advanced cases, and is usually regarded as being a sign of more value than cutaneous pigmentation. It may be found along the line of contact of the lips, on the cheeks, gums, or tongue, especially if there be any cause of irritation, such as a carious tooth. It is said to occur in the vagina⁵⁹. The conjunctivæ⁶⁰ may in some cases, though very rarely, be pigmented.

Situation of the Pigment on the Mucous Membrane.—According to Dr. Greenhow⁶¹ the pigment in the tongue is found in the same layer as in the skin, namely, the stratum Malpighii or mucosum. Haddon⁶² found the pigment quite as plentiful in the corium as in the stratum mucosum of the buccal mucous membrane.

Tizzoni⁶³ in the pigmentation produced experimentally in the buccal mucosa of rabbits, found pigmented carrier cells in the corium, and pigment in the deeper layers of the stratum mucosum.

Dixon Mann⁶⁴ draws a sharp distinction between pigmentation of the mucous membrane and that of the skin. Pigmentation of mucous membrane is not general; it requires certain local conditions (friction, local hyperæmia) for its development, and the pigment has a different histological site. The corium contains pigmented carrier cells, as is the case in the skin, but the stratum mucosum contains no pigment, possibly because the cells composing it do not have the finger-like processes⁶⁵ which in the skin are thought to receive the pigment from the carrier cells. When, exceptionally, pigment was found in the stratum mucosum it was seen to be not in, but between, the cells.

Pigmentation of the Serous Membranes.—Addison⁶⁶ described discrete pigmentation of the peritoneum in one of his cases, and a similar case is mentioned by Dr. Wilks.⁶⁷ Dixon Mann⁶⁸ has described the same appearance; the intestines are described as being otherwise normal, and no mention is made of any old peritonitis or tuberculosis.

Dr. Greenhow⁶⁹ records a case of Addison's disease with pigmented tubercle of the peritoneum, accompanied by signs of past peritonitis. The same writer also quotes⁷⁰ a case recorded by Van Andel of pigmentation of the peritoneum, but no mention is made of the presence or absence of tubercle or of past peritonitis. As its occurrence is so rare, it is probable that when present pigmentation is due to past inflammation, and probably of a tuberculous character of the peritoneum. No instance of similar pigmentation in the pericardium or in synovial membranes has been recorded. The marked pigmentation of the pia mater observed by the late Dr. Carrington⁷¹ seems to be unique, and might possibly have been due to the melanæmia of ague.

OTHER ANATOMICAL LESIONS MET WITH IN ADDISON'S DISEASE.

In some of Addison's cases a hypertrophied condition of the lymphoid follicles in the stomach and intestines, as well as some enlargement and softening of the spleen, were noted. The exaggeration of the lymphoid tissue has been frequently noted since, and Dr. Greenhow⁷² considered it as one of "the characteristic, though perhaps not quite invariable, lesions" of the disease. There does not appear to have been any diarrhoea in the cases in which this condition was marked. It might, perhaps, be considered that this condition of the lymphatic follicles is to some extent in favour of the view that when the suprarenal bodies are functionless, whether from destruction or atrophy, some toxin is circulating in the body. The occurrence of leucocytosis after the injection of bacterial poisons or even the injection of albumen⁷³ is presumably accompanied by increased activity of the lymphoid tissue of the organism. In some cases persistence of the thymus gland has been recorded.

A softened condition of the brain with an increased amount of cerebro-spinal fluid has been recorded in some cases⁷⁴ of Addison's disease, and was probably accidental, but its occurrence is of interest from the marked inflammatory changes resulting from removal of the suprarenal bodies in animals in Tizzoni's hands.

Tizzoni⁷⁵ quotes two cases of Addison's disease in which lesions of the central nervous system were found: 1. Bourredi's case: Caseous change in the suprarenal bodies associated with hyperæmia of the cord, perivascular inflammation, degeneration of nerve fibres, and changes of varying

degree in the ganglion cells. 2. Semmola's case: A normal condition of the suprarenal capsules accompanied by mucoid degeneration of the stroma of the abdominal ganglia of the sympathetic and small cell infiltration around the central canal of the spinal cord.

In connection with the view that Addison's disease is of the nature of a toxæmia, it would be natural to expect to find changes corresponding to those described by Bowman⁷⁶ in the spinal cord in pernicious anæmia.

