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SELECTED MONOGRAPHS

COMPRISING

ALBUMINURIA IN HEALTH AND DISEASE.

BY

Dr. H. SENATOR.

SOME CONSIDERATIONS ON THE NATURE AND PATHOLOGY OF TYPHUS AND TYPHOID FEVER.

BY THE LATE

ALEXANDER P. STEWART, M.D.

MOVEABLE KIDNEY IN WOMEN.

BY

Dr. LEOPOLD LANDAU.

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ALBUMINURIA

IN

HEALTH AND DISEASE

WITH TWO APPENDICES

(1) A CONTRIBUTION TO THE THEORY OF URINARY SECRETION.

(2) THE HYGIENIC TREATMENT OF ALBUMINURIA.

BY

DR. H. SENATOR,

PROFESSOR EXTRAORDINARY OF MEDICINE; PHYSICIAN TO THE ROYAL CHARITÉ HOSPITAL, AND TO THE AUGUSTA HOSPITAL OF BERLIN.

WITH ONE LITHOGRAPHED ILLUSTRATION.

TRANSLATED BY

DR. T. P. SMITH.

Dedicated

TO

HERR RUDOLF VIRCHOW,

IN

COMMEMORATION OF HIS SIXTIETH BIRTHDAY

AND OF HIS

TWENTY-FIVE YEARS' WORK

AS A

TEACHER IN THE FREDERICK WILLIAM UNIVERSITY.

Uniting, as I do, with that large concourse of your admirers who now approach you with expressions of gratitude and every good wish, I should like, so far as lies in my power, to bear witness to the fact that practitioners also look up to you as their pioneer and master, and that therapeutics, in the strict sense of the term, is not behindhand in gratitude and in the recognition of the enormous obligations, which it, no less than theoretical medicine, owes to you. It does not require my pen to recall these services to the memory of our contemporaries, nor to protect them from future oblivion, for so long as there is a history of medicine, the mighty epoch-forming impulse, which all branches of this science have received through you, will be duly recorded in its pages. I will dwell only upon one point, which, in the abundance of the meritorious services now so vividly

remembered, might be, if not forgotten, at all events too little appreciated. When about three decades ago a new development of medical science took its rise, due in no small degree to the powerful influence of your labours, when new branches shot up vigorously on the old but ever regenerating tree of medicine, and one branch alone, the oldest indeed —which seldom puts forth true blossoms, but when it does so, brings to maturity precious fruit, blessed by all mankind —remained sterile and, to all appearance, a hard, hopeless twig, when it was a mistake to believe in therapeutics—at that crisis you, most honoured Sir, were not afraid to come forward and avow your participation in this supposed error.[1] You had faith in therapeutics; you considered it to be not a dead branch, all but given up though it was by those who were especially called upon to make use of it. Your foresight has not deceived you; with the growth of the tree, so much of which it owes to you, that branch also has received a fresh stimulus, and the indirect effect of your labours and the direct effect of your words, spoken in due season, have been to introduce it to the workmen and the care of which it stood so much in need. Therapeutics has once more become prominent, having received from you both aim and direction.

In this treatise, which I have the privilege of presenting to you, a place could not be found for therapeutics, for the reason that albuminuria, which is its subject, is upon the whole but little amenable to treatment. But as you have not shown any scepticism on therapeutics in general, when that science seemed all but extinct, so we are not without hope that in this particular case the power of medicine will some day be proved. These pages appear to me to be not unworthy of being dedicated to you, because their contents fall within the lines of "pathological anatomy and physio-

[1] 'Handb. d. spec. Pathol. u. Therap.,' i, 1854, Vorwort, S. xi.

logy and of clinical medicine," departments to which you have given such abundant space in your Archives, the imperishable monument of united work in scientific medicine and a motto for all your successors.

H. SENATOR.

BERLIN;
October, 1881.

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ALBUMINURIA

IN

HEALTH AND DISEASE.

Introduction.

"History teaches us that the views of modern times constantly revert to those points which were regarded by earlier observers as settled, and thus, particularly nowadays, when so few have leisure for the historical study of science, there is perhaps ample justification for bringing old notions within the intellectual view of a succeeding generation."—R. Virchow's Preface to his '*Gesammelte Abhandlungen zur wissenschaftlichen Medicin*,' 1856.

I can think of no better introduction to the following treatise than Virchow's words which I have placed at its commencement. For, as holds good with regard to so many other scientific questions in medicine, so likewise and in an especial manner with reference to the doctrines on albuminuria, they possess in my opinion complete validity and at present perhaps more than at any previous time. The older views, nowadays if not entirely abandoned, at all events but little regarded, assigned as is well known, various causes for albuminuria, viz. alterations in the circulation in the kidneys, alterations in the membranes placed between the blood and the urine, and changes in the condition of the blood. To one or other of these three factors was a greater or less share attributed in the origination of albuminuria, and according to the existing state of science, attempts were made to determine the influence of each of them. The most energetic and successful opponent of these doctrines was J. B. Stokvis, who, supported by extended experimental and clini-

cal investigations, declared that every alteration of the circulation which checks the afflux of arterial or the escape of venous blood, causes albumen to pass into the urine, but that all other conditions to which the phenomenon had been formerly attributed are to be rejected as ineffectual (1). According to this view, the only recognised cause of albuminuria is a retardation of the flow of blood in the kidneys, for such is the necessary result of every alteration in the circulation, in the sense applied to the term by Stokvis. Only a slight advance was now required in order to determine which of the two functionally different portions of the vascular system of the kidneys was the site of the circulatory disturbance which caused the albuminuria, and the results of the most recent investigations made with the view of determining this question agree in showing that the Malpighian tufts are the portions implicated in the disturbance. According to Runeberg, (2) and Posner, (3) with whom Ribbert, (4) and Litten, (5) agree, these vessels are the sole or the only necessary seat of the excretion of albumen; the cause of the escape of albumen from them is said by Runeberg to be the diminution of the pressure of the blood, while importance is attached to the retardation of the current by Posner and Litten, the latter being supported by Runeberg's views with regard to the effect of the lowering of pressure upon the excretion of albumen.[1] Retardation of the current, when prolonged, changes, as Cohnheim (7) has shown, the condition of the walls of the vessels, inasmuch as it renders them abnormally permeable by the constituents of the blood and especially by the albumen. Cohnheim himself likewise (8) considers the disturbance of the circulation in the Malpighian tufts to be the cause of the albuminuria, but the ill effects of this disturbance are of less serious import upon the walls of the vessels than upon the epithelium which covers the glomerular vessels, for he agrees with Heidenhain (9) in attributing

[1] It is true that elsewhere Litten has expressed a different opinion, viz. that the epithelium of the convoluted tubes is concerned in the excretion of albumen, inasmuch as under pathological conditions it does not remove the albumen from the transuded secretion pressed through the loops of the vessels of the glomeruli, and which in the normal condition contains albumen (6).

to this epithelium the retention of the albumen under normal conditions.

Such then is the shape which in recent years the theories with regard to albuminuria have assumed, mainly in Germany, but also in other countries, as may be inferred from the latest writings of Charcot, (10) Lépine, (11) and others. It is obvious that this theory has the advantage of great simplicity as compared with the older views, for all other factors, to which formerly so much thought and investigation were directed, are now held to be inoperative; they are even scarcely mentioned. Posner, indeed, does not think it worth while to discuss them, for he asserts that they are devoid of any real basis. He goes, however, much too far in his assertions; for some time past facts have accumulated clearly indicating the dependence of many forms of albuminuria upon alterations in membranes other than those of the Malpighian tufts, and yet concerned in the secretion of urine, or upon changes in the condition of the blood. The facts referred to could not therefore be excluded from a treatise on this subject, and they will be examined in the following sections after the influence of circulatory changes has been discussed, for to these latter they are undoubtedly subordinate in importance.

I. The Various Forms in which Albumen is Excreted.

In calling attention to the older views with regard to albuminuria, I do not mean that they should be simply received in the somewhat vague and indefinite form in which they have been handed down, but I wish to show that, as compared with the most recent, simple, but one-sided doctrines, they make better allowance for facts, inasmuch as they proceed from a higher stand-point and embrace a larger field of view. It is true that even they do not so comprehend all points of view that none are left out; the circle of possibilities may extend itself beyond past and present ideas. How much of them is worthy of consideration, and for what cases the one or the other possibility is deserving of notice will be discussed in the following pages.

