than we are with regard to any other science. We thus find that, in its progressive formation and in its intimate constitution, there is no one character which we can assign to the vaccine vesicle as peculiarly its own. In what way, then, does this vesicle differ from all other vesicles? We are told that it is in its results—that it prevents the evolution of a certain specific virus, *viz.*, that of small-pox, in the bodies of those who have felt its influence. In order that we may test the truth of such supposed results, let us consider the aspects of the disease it is considered to forestall. We have seen that in the present state of science we are unable to isolate the virus of vaccinia; it is, therefore, needless to expect to be able to do so in the case of small-pox.

The experience of Chauveau and Sanderson on the subject tend (as far as I can ascertain) only to prove that, in the case of lymph carefully diluted by the addition of a layer of water to its upper surface, the weak and watery superficial layers, although containing a certain amount of albumen and salts, are almost innocuous as regards inoculating powers; whereas the deeper and less diluted layers possess in a

greater or less degree the active properties of the original fluid lymph, results which might *à priori* have been expected. We must, then, content ourselves with the consideration of the phenomena attendant on a typical case of small-pox; as such, however, are ordinarily described in all text-books upon medicine, and are thus necessarily well known to the members of the Society, I shall not waste the time of the meeting by describing them. I may, however, mention that the actual time of incubation of the disease, upon which so much stress is frequently laid, is exceedingly difficult in practice to estimate. The period at which the eruption appears, its characters, and the stage in the local tissue-changes at which defervescence takes place, are all subject to great variations: these are facts which any medical man may ascertain for himself during the prevalence of the present epidemic. We thus find, owing to the variability of the symptoms attendant on small-pox, a disease which the vaccine vesicle is considered to prevent, that even in its results the properties of such a vesicle are doubtful. Admitting that the inoculation of vaccine checks the operation of another morbid poison on the animal economy, it is at all events admissible to inquire what the poison is that it so checks. As yet, however, we must content ourselves with watching the phenomena of successive tissue-changes in such cases, and with accepting the doctrine of their origin by means of a morbid poison, without asking a question as to its truth.

The next subject which I wish to consider is, what the definite sequence of events observable (as I before stated) in these and similar local eruptions implies. In the simple erythema we see certain changes occurring in the nutrition of a part, a certain excess of the natural waste of tissue (as usually shown by a slight desquamation) coincident with an excess of the reparative material introduced by the local hyperæmia. In the second (or serous exudation) stage, we find a corresponding greater loss of tissue (the elevated cuticle) associated with an increased supply of the reparative plasma, *viz.*, the contents of the bulla. Finally, in the third and last stage, a still greater loss of tissue necessitates the most violent efforts on the part of the surrounding tissues and blood-vessels at repair, and pus is formed. The rapid formation of plasmic elements in the last case appears at first sight to defeat its own object, if that is, as I interpret it to be, the repair of the lost tissues. We must, however, remember the prime necessity that exists in all such structural changes to remove effete or waste material prior to any attempt at repair. If, owing to the large amount of such dead matter at one time, the neighbouring structures be unable to remove it by the ordinary processes attending the natural waste of tissues, a rapid solution of continuity between the dead and living tissues becomes necessary, and this is possibly best done by the abundant evolution of plasmic elements possessing slight, if any, cohesive properties. When the violent reaction attendant on the removal of such effete material has in some degree subsided, the plasmic elements more slowly formed become adherent as granulations, and eventually, as lowly organised connective tissue, replace the structures lost by the slough. This, then, is my interpretation of the processes accompanying acute inflammatory lesions of the integument. There are certain phenomena which occasionally present themselves at one or the other of the above stages of skin-eruption, and they must therefore be looked upon as accidents depending upon peculiar conditions of the surrounding tissues. Such, for instance, are the *hamorrhagic cutanea* of Hebra and Neligan, and to some extent, possibly, the *ulcerations* of the former writer. During any of the three previously noticed stages of cutaneous eruption, a rupture in the hyperæmic vessels may arise, and an extravasation of blood takes place into the surrounding tissues. Such an event in the hyperæmic stage will induce petechiæ, vibices, or ecchymoses; less frequently it is seen in the later inflammatory stages as in the pustules of variola nigra. The acute general erythema observed in the early stages of some of the so-called malignant fevers is usually accompanied by such extravasations. The cessation of simple hyperæmia is usually marked by the loss of the outer layers of the epidermis (as before noticed) in the form of a bran-like scurf, a mealy powder, or scales. In pityriasis, lichen, psoriasis, and lepra, the principal local objective symptoms consist of recurrent patches of hyperæmia, followed by a partial resolution and the discharge of the superincumbent epidermal scales. Here, again, we only see an exaggerated activity of the natural functions of the cutis leading to the constant renewal of the epidermis.