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THE PATHOLOGY OF OEDEMA.¹

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(Concluded from page 637.)

WE come now to the consideration of the œdema which accompanies inflammation. This œdema has long been regarded as the result of an injury to the blood vessel walls, whereby they allow fluids to exude with more ease than normally. That exudation from the blood vessels occurs as the result of a stimulus which is sufficiently powerful to impair the vitality of the tissues, though stopping short at actually destroying them, there is no doubt. The question we have here to consider is the reason for that exudation. Now the explanation of the œdema of inflammatory origin as dependent upon damage to the vessel wall really depends upon the fact that injury to the tissues is known to occur, and it is assumed that the blood vessels are injured as well. No doubt this is true, but if the exudation occur from damaged blood vessels it is necessary to prove that this exudation takes place in the actual region of damage. This, however, is not the case; the blood vessels which exude the fluid, namely, those which are actively congested, are at

¹ The expenses of some of the experiments referred to in this paper were in part defrayed by a grant from the British Medical Association.

some little distance from the initial seat of injury, and therefore are least likely to be damaged. Moreover, in the case of passive oedema I have over and over again in frogs made microscopical examination of the small blood vessels, veins, arteries, and capillaries, and in no case have I found any evidence of alteration from the normal either in appearance or in their behaviour to staining reagents.

If we dismiss from our minds for the moment the question of damage to the vessel wall, and consider the phenomena accompanying the exudation in inflammation, we see that there is no line of demarcation between the physiological and the pathological. If the hand and arm be placed in warm water at 40° C., they become red, the arteries are actively dilated, and presumably the lymph flow is increased.² The reason is that the increased temperature leads to increased metabolism, and increased metabolism means an increased need of nutrition. This increased nutrition can only be carried out by means of a greater interchange of blood, and that interchange by active dilation of the arteries. So far everyone will allow the phenomena are physiological. Increase the temperature of the water somewhat, and inflammation occurs; the phenomena are exactly the same, but more marked. It is not necessary to call in the action, or rather the want of action, of a vessel wall; the increased need of the tissues is amply sufficient to explain the excessive amount of exudation, as has been seen in the case of passive oedema. Further, inflammation is an essential part in the process of repair, and repair is a physiological and not a pathological process.

For these reasons, I would suggest that the oedema which accompanies inflammation should be regarded as beneficial in object, though excessive by reason of the excessive degree of starvation of the still living tissues. The processes whereby it is brought about are exactly similar to those occurring whenever the supply of food is not equal to the demand, and I therefore think it unnecessary to introduce a hypothetical injury to the vessel wall.

That the vessel walls themselves are modified in some way within the area of activity of the cause of inflammation there can be no doubt. They must suffer neither more nor less than the tissues around them. In other words they must be starved also; but this is not quite the same as saying that they are damaged, though no doubt the line between starvation and damage is a difficult one to draw. That their function is in some way modified by their condition of starvation is probable, though we have no evidence of the fact beyond the alteration in the nature of the fluid which leaves them. The oedema fluid in inflammation is of higher specific gravity, and contains more albumen than does ordinary lymph, and this we may perhaps regard as a sign of modified function, but we can go no further at present. Heidenhain's work on lymph formation, however, goes far to prove that the lymph is secreted, and it is easy to understand that, if such be the case, variations in composition of the lymph may be brought about by modifications in the nutritive condition of the secreting cells.

There is, however, the phenomenon of stasis and the adhesiveness of the white blood cells to the vessel wall, which is regarded as evidence of an alteration in the condition of the vessel wall. But such appearances are only those compatible with a slackening of the flow and a thickening of the blood. They do not prove that there is a modification of the vessel wall any more than the deposit of precipitate on a filter after the filtrate has passed through shows a modification of the filter paper. In both cases fluid leaves the containing vessel, which is of lower specific gravity than the mixture in that vessel; the material left behind is of higher specific gravity and the particles are more closely packed together. A greater force therefore is necessary for the propulsion of the thickened material than before. In the case of the blood vessels this is not forthcoming, and as a natural consequence the blood comes to rest. The adhesiveness of the white cells is also explicable in a very simple manner. Normally, the white cells being of lower specific gravity than the red, they occupy the plasmatic layer of the blood; slacken the flow somewhat, and they are enabled to remain in contact with

the wall in doing which friction also comes to their aid. But the very fact that they are less readily detached than normal as a result of the slower stream of blood, causes the numbers at any given moment and at any given place to be greater than normal. This, however, is not evidence of a modification of the vessel wall. It were equally just to ascribe the altered condition to a perverted disposition on the part of the white cell.