There is one general meeting-point for all received theories of albuminuria, old as well as new, inasmuch as without exception they all place the source of the urinary albumen in the blood, and almost exclusively in the blood of the Malpighian tufts. There is a universal absence of doubt on this point, so that the question as to the possibility of any other source of the albumen has never once obtruded itself and has certainly never been discussed.[1] But a thorough and unbiassed consideration forces us to acknowledge that the albumen may have places of origin other than the blood of the Malpighian tufts; and as none of the received theories, which make no reference to such a possibility, suffice to explain in a satisfactory manner all the various forms of albuminuria, as further description will show, a discussion of the other ways in which albuminuria may originate does not appear to be superfluous, however little it may harmonise with our ordinary notions. It is certainly not necessary in discussing the origin of albumen in the urine, to claim as possible sources everything in the kidney that contains or is composed of albumen, and this remark is especially true with regard to the constituents of the tissue, such as the interstitial connective tissue, the substance of the walls of the blood-vessels and lymphatics, or the basement membrane of the urinary tubes, for only if these tissues were completely destroyed could their albuminous elements pass into the urine. There remains, however, the blood of the interstitial vessels, the lymph and the various epithelial cells of the kidney, all of which might yield albumen to the urine, and there is no reason of any kind for totally excluding these from discussion. On the contrary, there is abundant cause for examining into any possible participation of these tissues in the production of albuminuria, because conjointly or separately they are more or less affected in the processes which lead to albuminuria—in congestion, and in the various forms of inflammation and degeneration of the kidney.

In the first place it is necessary to form a clear conception

[1] On a former occasion (Virchow's 'Archiv,' Bd. lx, S. 478, u. 497) I have drawn attention to other possible sources of albumen in the urine, and Bartels has partially assented to my views (in v. Ziemssen's 'Handb. der Pathol.,' ix, 1, s. 36, u. 38).

of the term "albuminuria," for the present limitation of its meaning has become insufficient, owing to the increase of our knowledge with regard to the albuminous substances which are found in the urine. The custom has been to distinguish only "genuine or true albuminuria" from "false albuminuria," and to signify by the latter term those conditions in which non-albuminous urine from the parenchyma of the kidney subsequently receives an admixture of an albuminous fluid, such as the semen or prostatic fluid, or blood, pus, lymph, or the fluid of disintegrated tissue. This false albuminuria requires no further explanation. In true albuminuria, in which albumen together with other constituents of the urine is yielded by the parenchyma of the kidneys, our thoughts are usually directed only to the excretion of albumen coagulable by heat. The property of coagulation possessed by the urine led to the discovery of albuminuria by Cotugno. It occurs in nearly all cases, and harmonises, in a general way, with the idea which regards the blood alone as the source of the albumen. We can therefore readily understand how that at the present day the term "albuminuria" is used in the same sense as it has been for a century past, and that it leads to confusion when albuminous substances, non-coagulable by heat, are found in the urine, even if in other respects they entirely resemble urinary albumen, and that such cases are not regarded as belonging to albuminuria. Coagulation, however, on being heated, is no more a property of all albuminous substances than it is peculiar only to one of them, so as to render it possible for the term "albuminuria" to be limited to the excretion of this one coagulable form of albumen. On the contrary, if we accept the current idea of the origination of the urinary albumen from the blood, it would necessarily follow that more than one coagulable albuminous substance should pass into the urine, for the blood normally contains at least two such substances in solution, viz., the serum-albumin (serine) and the globulin. There is not the slightest ground for the assumption that always, and without exception, only one of these two substances, possibly the serum-albumin, as generally supposed, escapes from the blood; there is more *à priori* reason for expecting that, if

not always, at least in a greater or less number of cases, both substances escape, though possibly not in equal proportions. And the fact accords with our expectation, for in most cases in which coagulable albumen is found on heating the urine, we are able to demonstrate the presence of both serum-albumin and globulin. It therefore certainly appears not impossible that, under certain circumstances only one of these substances should pass into the urine, at least in an appreciable amount, for different as may be the views with regard to the process of excretion of albumen in the kidneys (and these views are by no means clear, as we shall presently see), they all agree in allowing that the capacity for diffusion and filtration possessed by the various albuminous substances plays a considerable part in their excretion.

In this respect, however, there is an important difference between the two albuminous substances which normally occur in the blood, for globulin is much more diffusible (possibly also more capable of passing through a filter) than serum-albumin, and therefore much more likely to be found in the urine, supposing that fluid to contain only one albuminous substance in appreciable quantity. If the recent statement of Estelle (12) be verified, which is to the effect that sometimes only the albuminous substance which is precipitated by magnesium sulphate (viz. globulin) is to be found in the urine, this theoretical assumption would be thereby remarkably confirmed, and it would be necessary to make use of the term "globulinuria." At present, however, sufficient attention has not been paid to this subject, and the methods in use for demonstrating the presence of globulin are, as Hammarsten (13) has shown, unsuitable for the purpose, inasmuch as they are inexact, and altogether fail for the detection of small quantities. It is therefore to be anticipated that the new method described by Hammarsten, and used also by Estelle, will enable us to detect globulin in the urine, in larger quantities, and more often than at present. That serum-albumin alone should pass into the urine, that is, that a pure "serinuria," as it might be called, should occur, is less probable from the reasons given above. It is true that up to the present time the fact that globulin has not, in all cases, been found associated with the serum-

albumin has rendered probable the existence of a pure serinuria, but the failure to detect the globulin has been doubtless due to imperfect methods of examination.

The foregoing statements have conclusively shown that even when the term "albuminuria" is used in its present limited signification, it does not describe the excretion of one and the same albuminous substance. And in addition to this, albuminous substances have been recently discovered in the urine which are deficient in the capacity for coagulation hitherto deemed a distinguishing characteristic. Peptone is the most remarkable example of this kind. Whereas formerly nothing was known of its occurrence in the urine, and subsequently it was regarded as a doubtful constituent or as a curiosity, quite recently Hofmeister and his pupils have shown that under certain, by no means rare, circumstances, it occurs in the urine in considerable quantities and that this excretion, this "peptonuria" is of scientific and practical importance (14). At the same time it must be remembered that only slight traces of peptone are discoverable in the blood, and that often these are absent. Compared with the current theory of the origin of all urinary albumen from the blood, this might seem very puzzling, did we not possess other experiences which help to solve the mystery. We know that the kidneys by virtue of a peculiar attractive power excrete certain materials in large quantities, and withdraw them from the blood which contains scarcely more than mere traces (as, for example, urea, uric acid, kreatin, &c.), and in like manner Hofmeister explains peptonuria as the result of a specific peculiarity of the excretory elements of the kidneys, inasmuch as he found that after the injection of peptone into the blood, or under the skin, the urine is surcharged with it, while scarcely an appreciable trace is discoverable in the blood. This explanation appears to be very plausible, and is doubtless accurate for certain cases which are analogous to this experimental administration of peptone, as for example, when exudations containing peptone are absorbed, as occurs in pleuritis, pneumonia, and rheumatic arthritis. It is another question as to whether this is the only way in which peptonuria can originate, or whether it may not have other sources. This question appears quite

justifiable, as we are taught by recent observations, to the effect that no such sharp distinction exists between albuminous substances proper and peptones, as we were formerly disposed to believe. Not merely as a result of digestion, but likewise in consequence of various influences of a different kind and especially of incipient putrefaction, albumen undergoes modifications which more or less resemble that which we call "peptone." It is quite conceivable that in certain morbid conditions, the ordinary albuminous substances in the blood and the fluids of the body or in the kidneys, become so changed as to lose their coagulability on boiling and their capacity for precipitation on the addition of certain acids and metals. There is indeed no lack of indications of the occurrence in the living body of such processes as can effect alterations in the albuminous substances, similar to those produced by digestion and many fermentative and putrefactive processes. Thus, in these days, the idea has become very familiar to us that in fever certain fermentative processes take place in the body, and it is in febrile diseases that the occurrence of peptone-like bodies in the urine has surprised observers more often than was the case in former times. And the processes of tissue-metamorphosis in various conditions of poisoning, especially in phosphorus poisoning, present numerous analogies with the processes of fermentation and putrefaction, and Selmi (15) has quite recently drawn attention to their similarity, and these again are exactly the circumstances (phosphorus-poisoning being the most remarkable of them all) in which these bodies are to be found in the urine. These examples are sufficient to show that by no means invariably only that form of peptone which is contained in the blood and lymph is excreted with the urine, but that possibly, or probably, changes of combination occurring in the blood, the lymph, and even in the elements of the tissues, may convert the ordinary albuminous substances into peptone-like modifications. We are likewise justified in supposing that certain intermediate products, which are formed during the transition of the albuminous substances proper into peptone, may occur in the economy, and are also excreted by the urine, and that there is just as little reason for maintaining a strict distinction between

"peptonuria" and albuminuria as between peptone and albuminous substances proper.