I shall not have time to pursue this part of the subject more fully to-night; but I trust that I have shown the Society sufficient facts to establish the truth of my proposition, *viz.*: that, as regards the integument, an excessive waste of material will have a tendency to induce in an organism a correspondingly vigorous attempt at repair.

It will be necessary now to pass to the inflammation of the more deeply seated tissues. If the erythematous affection produced by a burn, as before noticed, extend inwards to the cutis and subcutaneous tissue, it is called an erysipelas. The objective symptoms—the redness,

heat, and swelling—are intensified. The rapid exudation of serum into the affected parts may produce both bullæ superficially, and that deeper seated infiltration of the tissues characterised by “pitting upon pressure.” When the inflammatory affection is violent, the same sequence of events occurs as in the superficial diseases of the skin; the evolution of plasmic elements is in excess of that necessary to supply the ordinary waste of the parts—in other words, suppuration is established. In the resolution of acute inflammation, if the process have not arrived at the evolution of the semi-solid pus-corpuscles, and the tissues return (as far as one may judge) to their condition prior to the attack, we may suppose the reparative material, although in excess of what is natural, is in a form that can be readily reabsorbed by the blood, and excreted from the body, if necessary, by an increased activity of certain special organs. The interdependence of tissues and organs throughout the body makes it probable (although we have no means of ascertaining this) that every pathological tissue-change, however trifling, exerts an influence on the system at large. During the violent reactionary process of phlegmonous erysipelas, the whole body becomes, according to Virchow, more highly charged with fibrine and plasmic elements than it does in health. Knowing what trivial extraneous circumstances may induce inflammation of the skin, it is not a matter of surprise that under these conditions secondary deposits of pus should occasionally take place in different parts of the body. If we examine the viscera of a person who has died of pyæmia, we shall probably find them the seat of two distinct lesions—localised patches of hyperæmia, and localised deposits of pus and tissue-debris. If we refer to the sequence of phenomena observable in inflammation of the cuticle, we are forced, I think, to conclude that a similar sequence occurs in both cases. It so happens that pyæmia is usually ushered in by rigors, headache, quickened pulse, hurried respirations, furred tongue, loss of appetite, great thirst, and so forth—symptoms “which,” to quote Mr. Savory’s words, “are collectively expressed by the common term fever,” and it is not surprising that this disease should sometimes have been confounded with the so-called exanthems. The difficulty of diagnosis is still further increased by the frequency with which those fallacious “landmarks,” skin-eruptions, are seen in this disease.* It thus happens that we find it sometimes simulates typhus,† at others typhoid fever,‡ § glands, and small-pox;§ and it is only on the appearance of the metastatic abscesses that the diagnosis can be confirmed. If, in case of the non-specific disease pyæmia, we probably find similar causes produce such dissimilar effects as these data show, are we to assume that every definite series of phenomena attendant on an acute specific disease is necessarily induced by the action of a peculiar poison? Would it not be quite as consistent with our present knowledge, if not more so, to ascribe these deviations in detail to the known interdependence of the organs and tissues of the body, their anatomical arrangements and peculiarities; and the possibility that, when any change of the nutritive balance in a part takes place, this change may press with more or less severity on other parts, according to the situation of the prime lesion, its importance, and its correlations?

I have thus far, gentlemen, attempted to show you that to my mind the doctrine of the specificity of acute diseases requires a thorough revision before it will bear that full investigation which science demands us to make, ere we admit an hypothesis (such as this) as an article of professional faith. Before concluding, however, permit me to say a few words on the action of those material poisons and drugs with which we are all acquainted. It is easy to perceive that the hypothesis of zymotic diseases has been strengthened by the known action of poisonous drugs upon the animal economy. Substances which can be seen and handled when introduced into a living being, give rise to a series of tolerably definite phenomena differing with the nature of the drug. I say advisedly “tolerably definite phenomena”; for I think most gentlemen will admit with me that, in cases of poisoning with the same drug, great variations are occasionally observable in the symptoms induced. It must be remembered that, besides the quality of the poison, the quantity given and the mode of administration have each definite effects on the results produced. A drug which stimulates, if it be taken in small quantities, will narcotise in excessive doses, or induce great nervous exhaustion if the time be prolonged over which its influence on the system is felt. The quality, or rather chemical constitution, of two poisons may greatly differ in their nature, and yet they may be productive of similar effects on the system. But these

effects are not really comparable with the phenomena attendant on acute specific disease, unless we can actually demonstrate the “material poison” which has induced the latter. It is not necessary, as we can at all times ascertain for ourselves, to seek the cause of such organic changes in gross materialism; the external forces of nature, heat, cold, electricity, and so forth, or such internal forces as originate in man’s own nature, from simple privation of food and drink, are sufficient to induce quite as great changes in our bodies as any we have been considering; and why may they not possibly play their part in these?