The evidence, therefore, of injury to the vessel wall as being the cause of the exudation in inflammation being unsatisfactory, it seems best to turn to phenomena which are known to exist. Increased exudation to meet increased demand for food we have already seen to occur with increased metabolism as well as in passive oedema, and since it adequately explains the oedema of inflammation, it is reasonable to regard this last as the endeavour of normal but actively dilated blood vessels to supply the need for increased nutrition in a part where such nutrition is imperatively necessary. Here also I would urge that it is the tissues which determine the occurrence, and not primarily the blood vessels.

There remains now for consideration the dropsy which accompanies disease of the kidneys. Of this there are three distinct varieties, namely, acute tubal and glomerular nephritis, chronic interstitial nephritis, and lardaceous disease, and corresponding to these there are forms of oedema which have somewhat different characters, and therefore it is advisable to consider them separately.

The whole question of renal dropsy was exhaustively treated of by Dickinson in a paper read to the Royal Medical and Chirurgical Society in 1892, but inasmuch as Dickinson treated the subject principally from the clinical point of view, for my present purpose I cannot refer so fully to the paper as its value deserves. He sums up, however, his view so far as concerns acute renal dropsy in the following words: "It cannot be doubted that renal dropsy is essentially a matter of increased exudation, not diminished absorption, and that of this the capillaries are the seat. The process is connected probably with some change in themselves or in their relation to the blood associated certainly with increased pressure behind them."

Speaking in the broadest way, it is undoubtedly true that in renal disease the oedema varies inversely with the amount of urinary secretion and inversely with the power of the heart; there are no doubt exceptions to these two rules, but they are in the main true. Now, I have made some experiments that bear upon the first of these two points.³ It was necessary in my experiments on the passive variety of oedema to produce anæmia of a dog's hind limb. This I did by means of an Esmarch's bandage. The effects upon the limb in question at a later part of the experiment have already been considered; let us now consider the effects upon the body to the exclusion of that limb. Instead of now holding the normal amount of blood and lymph, that amount was now increased by the fluid removed from the anæmic limb, and therefore there was a condition of plethora, produced, be it remarked, in a much simpler manner and one less open to objection than methods hitherto employed, in that the extra fluids thrown into the circulation were absolutely similar to those already in the body. The results were unexpected, for within a very short time—under half an hour—the tissues such as muscle and skin fell in specific gravity to a marked extent, or in other words became slightly oedematous. It is unnecessary here to go further into particulars, but this experiment, which I have confirmed in other ways, shows that the volume of the circulating fluid in the body is a matter of great importance, and if this volume be increased the tissues immediately take up a certain amount of the plasma and reduce the volume towards the original. If we now consider the bearing of this experiment, we may easily see how in acute renal disease, in which the renal secretion is to the greatest extent interfered with, the volume of circulating fluid is necessarily increased, assuming the intake of water be not regulated. If, therefore, the tissues act as a reservoir for the water, it is not astonishing that the amount of dropsy varies inversely as the amount of urine.

Probably, however, the question is not quite as simple as

² Later experiments have proved that the exudation of lymph is greatly increased. The subject of inflammatory oedema is still under investigation.

³ *Proc. Physiol. Soc.*, May, 1894.

might be supposed from the preceding words. It has been seen that a most important factor in the pathology of passive oedema is the storage in the tissues of the metabolic products of tissue change; it cannot be doubted that in renal disease this storage must also come in. But the removal of these waste products is hindered by the condition of the kidney in acute renal disease no less than is the removal of the water. Moreover, since they and the water are circulating in the blood over all the body, it follows that the whole of the tissues must be exposed to their influence. Now in the absence or abeyance of the excretory system the possibility of the equalising of supply and demand on the part of the blood and the tissues is out of the question, for if there exist a demand in the tissues, and the circulatory system endeavour to meet it in the usual way by active congestion, the effect is that not only is more lymph thrown into the part, but at the same time a proportionate increase of the same waste products which the tissues are so anxious to get rid of is unavoidably thrown in also. Under these circumstances it is hardly to be wondered at that the amount of dropsy in renal disease is excessive, that it comes on in so short a time, or that it is generally distributed over the body. In acute renal disease, therefore, it seems probable that two factors are at work both of which occur in the normal physiological condition—namely, an exudation of the watery part of the blood in order to keep the volume constant, and also an absorption with the object of removing the stored-up waste products. In the latter case, however, it is the demand of the tissues primarily that leads to the oedema, whereas in the case of plethora it is difficult to see exactly what part of the organism initiates the transudation, whether blood vessels or tissues.