Recent investigations have likewise shown that the conversion of albuminous substances into peptone takes place gradually and in stages, certain intermediate or transitional products being formed, and at least one such product is clearly recognised, analogous in some respects with albumen, and in others with peptone, viz. hemi-albumose or pro-peptone (16). The most important peculiarity of this substance is that it is not precipitated from its watery solution by boiling, but is precipitated in the cold state by acetic acid and ferrocyanide of potassium, nitric acid, acetic acid and concentrated solution of chloride of sodium.[1] This substance, or one very similar to it, has been discovered by Virchow in the medulla of bones in cases of osteomalacia, and recently by Fleischer in the normal bony medulla. An exactly similar substance, or perhaps a mixture thereof with another form of albumen, had been previously discovered by Bence Jones in the urine of a patient suffering from osteomalacia. Langendorff and Mommsen made a similar discovery in a case of this nature, although they were not able to demonstrate the presence of any similar albuminous body in the bones after death, and lastly Kühne found hemi-albumose in the urine of a patient the subject of osteomalacia. There are, in addition, a few isolated reports with regard to the presence in the urine of an albuminous substance, non-coagulable by heat, or dissolved by boiling, if precipitated in any other way (17). However, from the generally very meagre character of the statements it is impossible to decide as to how far they always refer to the substance in question. Prout, Gerhardt, Beneke, Fürbringer, and Gowers make, however, sufficiently definite statements on this subject, and from these we may infer the occurrence of hemi-albumose in various morbid conditions (18). In addition, Stokvis (19) has found it in the urine of a dog after injecting it into the rectum; and finally, Lassar (20) asserts that having rubbed petroleum into the skin of rabbits, he found, after a certain interval, hemi-albumose with all its characteristic peculiarities in the urine. On the whole it would appear from these

[1] For additional reactions I refer to Kühne and Salkowski's statements.

scanty and not altogether positive statements, that hemi-albumose or pro-peptone (pro-peptonuria,[1] as I shall henceforth call it,) was an extremely rare occurrence in the urine, and unworthy of consideration. My own observations, however, directly contradict such a notion, for they show that though pro-peptonuria is not very common, it is vastly more frequent than current ideas and the literature of the subject would lead us to believe. During the past three or four years I have met with seven cases, one being a man aged forty-four, who had several times been the subject of syphilis, and in the course of years had undergone various courses of anti-syphilitic treatment. When first admitted into the Augusta Hospital, he was fairly well nourished, slightly jaundiced, his liver and spleen were considerably enlarged, and there was profuse diuresis, to an extent of 6000 cctm. daily, corresponding with the somewhat intense thirst. The urine had a sp. gr. of 1004—1006, and was free from sugar. It exhibited a decided cloudiness on the addition of acetic acid and ferrocyanide of potassium, and a similar reaction with the same acid and a concentrated solution of sulphate of magnesia. It remained clear on boiling, and the subsequent addition of nitric acid made no difference. When warmed with solution of caustic soda and a very small quantity of sulphate of copper, a red colour was produced. For more minute examination a certain quantity of urine was mixed with about three times its bulk of absolute alcohol, and the precipitate, after settling, was mixed with distilled water and filtered. The clear solution became cloudy on the addition of nitric acid, and also when acetic acid was used; excess of the latter, however, caused the clearness to return; boiled with nitric acid it became clear and a faint yellow in colour, with soda and sulphate of copper the solution assumed a beautiful violet, and not a red colour. The urine gave the same reactions on further examination a few days later, but not afterwards, the quantity and specific gravity having at the same time become normal. The patient was discharged

[1] The name hemi-albumose, which has been applied by Kühne to the product of digestion referred to, is older than the term pro-peptone suggested by Schmidt-Mühlheim, but I have preferred the latter as being more euphonious.

much improved after a five months' stay in hospital, and on his subsequent return on two occasions for short periods and for slight ailments, nothing abnormal was discovered in his urine.

The other cases were as follows :—A man, aged 61, with right hemiplegia ; a woman, aged 58, with pneumonia on both sides ; a child, with diphtheritic laryngitis, on whom tracheotomy had been performed in the surgical ward ; a man, aged 55, with cancerous stricture of the œsophagus ; a man, aged 26, with pneumonia on the left side ; and lastly a theological student, aged 21, who, as stated by his physician, had suffered four years previously from diphtheritic inflammation of the throat, with dysentery and hæmorrhagic nephritis as sequelæ, and lastly from neuritis of the left brachial plexus, followed by paresis and atrophy of all the muscles of the arm. This latter affection had entirely subsided after lasting about a year, so that when I examined him in July, 1880, the action of the left arm was almost normal, and the limb appeared to be only a little weaker than the right. (Its circumference, as measured at various points, was still .5—2 ctm. less than that of the right.) For some time albuminuria had remained as a sequela of the nephritis, and the patient had learnt how to test for albumen by boiling his urine. After about a year and a half had elapsed, during which time the quantity of albumen had gradually diminished, nothing abnormal could be discovered by means of this test ; subsequently, on boiling the urine, albumen was occasionally discovered. When I examined him, only the urine passed after the midday meal became permanently cloudy on long boiling and the addition of acid (see II and V, Albuminuria of Digestion), the urine passed at other times contained no albumen coagulable on boiling, but certainly pro-peptone. It should be remarked that the patient had never suffered from œdema, and in spite of the four years' continuance of the albuminuria there was no trace of any cardiac hypertrophy. He felt also perfectly well, and applied to me only for an explanation with regard to the state of his urine.

In all cases the test for pro-peptone was that the urine remained clear on boiling, but became cloudy or yielded a

precipitate with acetic acid and ferrocyanide of potassium. For a further examination, we adopt the method described in the first case, that is, by precipitating by alcohol. It was not always possible to test all the reactions for hemi-albumose, mentioned by Kühne and Salkowski, partly because they were not known at that time, and partly because the propeptonuria had disappeared before the minute and systematic examination could be made. But this much was determined, in addition to the reaction with acetic acid and ferrocyanide of potassium, that boiling alone left the urine clear, that nitric or acetic acid produced cloudiness in the cold urine, and that clearness was restored by heat. Small differences in the behaviour of the urine, and deviations in the reactions of the hemi-albumose obtained during digestion repeatedly occurred, thus in the biuret test violet often took the place of red, or acetic acid and concentrated solution of sulphate of magnesia, or of chloride of sodium, caused no cloudiness, or only a very slight degree of it in cold urine, and still less on boiling. In my opinion much importance need not be attributed to these deviations, for the quantity of saline constituents of the urine and other admixtures may well account for them.

It appears more important to call attention to the fact that it is highly probable that cases occur in which hemi-albumose and albumen coagulable by heat (serum-albumin and globulin) occur together in the urine,—cases which can be designated by the term "mixed albuminuria." I have repeatedly met with cases, and my experience is certainly not solitary, in which the urine yielded a slight precipitate on boiling, while the addition of nitric acid without previous heat, or of acetic acid and ferrocyanide of potassium, produced a copious precipitate, and further that the precipitate caused by the nitric acid was diminished, and not increased on boiling the mixture. I had hitherto always thought that an explanation must be found for this in the variety of the saline constituents of the urine, or perhaps in the fact that in the cold urine uric acid was likewise precipitated, until I closely investigated a case of sub-acute nephritis, in which these reactions were very striking. The urine which was very acid was boiled, while a drop of acetic

acid was added, a flocculent precipitate was the result; the hot fluid was thrown on a filter and the clear filtrate, mixed with thrice its bulk of absolute alcohol, allowed to stand for twenty-four hours. The white precipitate was readily soluble in distilled water, forming a clear solution, the slightly acid solution remained clear on boiling, but became decidedly cloudy on the addition of nitric or acetic acid, and cleared up completely on boiling, the solution containing the nitric acid becoming yellow, and the cloudiness not returning on cooling. The precipitate caused by acetic acid was soluble in excess, but not so that caused by nitric acid. In the solution, again made clear by excess of acetic acid, ferrocyanide of potassium caused a considerable precipitate. The addition of soda and sulphate of copper produced a beautiful violet colour. The behaviour of this precipitate certainly differs in some respects from that of hemi-albumose and especially in this that the precipitate caused by nitric acid and dissolved by heat does not reappear on cooling, but, on the other hand it differs so essentially from the ordinary albumen of the urine that we can only assume that we are dealing with different albuminous substances, or that some modification has taken place as a result of the treatment (boiling and alcohol) to which the urine has been subjected. But inasmuch as ordinary albuminous urine treated in a similar manner does not become thus modified, the conclusion is perfectly natural that the case is really one of "mixed albuminuria." Additional instances of its occurrence are to be collected.