THERAPEUTIC MEMORANDA.

USES OF HYDROCHLORATE OF AMMONIA.

In Dr. Murchison’s lecture (No. 10) upon dropsy, under the head of “Diuretics,” he remarks that these remedies “have no effect upon portal dropsy.” In the list of diuretic remedies, however, Dr. Murchison omits any mention of the hydrochlorate of ammonia. Now, whether it be by its direct action as a diuretic, or by its immediate action as a cholagogue, I have for many years found the hydrochlorate of ammonia one of the most efficient of our remedies in cases of portal dropsy, prescribed in scruple or half-drachm doses, given, tolerably diluted, every six or eight hours. The recent accounts of the success attending the employment of the hydrochlorate of ammonia in hepatic affections in India will, I trust, lead to its more extensive use in this country; as yet it has scarcely received the attention it deserves, partly, perhaps, on account of the great drawback, its unpleasant taste.

SPENCER THOMSON, M.D.

Ashton, Torquay, June 1872.

CLINICAL MEMORANDA.

TEMPORARY HEMIPLEGIA AND APHASIA.

L. M., aged 29, in the eighth month of her third pregnancy, wife of a labourer, after a week of unusually hard work at washing, had been sewing almost all day, when, in the afternoon, she suddenly experienced a numbness in the right hand. She rose to call her husband, but fell into a chair, and then found her head was giddy, and that the right foot and the whole of the right side were numb and felt cold. The right side of the face was drawn down. There was not any unconsciousness; she understood everything said, but could not speak for fifteen minutes, at the end of which she spoke as well as before. Her hand was useless for an hour, and her foot dragged during the remainder of the day. She had never before had a fit of any kind. During the last two years she had occasionally found an apparent thick mist, coming on suddenly and quickly becoming total darkness, before both eyes, the right especially. In this obscurity, she had seen vibratory and momentary curved lines, showing the colours of the rainbow; these “darted backwards just like shooting stars,” until they disappeared. These attacks had lasted about a quarter of an hour; in one of them, a year ago, she appeared to see bright sunshine with varied hues, when the sun was not shining. The perversions of vision had been usually succeeded, sometimes preceded, by lancinating headaches, principally confined to the left temple, and never accompanied by sickness; occasionally there had been no headache before, with, or after. The cardiac sounds were normal. The eyes had not been examined ophthalmoscopically; the pupils acted well, and were of equal size. The patient had not noticed any relation between the attacks and menstruation; they usually followed hard work, and began by dimness all over the field of vision. There was not any œdema.

The patient was quite well from the evening after the seizure until labour came on at the full time, on April 7th. I then first saw her, and found that pains had commenced sixteen hours previously. Four hours later, a living male child was born; the patient had a very good convalescence. Since the birth of the child she has had several feelings of numbness and coldness of the right hand and foot, with giddiness, sometimes lasting some hours; her speech has not been again affected, and she has not found any deafness nor disturbance of vision.

Her mother became epileptic, in consequence of sudden fright on her brother being drowned, six years before the patient’s birth; the epilepsy used to be very frequent, and still recurs. The father has never had any illness. An elder and a younger sister (both dead), and three brothers (all now living), all born after the mother became epileptic, have been free from any affection of their nervous systems. The youngest child, a sister aged 19, has had epileptic fits since childhood.

June 28th, 1872.

STANLEY HAYNES, M.D., Malvern.

* See the Difficulty of Discriminating between Varicella and Pemphigus (Gee, *St. Bartholomew’s Hospital Reports*, v, p. 122).

† Murchison, *Continued Fevers*, p. 190 (1862); Savory, *Statistics of Pyæmia (St. Bartholomew’s Hospital Reports*, iii, p. 80); *Surgical Lectures of Abernethy*, quoted by H. Lee (*Lectures on Practical Pathology*, i, p. 5).

‡ H. Bennett, *Principles and Practice of Physic* (third edition), p. 894; and a case in *BRITISH MEDICAL JOURNAL*, recorded by Mr. Nunn, May 20th, 1870.

§ Wilks, *Guy’s Hospital Reports*, vii, p. 153.