In the same way it is intelligible that increased force of the heart's beat and increased tension (which means increased output of the ventricle) should lead to a diminution of dropsy. For both these factors are favourable to a re-establishment of the renal secretion, and therefore to a readjustment of the balance between the contents of the blood vessels and the amount of fluid in the tissues on the one hand, and the formation and the removal of the waste products of tissue change on the other hand.

The characteristics of the oedema which accompanies chronic interstitial disease of the kidney suggest that in some degree there is a difference between it and the oedema which accompanies acute parenchymatous nephritis. The distribution of the dropsy is similar, it is true, in many cases though not in all. In chronic interstitial disease the oedema frequently takes on a "cardiac" type—that is to say, it frequently shows itself to a considerable extent in the legs before it is more than indicated in the face. The explanation of this, no doubt, as has long been recognised, is that the dropsy is truly of cardiac origin, and depends upon failure of the hypertrophied left ventricle. To this result it is probable that the anæmia so frequently noticed in the subjects of chronic granular kidney materially contributes. Owing to the polyuria which accompanies this form of kidney disease, it is perhaps doubtful whether the effect of an increase in the volume of the circulating fluid (such as probably exists in acute renal disease) upon the occurrence of dropsy comes into play. It is frequent, however, to find that when these subjects of chronic renal change become affected to a considerable degree with oedema, the amount of urinary secretion has become diminished. So also the dropsy is greatest in those cases in which an acute parenchymatous change has been engrafted upon the chronic interstitial change. Both of these facts render the effect of the chronic disease upon the volume of the circulating fluids doubtful. The excretion of urea, however, is certainly diminished in most cases, and this, together with the anæmia and the failure of the left ventricle, which invariably supervenes if the patient live long enough, forms a collection of causes sufficient to explain the occurrence of dropsy, while the abnormally small amount of urea excretion probably increases the amount of urea in the blood, and therefore poisons the tissues in the manner already pointed out. That the amount of urea (or, speaking more generally, nitrogenous waste products) in the blood is actually increased in chronic granular kidney is not absolutely known from experiment, however probable it may appear. If it occur it is

ample reason for the general disposition of the oedema which distinguishes renal dropsy from cardiac dropsy.

The dropsy of lardaceous disease of the kidney has always been a subject of much uncertainty on account of the marked differences which occur, especially in the cardio-vascular apparatus, between this and other forms of Bright's disease. Judging from the fact that the lardaceous material is deposited in the coats of the blood vessels, and that it therefore causes narrowing of their lumen, which, moreover, is not compensated for by cardiac hypertrophy, it seems probable that the dropsy in lardaceous disease depends upon the anæmia of the tissues. To this it must be added that the causes of lardaceous disease, namely, prolonged suppuration and syphilis, are particularly likely to induce a cachectic condition, and, therefore, to bring the tissues into a devitalised or starved condition. It must also be remembered that the lardaceous change is a general one, not confined to the kidneys, and that, apart from the dropsy of lardaceous renal disease, which is often really the dropsy of acute parenchymatous nephritis imposed upon a primary lardaceous condition of the organ, there is a general doughiness of the skin met with in the subjects of this particular change which is not usually termed "oedema" but which is certainly akin to it, if not a more extensive but more indistinct manifestation of it. The acute parenchymatous, chronic interstitial, and lardaceous diseases of the kidney are certainly the most important, if not the only, causes of renal dropsy, and with these I will conclude what has necessarily been an imperfect sketch of the whole subject of oedema. Other but less important and rarer conditions in which dropsy occurs I do not propose to mention, as they probably do not differ materially in pathology from the forms already given.

There remain two small points, however, which are of interest, and they are the following: Whence comes the fluid which finally constitutes the oedema fluid of a part? and why do the skin and muscles, but particularly the skin, show to the naked eye and to touch evidences of oedema to the exclusion of other parts?