The peculiarity of hemi-albumose (pro-peptone), of not being coagulated by heat, certainly accounts for the fact that so little is known up to the present time of pro-peptonuria. For, in the method commonly used for discovering albumen in urine, viz., boiling, and then adding nitric or acetic acid, its discovery is impossible, unless the mixture is set aside and examined when perfectly cold, but this detail is, as a rule, neglected, and if attended to there would be this source of doubt viz., that uric acid and the products of the urinary colouring matters gradually separate from urine thus treated. It has also been repeatedly pointed out that this test is inadequate in other respects, inasmuch as it is not sufficient for the detection of very small quantities of ordinary albumen

in the urine. As, however, notwithstanding these drawbacks, the test in question is the only one in common use, it will not be superfluous to give a brief account of far more reliable tests which should be used in doubtful cases, or I would rather say in all cases in future. These are as follows: (1) to acidify the urine with acetic acid, and then add carefully a (concentrated) solution of ferrocyanide of potassium, according to Hofmeister the most delicate of all the tests for albumen, and which precipitates all the albuminous bodies, but not peptone.[1] 2. The careful addition of nitric acid to the (non-warmed) urine, followed by boiling if any cloudiness results. 3. The addition of a concentrated solution of chloride of sodium, or sulphate of magnesia to urine acidulated by acetic or nitric acid. All three tests, or at least the first and one of the others, should invariably be tried. If after applying the second or third, a precipitate which is formed on heating, entirely or partially disappears, the presence of pro-peptone is indicated. Hindenlang (21) has lately recommended metaphosphoric acid as a delicate test for albumen and it also precipitates peptone. As far as my present experience goes, and as regards delicacy and convenience of testing, I can support his recommendation, but for cases where great exactness is required, I would recommend that recourse should also be had to the other tests, both for the purpose of checking results, and also because when the quantity of albumen is small, the saline constituents of the urine, the presence of other organic substances and other unknown influences are apt to cause slight differences in its behaviour towards one or other of the reagents. For the discovery of pro-peptone, however, it is necessary to have recourse to one test which is applicable to cold urine and to another which requires the addition of warmth.

If, as we may assume from Hindenlang's statement, metaphosphoric acid is adopted for the discovery of very small quantities of peptone in the urine, we shall have obtained a very convenient method for the detection of

1 Salkowski states that this test fails only when a large amount of chloride of sodium is present, but the urine never contains this constituent in such quantities as to prevent the precipitation of albumen.

peptonuria. A precipitate caused by the acid, but not by acetic acid and ferrocyanide of potassium, in the original urine will indicate peptone to the exclusion of other substances. But if the urine gives a precipitate with the two last-named reagents, it is necessary first to boil and then filter it so as to remove the coagulable albumen (serum-albumin and globulin) ; the filtrate, which must remain clear on the addition of acetic acid and ferrocyanide of potassium, may then be tested with metaphosphoric acid for peptone (and pro-peptone ?). At any rate the filtrate could be concentrated by evaporation and the precipitate caused by excess of strong alcohol could be mixed with water and tested. As a matter of course the positive results yielded by the tests must be confirmed by other reactions.[1]

The occurrence of such various albuminous substances in the urine, especially of such as are not found in the blood under normal conditions, suggests the idea that changes in the composition of the blood may be concerned in the production of these peculiar forms of albuminuria. We have in consequence learnt to recognise an additional factor for the causation of albuminuria and shall therefore have to consider the kidneys, and also changes in the conditions of the circulation and of the epithelium and likewise alterations in the condition of the blood.

II. Albumen as a Constituent of Normal Urine.

The question as to whether the urine in its normal condition does or does not contain albumen must always form the starting-point for all investigations with regard to albuminuria. That this question should be raised at all may seem heretical

[1] The following method is recommended by Hofmeister for the demonstration of peptone for clinical purposes. About one tenth of its volume of concentrated hydrochloric acid is mixed with the urine, an acid solution of the phosphortungstate of sodium is added, and the mixture is filtered without allowing any precipitate to subside. The precipitate is washed with dilute (3—5 proc.) of sulphuric acid, placed in a mortar and rubbed up very thoroughly with baryta in substance, then mixed with a little water and warmed for a short time. The fluid filtered from the insoluble baryta compound is then used for trying the biuret reaction.

to many persons, inasmuch as it is regarded as long since disposed of, and answered in a decidedly negative manner. However, in spite of the fact that the doctrine that normal urine does not contain albumen, and that albuminuria is invariably a sign of disease, has not yet been exploded, it is nevertheless true that occasional doubts as to its validity make themselves heard with increasing force, and even its most determined supporters, in consideration of the most recent observations, which I shall presently mention, now admit that exceptions exist to a dogma which only a short time ago was held not to admit of them. This change of opinion was caused by the demonstration that, without any renal disease, small quantities of albumen, formerly overlooked, appeared in the urine, but without assuming significance, in the course of many varying morbid conditions, and in the second place by the discovery of albuminous substances, already referred to, distinguished from the albumen coagulable by heat, ordinarily occurring in the urine, by being imperfectly coagulable, but more soluble and diffusible, and whose escape therefore from the blood must, according to current ideas, seem very obvious. Improved methods of investigation, and the discovery of delicate reactions have further resulted in the discovery of albumen, in very many instances, but in very minute quantities, in the urine of perfectly healthy men, and this albumen, so far as it was possible to judge from these small quantities, differed in no respect from the albumen of the ordinary forms of albuminuria. I make this assertion, because some years ago statements were made to the effect that a substance with properties resembling albumen or peptone could be found in all specimens of normal urine ; but little reliance was placed on these statements, by reason of the defective methods of demonstration, or certain irregular reactions exhibited by the substance demonstrated. Thus, to pass over the statements of earlier observers, Harley, but especially Béchamp and after him Foster, Vintschgau and Cobelli have obtained from the alcoholic precipitate of urine a substance called "nephrozymasis," which, however, on account of its diastatic properties was regarded as a ferment, and not as a form of albumen (22). It is not, however, possible to make a complete distinction

between albuminous substances and ferments, even supposing that the latter could be obtained in a pure state, inasmuch as Seegen and Kratschmer's (23) investigations show that substances unequivocally albuminous may exhibit diastatic properties. Moreover, Leube, who in examining the urine of twenty-one healthy men, found albumen in the alcoholic precipitate in fourteen instances, distinguishes the latter from the diastatic ferment, since in four of those fourteen cases albumen occurred alone, and in seven out of the twenty-one only the ferment was demonstrated. In its reactions the albumen resembled paralbumen (24).

Be this as it may, there are now additional observations with regard to the occurrence of albuminuria in healthy men. That in these cases the substance referred to is albumen in the ordinary sense, there is no room whatever for those doubts which were constantly evinced towards the statements of the older observers, such as Becquerel, Simon, C. Schmidt, and Canstatt. In recent times such observations have constantly accumulated in proportion as urinary investigations have been more numerous, and conducted with greater care and more improved methods than formerly. I have already referred to these methods (see p. 14), and we may with certainty expect that as they become more frequently employed, the discovery of albuminuria will become much more common than hitherto. There is, however, at the present time a considerable number of observations of a trustworthy character and to a great extent demonstrative, although the most delicate methods have not always been employed, of the excretion of albumen going on in healthy men for a greater or less length of time, no sign of disturbed health showing itself while this was going on, and during an interval of some years. Cases of this kind have been noticed by Frerichs, J. Vogel, Ultzmann, Guéneau de Mussy, Leube, Gull, Moxon, Rooke, Dukes, Saundby, Edlefsen, Marcacci, Munn, Bull, Fürbringer and Kleudgen (25).

This large number of recent observers, a portion of whose discoveries were made quite accidentally, forbids us to suppose that this albuminuria forms a very rare exception or curiosity unworthy of consideration. Still more cogent in this respect is the proportionate frequency discovered by

certain observers who have specially investigated this subject. According to Leube albumen was found in the urine of 19 out of 119 healthy soldiers, or in 16 per cent.; according to Munn in 24 (12 per cent.) out of 200 apparently healthy persons who presented themselves for examination for life-assurance. Fürbringer found albumen in 7 out of 61 healthy children (11·5 per cent.), Kleudgen in 14 out of 32 healthy nurses, (44 per cent.), and likewise in the others when the urine was concentrated and its specific gravity exceeded 1014. Kleudgen, therefore, thinks that (apart from imperfect methods of testing) the dilution of the urine is the sole cause why, as a general rule, no albumen is discoverable in it. Certainly the dilution of the urine is an element of very considerable importance in the demonstration of its albuminous constituents, a point to which I shall draw particular attention; but it must not be supposed that by merely concentrating the urine until its specific gravity becomes very high, albumen previously undiscoverable will be invariably and at once found. This is by no means the case, as we can convince ourselves by evaporating every specimen of urine which the ordinary tests show to be free from albumen. The saline constituents, which are thereby increased in proportion, constitute an additional element in reference to the demonstration of albumen, and as these are liable to considerable variations in natural urine and under physiological conditions, there is at least one obstacle, of a physiological character, to the discovery of albumen in normal urine, and even though it be of a high specific gravity. On the other hand, in concentrated urine, after a portion of its saline constituents has been precipitated by alcohol, it is certainly easier to discover albumen than it was before. It would appear, however, that other physiological factors are at work in producing the result that albumen is sometimes found in the urine and is absent at others. My own observations are definite on this point. Some years ago I had, in common with other observers, occasionally found albumen in the urine of perfectly healthy persons, but especially in convalescents or in patients suffering from ailments of a slight nature and totally unconnected with renal affections. The discovery naturally gave rise to the suspicion that the persons were the

subjects of chronic interstitial nephritis (true atrophy of the kidney, renal sclerosis), but this was not confirmed by further observation. My attention having been aroused by this varying symptom, occurring in myself and my three young colleagues (assistants in the Augusta Hospital), all of us enjoying excellent health, I examined the urine at different hours of the day for a long period, and at one time or other discovered albumen in the urine of each of us, certainly only in faint traces, which might well have been overlooked had less delicate tests been employed.[1] No definite rule governing its appearance could, however, be discovered, for the urine might be examined for several days without once finding albumen, which would then appear for a day and again vanish as before. In my own case I found it most frequently during the morning (11—1 o'clock), and only exceptionally in the late afternoon after the principal meal. In one of my colleagues it was several times found after dinner, in another likewise during the period of digestion, but only when much meat had been taken; in the third the urine was examined only a few times, but sufficiently often to show the occasional presence of albumen.

Many observers have asserted that it is easy to discover albumen in the urine during digestion, others have indicated muscular activity as a factor giving rise to this result. The physiological conditions are, however, by no means exhausted by these statements, as my own observations incline me to suppose, indeed they supply good reason why I should consider it not improbable that if we were to examine the urine for long periods at different hours of the day and with great care, we should, sooner or later, find it to contain albumen in the case of every healthy man. If, however, the usual custom be followed, and the total quantity or a large proportion of the urine passed in a single day be submitted to examination, the small amount of albumen contained in one portion is still further diluted by mixture with the remainder, and the obvious result is that even after prolonged examination the urine is never, or only exceptionally, found to be albu-

[1] To avoid any error which might be caused by admixture of semen or prostatic secretion, the portions of urine first passed were not used for examination.

minous. It is much to be desired that the urine passed at different times of the day, by a still greater number of healthy men, should be subjected to methodical and continuous examination.[1]

There is, however, something more to be said with regard to the observations that have been made. To take only the smallest of the numerical proportions referred to, if it be true that among eight or nine healthy men, there is one who excretes albumen once or more often in his urine, it is impossible simply to regard albuminuria as a symptom morbid in its character under all circumstances; but the admission must be made that if the occurrence in question does not form the rule, it comes at any rate within the bounds of a physiological condition. It must be admitted either that albumen is present in all urine, but in varying quantities, so that sometimes it eludes observation by the means at our command and sometimes, under certain physiological conditions, it can be discovered, or that it is altogether absent at certain periods and appears in the urine only when certain conditions are in existence. The former of these two assumptions, viz. that the urine always contains albumen but only occasionally in appreciable quantities, appears to me to be the more reasonable and eligible, for the two following reasons. In the first place it cannot be denied that our power of demonstrating albumen in urine has a certain limit, even in cases where there is no doubt as to its presence. The truth of this assertion is supplied by specimens of urine in which no albumen is discoverable by the ordinary methods. For it is well known that all normal urine, after remaining at rest for some time, exhibits a slightly opaque cloudiness (nubecula), which is caused by the epithelium of the urinary passages, and contains albumen, as can be proved by collecting it in considerable quantities (16 *a*). The objection might be raised that this albumen is mixed with the urine but not dissolved in it, but even when its solution is effected by adding liquor sodæ, no reaction due to albumen is shown by the urine, as was likewise the case before the addition of the

[1] The urine of the animals commonly used for experiment (rabbits, dogs, and cats) very often contains traces, and not seldom considerable quantities, of albumen under conditions of apparently perfect health.

soda. We are therefore perfectly justified in stating that our present methods are not sufficient for the detection of extremely minute quantities of albumen in the urine, and that therefore urine in which no albumen can be found may yet contain traces of that substance. In the second place, it is more in accordance with our other experiences and ideas to assume quantitative oscillations of a function within physiological limits rather than qualitative changes. For example, still to confine ourselves to urine, we daily see that any one of its normal constituents may be excreted in quantities greater or less than the average, but it is a far rarer occurrence, if after all under physiological conditions, for an entirely new substance and one hitherto absent to appear in the urine, unless it or something from which it could be immediately derived, had been introduced into the blood. Indeed, when something of this kind takes place, *i.e.* when a substance previously unknown in normal urine is discovered therein, we are, *à priori* and with justice, disposed to assume that it is a normal constituent of the urine, but one which has hitherto escaped recognition, by reason of the minuteness of its quantity or because our methods of investigation were inadequate for its detection.

Of late years numerous experiences of this character have been afforded by various constituents of the urine. I refer especially to two of them, viz. grape sugar[1] and oxalic acid, because they play in pathology a part similar to that of albumen. How much controversy has taken place as to whether these substances belong to the normal constituents of urine, or whether their occurrence is under all circumstances a morbid phenomenon! Nowadays we know that they do occur normally in urine, but in such insignificant quantities that a large volume of urine and very delicate methods are required for their demonstration; and we also know that

[1] According to the most recent investigations the reactions supposed to be due to the presence of sugar in the urine may depend upon glycuron acid. For our purposes, however, this question is of no consequence, for sugar is referred to here only for the sake of comparison, and besides it and oxalic acid there are several other substances with regard to which the above statements equally hold good. These are hippuric acid, glycerin-phosphoric acid, inosite, xanthin, indoxyl-sulphate of potash (indican), phenol, pyrocatechin, &c.

sometimes, and even under physiological circumstances, they appear in larger quantities easily recognisable, and that therefore we must admit the existence of a physiological glycosuria and oxaluria. Considering that there are admitted limits to the physiological increase in the normal excretion of sugar or oxalic acid, why should not those cases of the excretion of albumen in healthy men be regarded as exhibiting simply the physiological increase of a normal process? Nothing prevents us from assuming the occurrence of a physiological albuminuria which takes place just as physiological glycosuria, oxaluria, or other physiological increase of those normal urinary constituents, the discovery of which is attended with difficulty,—that is to say, that certain conditions may cause them to make their appearance in larger and therefore more demonstrable quantities. The conditions with regard to the appearance of albumen are by no means more unfavorable than for that of other substances.