Taking these questions in order, it is evident that the oedema fluid comes immediately from the blood, but this is not all, as can be seen on closer examination. In my experiments on localised oedema I have considered parts other than the oedematous limb and have found that not only does the specific gravity of the muscle and of the skin fall in the oedematous part, but also that the specific gravity of the muscle and of the skin in the rest of the body rises. The tissues, therefore, of the body at large give up water to the blood to supply the drain on the blood that is taking place in the region of oedema—a further proof of the importance of the tissues in the pathology of oedema. It is probable that the method by which this outpouring of lymph from the muscles and skin into the blood takes place is the converse of that which occurs in plethora, as we have already seen, but it is also possible that the needs of one particular part which requires food (the part which is becoming oedematous) is able to make known its wants to the rest of the economy, whereby other parts are able to assist in meeting that excessive demand.

In the second question it is a matter of common experience that oedema is usually recognised exclusively in the skin. So far as I am aware no observations have been made in the *post-mortem* room on the specific gravity of the tissues, but clearly there is room for work in this direction. More particularly it is necessary to know whether in cases in which there is marked oedema the muscles share in this oedema. In experimental cases there is no doubt of the fact, and in all probability it will be found that the muscles of the human subject in cases of dropsy are below the normal in specific gravity. The fact remains, however, that oedema is principally manifested in the skin and muscles; and yet the whole body must suffer from malnutrition in disease of the heart or kidneys. Moreover, one would be inclined to think that organs such as the brain and heart, which are particularly sensitive to any alterations in the quality or quantity of their blood supply, would feel the effects to the greatest extent. The brain can ill bear starvation, and hence, its demand being greater than that of the skin, one would think that the brain ought to become oedematous sooner and to a greater

extent than the skin, if œdema depend upon starvation and poisoning of the tissues. Voit's researches upon the proportionate losses in weight of the various organs in starvation offer an explanation, however, of this apparent paradox. It will be remembered that Voit's experiments show that those organs which are of the greatest importance to the economy are nourished in prolonged starvation at the expense of those organs which are of less importance. Thus, the brain and spinal cord lose only 3 per cent. of their weight while fat loses 96 per cent. Now, in the starvation which leads to œdema the same law no doubt holds good, and since the brain, for example, gets its full complement of food when the heart is failing or when there is renal disease, it follows that not only must the less important tissues, such as fat, skin, and muscle, get a diminished supply, dependent upon the cardiac or renal cause, but that that diminution must be further augmented by reason of the deficiencies which are being made up in the brain. The brain, therefore, does not suffer, but the skin, etc., suffer doubly. It is no cause for wonder, consequently, that œdema manifests itself principally in the skin.

It would be reasonable to expect that in renal disease the requirements of the brain should not be so fully met as in cardiac disease, on account of the greater degree to which poisoning enters into the question in renal disease. I speak with great reserve on this point, but I think the brain in renal disease is seen *post mortem* to be "watery," whereas such a condition is uncommon in other diseases. The question can be properly settled only by direct investigation.

The last point to which I wish to draw attention is that in 1889 Wooldridge gave reasons for believing that the œdema of cardiac disease depends upon poisoning by "tissue fibrinogen." He injected into the veins a solution made by extracting the tissues, and obtained œdema of the lower limb after ligaturing the femoral vein. Under normal conditions ligature of this vein does not cause any trace of œdema. It is obvious that what Wooldridge did in his experiment really was to increase the amount of metabolic waste products in the body, and to confine them to some degree in the lower limb by ligature of the vein. Under these conditions we have already seen that œdema occurs. Though the meaning of Wooldridge's experiment was unfortunately not seen during his lifetime, it was undoubtedly the first experiment which pointed to the importance of the storage of waste products in a part upon the occurrence of œdema in that part.