As regards the origin of the urinary albumen in all these cases, there is no lack of a source extremely rich in this substance. This, as a matter of course, is the blood flowing in the Malpighian tufts, for the blood of the interstitial vascular system, or the lymph, or any other source, under normal conditions, needs not be considered, and this statement requires no further explanation. The albumen therefore must have its origin in the blood of the Malpighian tufts. This statement corresponds, to some extent, with the theory of the excretion of urine, which for some time past has been advanced by Küss (26), von Wittich (27), and Henle (28), but which has met with few supporters and many opponents, in the first place, because it makes it difficult to explain the absence of albumen in ordinary normal urine, and likewise because it is not altogether in accordance with the fact recently discovered of the slight diffusibility of albumen as a colloidal substance. After what has been stated with regard to physiological albuminuria no importance can be attributed to the first objection, the fact being that normal urine is often found to be albuminous, if properly examined, and all causes may be assumed to account for albumen being a normal constituent of the urine, though its presence cannot always be demonstrated. As regards the other objection,

the slight diffusibility and capacity for filtration of albumen, this must, at least to some extent be waived, inasmuch as membranes, permeable by albumen, are to be found throughout the body. On the other hand, quite recently the belief has arisen that the escape of albumen from the Malpighian tuft is prevented by the epithelial investment of the vessels; and this theory is now acquiesced in by all those who endeavour to explain the absence of albumen in the fluid which leaves the tuft, this view being adopted especially by Runeberg, Heidenhain, and Cohnheim, as already mentioned.[1] Nothing could appear more simple and intelligible than this explanation, and it might be adopted forthwith if it could relieve us of any one difficulty, and did not too obviously contradict patent facts. In the first place, it contradicts the well-known fact, insisted upon always, except only upon this occasion, that egg-albumen passes without difficulty into the urine, and, as has been shown by Nussbaum on frogs, and Ribbert on rabbits, through the vessels of the Malpighian tufts. The explanation for this is sought for in the greater ease with which egg-albumen passes through a filter, an explanation to which as yet no one has raised any objection, and the same will apply to peptone. This marked capacity for filtration has been incidentally shown on dead animal membranes. It is obvious that, when with regard to these cells, it is only a question of a greater or less capacity for filtration, it is impossible to understand how they can be completely impermeable for the normal albuminous constituents of the blood. For there is no doubt whatever that these albuminous substances are capable of filtration, especially from saline

[1] A different view is adopted by Ribbert in his recent work on 'Nephritis and Albuminuria' (29). He attributes no importance in this respect to the epithelial covering of the Malpighian tufts, but thinks that these capillaries, which, according to him, are almost entirely without nuclei, possess more cohesion than other capillaries, and therefore prevent the escape of albumen. It is hardly possible to take this view even if we choose to admit as correct Ribbert's isolated assertion with regard to the paucity of nuclei of those capillaries. There are, moreover, no capillaries which are impermeable by albumen, and experiments made with this view on kidneys both before and after death have demonstrated that there is no difference between these and other organs in this respect; and, lastly, the investigations of Frommann and of Riemer on argyria have shown that solutions of silver pass as readily through the Malpighian tufts as through any other vessel (30).

solutions, such as the serum of the blood. We are therefore forced to assume that they also filter through the Malpighian tufts, but in an extremely minute quantity, corresponding with their small capacity for filtration.

Let us then consider the epithelium somewhat more closely, as to it is assigned the task of retaining the albumen. This consists, after birth, of extremely flat, thin cells, which most closely resemble the endothelium of serous membranes. "This epithelial investment," says Cohnheim, "is something quite peculiar to the glomerulus, having at the most a distant analogy with the endothelial membrane covering the choroid plexus, the seat of the secretion of the cerebro-spinal fluid, which is almost free from albumen."[1] If the analogy referred-to be real though distant, there is clearly one conclusion to be drawn therefrom, and this is that the capillaries of the Bowman-Müller's tufts permit the escape of a fluid "almost," but not quite, free from albumen. And we are the more justified in arriving at this conclusion, from the fact that it is in complete harmony with what we know regarding other normal transudations (and it may be added abnormal ones also) which pass through capillaries and epithelial membranes, and which all without exception contain large and varying quantities of albumen. Not merely does the cerebro-spinal fluid contain albumen, but the transudations of all serous membranes which have an epithelial investment are albuminous, and normally so, as is evidenced especially by the pericardial fluid; in the majority of the other sacs too little fluid is present for purposes of demonstration. The aqueous humour which transudes in the anterior chamber of the eye, which has a complete epithelial covering, contains albumen, the perilymph and endolymph of the auditory labyrinth, which are certainly separated by epithelium from the secreting capillaries, contain albumen. Certainly all normal transudations (to say nothing of abnormal ones) contain albumen; in whatever part of the body fluid escapes from the blood *without the interposition of specific glandular epithelium*, it contains albumen, even when it has to pass through an epithelial covering, as well as a capillary wall. It would therefore be marvellous if the

[1] This fluid contains albumen in the proportion of ·3—3 per 1000.

fluid which escapes from the glomerular vessels of the kidney did not contain albumen.

It is true that we assume that this fluid is in reality only a transudation, that is, pressed out by filtration, but not a product of secretion, and in this we are in complete harmony with the general opinion.[1] Quite lately Heidenhain alone has expressed a different opinion to the effect that the process which goes on in the kidney is a real secretion, just as that of other glands, since the excretion of water in the kidneys depends upon the functional activity of those cells of the glomerular vessels, the quantity of blood contained in them in a unit of time determining the rate at which the process is carried on. The function of these cells is, therefore, the separation of water (and of the salts by which it is always accompanied) and the prevention of the passage of albumen from the blood. When this function is interfered with, as may occur when the flow of blood through the arteries is checked for a short time, water is separated in smaller quantity and the secretion contains albumen. Heidenhain himself does not attempt to conceal the difficulties connected with this assertion, and, indeed, it is very difficult to attribute the possession of secretory glandular activity to epithelial cells, such as those which cover the glomerular vessels, and are altogether different in their nature from that of all known glandular cells. A greater objection, however, to this assumption is formed by the condition of the secretory function of the kidney, when the blood-pressure is increased without diminution of the flow through the arteries, a state of things which may result from impeded discharge from the veins, the heart's action remaining undisturbed, or from active congestion under conditions of increased afflux without any impediment to the escape of blood. In the first case, the rapidity of the flow is checked, in the second, it is increased. According to Heidenhain the retardation of the

[1] Runeberg, who very justly refers to the fact that all normal and pathological transudations contain albumen, nevertheless asserts, in contradiction to this statement, that epithelial membranes do not permit albumen to pass through them, and for proof of this he relies upon the tears and the perspiration. Both of these, however, are not transudations, but secretions. It may also be remarked in passing that albumen has been found in both.

current must produce a disturbance of the nutrition and function of those epithelial cells, and as a consequence, diminution of the quantity of urine and escape of albumen. In the second case, increased secretion of urine free from albumen is the necessary consequence. But this is not what really takes place. The first condition, increased pressure with retardation of the current, can be very easily produced by entirely or partially closing the renal vein or the inferior vena cava, but it is difficult to form any definite conclusion with regard to the alterations in the quantity of urine under these circumstances, the only positive fact being that the urine is thereby rendered albuminous. In the human subject, in which it is easier to observe quantitative changes in the urine, occlusions of the renal vein or inferior vena cava very rarely occur, and almost always under conditions in which the arterial blood-pressure is reduced, as takes place in marantic thrombosis, or in occlusion due to cancer, &c. Only in very rare exceptions has arrest of the escape of blood from the kidney been observed in conjunction with well-maintained cardiac activity and normal arterial pressure. For a case of this kind we are indebted to Bartels (31). It was one in which there was impeded escape of the blood of the renal vein, in consequence of thrombosis of the inferior vena cava, occurring in a very robust man, forty-four years of age; it was, therefore, not a case of ordinary congestion as a result of defective action of the heart, in which there is also diminution of arterial pressure. Now this patient, as Bartels states, passed "enormous quantities" of urine (on an average 1640 ccm. daily in spite of most marked œdema), generally containing a considerable quantity of blood, and with a specific gravity varying from 1·011 to 1·013, and always containing much albumen and a sediment of red blood-corpuscles and epithelial and other casts. This case furnishes evidence of a more decided character than any experiment against Heidenhain's view of the function of these epithelial cells. For in spite of the retardation of the current, which obviously existed, there was no diminution, but a considerable increase in the quantity of the urine. According to our view, which moreover is admitted as an explanation by Bartels, both phenomena, viz. the increased quantity of urine

and the escape of albumen and blood, find their interpretation in the increased pressure in the glomerular vessels. Heidenhain's supposition is also contradicted by other clinical experiences, not perhaps in such a striking manner as by the above-mentioned case, because the conditions are less simple. They are fully deserving, however, of being mentioned. Thus it is a well-known fact that in certain stages of chronic nephritis and nephro-sclerosis, a very watery and more or less albuminous urine is secreted, whereas the glomerular vessels are in a great degree or almost entirely destroyed, and their epithelial investment has completely perished. Under these circumstances no secretion of water can occur from these parts.

The second case, increase of blood-pressure with acceleration of the current, has often been a matter of experimental and clinical observation, and will be thoroughly discussed in subsequent pages. It may here be remarked that under these circumstances, in which there is no kind of question of any disturbance of nutrition and function, albuminuria may occur, a fact utterly irreconcileable with Heidenhain's view, but perfectly intelligible if we adopt the filtration theory. How it comes to pass that albuminuria is not to be discovered in every case of increased blood-pressure of a like kind, will be discussed later on, when we come to deal with this subject (S. III).

A consideration of these details will cause us to accept without scruple the generally received view which forms the starting-point of the above description, namely, that the fluid which escapes from the glomerular vessels is a transudation, formed in accordance with the laws of filtration, and not a glandular secretion. In this respect I adopt Ludwig's theory of the secretion of urine, but I differ from him in regarding the filtrate as not wholly free from albumen. On the other hand, in another respect, I completely assent to Heidenhain's theory, in so far as he considers that the epithelium of the tubuli uriniferi are actively employed in the secretion of certain specific constituents of the urine. It appears to me that this combination of the two theories satisfactorily gets rid of all the difficulties particularly connected with the simple filtration-theory, and which have

led Heidenhain to assume that the epithelial covering of the glomerulus is an agent in the secretion of water, as I shall show further on. To facilitate the comprehension of what follows, I will now only observe, that according to my view, the assumption universally received and which forms the basis of every theory, *i.e.* that the water of the urine is normally furnished only by the glomerular vessels, is thoroughly untenable, and that we ought rather to assume that the vessels in question supply only a portion, though certainly the larger portion, and that the remainder is yielded by the true secretory elements of the kidney, the epithelium of the uriniferous tubules. This last supposition is based upon the fact that we cannot imagine a glandular secretion without water, unless we picture to ourselves that the whole process consists in nothing else but fatty degeneration of the epithelium, as takes place for example in the formation of the sebaceous secretion of the skin. Nothing of this kind, however, can be thought of in connection with the kidney. Moreover, direct proof that these epithelial cells, *i.e.* those which invest the tubuli uriniferi, are actively engaged in the secretion of water, has been furnished by Nussbaum (32). The urinary water has therefore two sources, and is partly a transudation and partly a secretion. A firm grasp of this fact is an important aid in explaining the different changes which take place in the urine under various conditions.

If therefore the fluid which escapes from the glomeruli be a transudation, is there any reason why it should form an exception to all other transudations, as regards the albuminous constituent which is a feature common to all ? Certainly the glomerular vessels are exceptional in this particular, that in them the lateral pressure is greater than in any other capillary system of the whole body.

We know, however, that *ceteris paribus*, the quantity of albumen which filters through an animal membrane from an albuminous solution, increases with the increase of pressure, but not in a degree proportional thereto. And in fact this increase is an absolute one, and not relative in proportion to the total amount of the filtrate passing through in a certain unit of time, inasmuch as, with the augmented pressure, the degree of increase of the quantity of water which passes is

greater than that of the albumen. In other words, the greater the pressure under which filtration takes place, the greater the quantity of the filtrate in general, and the smaller its percentage of albumen ; but for all this, for equal periods the total amount of the filtered albumen is greater when the pressure is high than when it is low. It follows, therefore, as regards the glomerular vessels, that they must yield a transudation richer in water, but poorer in albumen than any other set of capillaries in the whole body.

The investigations which have been carried on in Ludwig's laboratory with reference to the phenomena of the formation and flow of the lymph, which are not influenced merely by changes of pressure, have caused us to regard as inadmissible the application of the laws of filtration, as they are found to apply in the case of animal membranes outside the body, to the processes of transudation which take place within the body. But although the connection between the formation of lymph and transudation from the capillaries is a very close one, yet the lymph which escapes from an opening in a lymphatic trunk cannot be regarded as a simple transudation, and just as little reason would there be in supposing that the conditions of the current in such a vessel were the same with those of transudations from the capillaries, so that our ideas with regard to the one could be simply transferred to the other. The fluid which escapes from a lymphatic vessel differs essentially and in many respects from transudations due to congestion of capillaries. Morphologically, the distinguishing characteristic of "lymph" is the abundance of colourless cells which it contains ; chemically, "lymph" and transudations are very dissimilar indeed. It is a well-known fact that "lymph" coagulates on exposure to air, whereas the real transudations never exhibit this phenomenon, unless they are mixed with blood, or "lymph," or products of inflammation. The transudations, without exception, contain either no potash whatever, or at most only very slight traces, as little, indeed, as the serum of the blood, when this is obtained perfectly free from corpuscles ; the lymph, on the other hand, invariably contains potash in considerable amount, for the analyses of Hensen and Dähnhardt show a proportion of more than three per cent. after incineration (33).

It might be considered, therefore, that these differences would suffice to indicate the diversity between the two fluids, and to suggest an origin, partly perhaps, but not altogether identical. Moreover, that observations made upon the discharge of fluid which takes place from an opening in a lymphatic vessel do not justify the conclusion that the process is identical with a transudation, is demonstrated by the fact that the current from such a vessel entirely ceases after a certain time, whereas there is no ground for supposing that transudation has ceased, and besides this, the stagnant current of lymph can be made to resume its movement, by active and passive movements, such as gentle rubbing, these manipulations producing no effect on transudation. There is, lastly, this fact, that even the total occclusion of all the lymphatics of a portion of the body is never followed by local œdema (34). If the current in a lymphatic trunk were in complete analogy with transudation from capillaries we should be justified in expecting that occlusion of the former would cause the transudation to accumulate and that œdema would result. But all these facts prove that the lymph-current is altogether unessential for transudation, and observations upon it cannot therefore enable us to draw any positive conclusions with regard to the conditions under which the latter process takes place.

The function of the lymphatics is that of auxiliaries to the veins. Fulness of the lymphatics indicates inadequacy on the part of the veins for the removal of transudation, but diminution in the current of lymph, so long as the flow through the veins is free, by no means indicates diminished transudation, for the power of absorption and of removing transuded fluids, possessed by the veins, can be increased far beyond the normal amount, as is evidenced by the non-appearance of œdema when the lymphatic current is arrested: the veins can, without aid from the lymphatics, fulfil their task, the removal of the transuded fluids, but the latter cannot thus dispense with the assistance of the former. It follows that the non-appearance of œdema under conditions of increased arterial pressure, the current through the veins being unimpeded, is no proof that the transudation is not increased. There is an increase in this respect, and also in

the rapidity of the current and the removal of the transudation by the veins, and only in rare cases, when these means do not suffice, does the current of lymph become more copious (35).

These are facts the truth of which has been firmly established by experiment, and they are in harmony with all pathological experiences. In addition to this, everyday practice at the bedside clearly teaches us that the formation of transudations, œdema, depends directly upon the pressure in the capillary system, and particularly that the quantity of the transuded fluid rises and falls with the pressure.