To sum up: 1. There are certain experiments which show that œdema does not depend upon purely mechanical causes even in the cardiac variety in which those causes have hitherto been deemed sufficient. 2. There is no evidence that the blood vessels are damaged in any other way than that which constitutes starvation. 3. Anæmia of the tissues, meaning thereby an insufficient supply of blood to carry out their proper nutrition, and the removal of waste products has been shown by experiment to lead to œdema. 4. Poisoning of the tissues by waste products is a more potent cause of œdema than pure anæmia. 5. Increase of volume of the circulating fluids in the body leads to an absorption of fluid from the blood by the tissues. 6. The processes occurring in œdema have their counterpart in the normal physiological state. 7. In both the pathological and the physiological conditions the tissues become œdematous, or tend to become so, as the case may be, through the medium of an active dilatation of the arterioles supplying the part. 8. The tissues of the body other than the affected part are drawn upon to supply the excessive amount of fluid which is being poured out in any particular region that is becoming œdematous.

Upon these facts I would base the view of œdema as being an excess of the normal process whereby the nutrition of the tissues and the removal of their waste products are carried out, this process being initiated by the tissues and not by the vascular system.

THE next meeting of the German Dermatological Society will take place at Graz on September 23rd and two following days. Among the questions proposed for discussion are the following: The Pemphigus Question; and the Relation of Tertiary Syphilitic Manifestations to Treatment in the Early Stage.

NOTES ON FIFTY-EIGHT CASES OF HÆMORRHAGIC DIPHTHERIA.

BY
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DURING the years 1893-94 there have come under our care at the Western Fever Hospital, Fulham, a large number of cases of diphtheria which presented the symptom of hæmorrhages into the skin and from the various mucous membranes.

This variety of the disease is but scantily described in the literature of diphtheria, though most writers on the subject have recognised its extreme malignancy. The occurrence of cutaneous hæmorrhages is of especial value as a prognostic sign in cases in which the issue is otherwise doubtful, and it is more particularly for the purpose of describing this symptom that we record the cases which have come under our notice.

From January 1st, 1893, to January 31st, 1895, 880 cases of diphtheria have been treated at this hospital. Of these cases 58, or 6.59 per cent., presented cutaneous hæmorrhages, all of which, with one doubtful exception, proved fatal.

Sex and Age.—These 58 cases comprised 27 males and 31 females, at the following age periods:

0 to 5 years	24	29 years	1
5 to 10 years	27	41 years	1
10 to 15 years	4	65 years	1

Local Symptoms.—These were all cases easily recognised clinically as diphtheria from the presence of false membrane in considerable quantity (except in 3, in which there was but little membrane present), and acquired early a severe local character. In the majority of cases the false membranes, together with the surrounding and subjacent mucous membrane, became the seat of hæmorrhages of greater or less extent, and soon underwent decomposition, in spite of energetic local treatment. All the cases presented membrane on the tonsils, uvula, and soft palate, while in addition 18 were laryngeal, 3 nasal,¹ 2 glossal, and 2 nasal and laryngeal. Although so large a proportion presented laryngeal symptoms, the obstruction caused was only sufficiently severe to call for operative interference in 3 cases.

Other Symptoms.—All the cases had marked cervical adenitis, in many of great severity; in only 1, however, did the inflammatory process lead to suppuration. Another symptom common to all was profuse rhinorrhœa, and albuminuria was present in every case in which examination could be made.

HÆMORRHAGES.

1. *Cutaneous.*—These presented two well-marked varieties, which, however, in a few instances assumed an intermediate type.

(a) *Ecchymoses* closely allied to ordinary traumatic bruises, varying in colour from a deep purple to a light green or brownish yellow. It was noticeable that the colour bore but little relation to the age of the ecchymoses, and gave a false impression of their duration to one not familiar with this characteristic. The shape was generally circular, with a tendency to oval elongation; the area from about 2 lines in diameter in the smallest to 1½ inch in the largest. They were in some cases palpably raised, and numbered from 2 to 20. In 23 cases they occurred on the face, in 36 on the extremities, being, as a rule, more marked on the extensor surfaces, but not uncommon in the flexures, and in 43 cases on the trunk, the lumbar and sacral regions being the most common site.

(b) *Purpuric Spots*, varying from minute petechiæ to hæmorrhages the size of a split pea. They were in some cases almost innumerable, in others not more than half a dozen were present. In 23 cases they were noted on the trunk, in 9 on the extremities, and in 1 case on the face. Unlike the ecchymoses, they showed a preference for the flexor surfaces.

These two varieties were not uncommonly combined in the same case, this occurring in 16, while in 25 cases ecchymoses alone occurred, and in 16 purpura. In 1 case the

¹ The term "nasal" is applied to those cases in which membrane was visible in the nasal passages.