Runeberg's views, put forward some years ago, differ from the above account, and contradict the universal assumption with regard to the cause of the passage of albumen through animal membranes by referring the phenomenon to a diminution of pressure below the normal height. But this theory, as Heidenhain has shown, has its origin in the fact that Runeberg has misinterpreted his own experiments. Properly estimated, and due care being taken to keep separate the absolute and relative amounts of albumen, they are in complete harmony with the law just described, and are as confirmatory thereof as the recent investigations of D. Newman (36), Gottwalt (37), von Bamberger (38), and numerous clinical experiences, to say nothing of the researches of an older date. If all the clinical facts are not sufficiently clear and simple to be regarded as demonstrative, they are nevertheless in complete harmony with our statement; on the other hand, it is difficult to reconcile them with Runeberg's views, and we can do so only by adopting forced premises, not altogether in harmony with one another. That this is so is best evinced by his attempts to explain by his theory all forms of albuminuria, as will be shown in subsequent pages of this treatise. I may here just refer to the simple and well-known example of ascites, the cause of which is increased pressure in the vena portæ, whereas according to Runeberg, it must necessarily occur when the pressure in the vena portæ sinks below the normal. This point is passed over by Runeberg, he mentions only the fact that when an effusion has once taken place into the abdominal cavity, the albumen of this transudation increases with the quantity of fluid, the certain result being that the difference between the

pressure inside and outside the blood-vessels, and consequently the filtration-pressure, become diminished. This phenomenon, which he regards as supporting his view, has been simply explained by F. Hoppe (Seyler) as exemplifying the generally received view concerning the influence of pressure, that is to say, that with increasing pressure more water is absorbed, the fluid becomes concentrated, and as a matter of course the quantity of albumen is thereby increased (39).

Notwithstanding the above objections, there is a sense in which we can accept Runeberg's assertion that albuminuria is caused by diminution of the blood-pressure in the kidney, but we do not agree with him in supposing that the altered circumstances permit albumen to escape in places where hitherto no such passage had occurred, the true explanation being that relatively less water escapes, the quantity of albumen is therefore increased, and the albuminous transudation is rendered more distinct—it becomes, that is, an appreciable phenomenon. The diminution of pressure therefore facilitates the discovery of the escape of albumen. In addition to this, the diminished pressure favours the appearance of albumen by reason of the influence it exerts upon the second factor concerned in the formation of urine, viz. the secretion of the epithelium of the uriniferous tubules, a factor altogether neglected by Runeberg, and all others who have endeavoured to explain the phenomena of albuminuria. We shall explain at length in a subsequent page how albuminuria occurs through the collective effects on both factors, of a reduction in blood-pressure when this is present (S. III).

Our principal task now is to show how it is that under normal conditions albuminuria, *i.e.* the manifest presence of albumen in the urine, is not an ordinary occurrence, and with regard to this, the second factor, the secretion of the true glandular epithelium, is of considerable importance.

This secretion, containing the so-called specific constituents of the urine, dissolved in water, and free from albumen—a fact requiring no demonstration with such views as are now prevalent—passes into the tubuli uriniferi and mixes with that transudation from the glomerular vessels, which, as we have seen, is extremely poor in albumen. It

is pretty certain that no direct increase of albumen takes place in the further passage through the urinary tubules, and there is no ground for the supposition that, owing to the absorption of water with or without salts, the contents of the tubes become relatively richer in albumen. It is well known that no decision has yet been arrived at on the question as to whether any absorption really takes place from the uriniferous tubules, as is assumed in Ludwig's theory of the secretion of urine, but in opposition to the general and well-grounded opinion on this point. It is, nevertheless, conceivable that absorption might occur, if not in all parts of the urinary tubules, at all events, perhaps, in that portion contained in the renal medulla, which is so copiously supplied with veins and lymphatic vessels, that is therefore in the straight tubes, and in the ascending and descending limbs. But if this be so, we could not expect that albumen actually in a state of solution should be excluded from absorption. For if this latter process involves all portions of the fluid contents of the urinary tubules, it would affect the albumen as one of them; but if, as Ludwig's theory requires us to assume, materials are selected for absorption, the specific constituents of the urine being excluded, the albumen must certainly be absorbed. For least of all can the albumen be considered as a specific constituent, and when it is present in a state of solution, and not merely floating about in a fluid, it is constantly taken up wherever absorption is going on.

The urine, which therefore represents a mixture of the transudation from the glomerular vessels and of the secretion of the uriniferous tubules, is probably on the whole more concentrated, that is, richer in other fixed constituents, but poorer in albumen, than the transudation peculiar in that respect, and whose proportion of albumen must be less than the lowest percentage of any transudation. If we represent the percentage of albumen in the transudation from the glomeruli as a, and the amount of the secretion from the glands which is added thereto as n, then, if no absorption takes place, the amount of albumen in the urine as a whole will amount only to $\frac{a}{1+\frac{n}{100}}$. It is, therefore, not hard to

imagine that the demonstration of albumen in normal urine is beset with difficulties. It is likewise to be supposed that differences may occur even in the normal condition, and that the discovery of albumen sometimes succeeds and at other times fails. Not only does the pressure in the glomerular vessels vary under physiological conditions, a fact which admits of no doubt, the result being that the fluid which transudes from them contains sometimes more and sometimes less albumen, but in addition, that portion of the urinary secretion which depends upon true glandular action, being under the influence of physiological conditions, is sometimes abundant and sometimes scanty. This latter fact is equally certain, and corresponds with our knowledge of all other glands. The more productive the activity of the glandular epithelium, the poorer will the urine be in albumen, other conditions being equal, and the more difficult the demonstration, and the reverse. And thus it comes to pass that in consequence of the varying action of the two sources of the urine under physiological conditions, different combinations can be imagined, as a result of which the discovery of albumen may be easy, or difficult, or impossible, that is to say, that albuminuria may or may not be demonstrated, according as both sources either co-operate or act in opposition with reference to the quantity of albumen in the urine. Increase of the pressure under which filtration takes place in the glomerular vessels, and of the secretion in the epithelium, will cause albuminuria to be absent (*i.e.* obvious excretion of albumen), diminution of both those factors will produce its appearance, increase of the one and diminution of the other will produce a result dependent upon the influence which preponderates, as will be shown in special cases in subsequent pages (p. 39 *et seq.*). If to all these considerations we add the individual differences with regard to filtration and secretion, which are properly allowed to play so common a part in the dissimilar reaction of different individuals under physiological and pathological conditions, we shall have scarcely any difficulty in understanding the fluctuations to which physiological albuminuria is subject.

All this discussion, all the matters of fact and the conclusions deduced therefrom, are therefore incorporated in

the assumption that under normal conditions a fluid extremely poor in albumen transudes into Bowman-Müller's capsules, and that physiological albuminuria is dependent upon the albuminous contents of this fluid. An assumption certainly is not equivalent to a direct proof, but, in the absence of any such, is perfectly justifiable so long as it cannot be shown that there is any error in the facts upon which it is based, or that experiences and analogies are unjustly brought forward in support, or lastly, until we have direct proof to the contrary. Not long ago it was regarded as impossible, by direct observation or examination, to prove whether the fluid contained in the Bowman-Müller's capsules is albuminous or not. To Posner is due the credit of having indicated a method of directly observing that transudation by an improved method of boiling—and to him we are indebted for the positive proof that in many cases of pathological albuminuria the source of the albuminous secretion is to be found in the glomerular vessels—a fact which until recently could only be guessed at with more or less probability. But Posner has gone further than this, for he believes that he has positive proof for his assertion "that the kidneys under normal conditions are not albuminuric, that is, that no albuminous substances transude from the blood-vessels of the glomeruli." He holds this opinion, because in normal kidneys he has not been able to detect any excretion of albumen in the capsules, but he manifestly over-estimates the efficiency, as a test, of the method of boiling and of the microscope. For there is no doubt that albumen may be and is present without becoming visible after boiling.[1] I will not discuss the fact that in normal kidneys, even after boiling, no trace of coagulated albumen can be discovered in the lymphatics, which, according to Ludwig and Zawarykin (40), surround the vessels and the urinary tubules, and, according to Ryndowsky, (41) probably enter the capsules and wind round the glomerular vessels. Can there be any doubt of the presence of lymph and albumen, simply because they are invisible? But more than this; albuminuria can be induced in various ways, and the most minute examination may fail to

[1] What is true of boiling holds good also with regard to hardening and coagulation by alcohol.

detect any excretion of albumen in any part, and especially in the capsules of the boiled kidney or after hardening by alcohol.

This may be very easily and securely effected in rabbits, by rapidly heating them (see page 48), or by injecting a very small quantity of phosphorised oil ($\frac{1}{3}$—$\frac{1}{2}$ cctm. of a solution of one part phosphorus to eighty olive oil) under the skin (see S. IV). Often immediately after a single injection, but always after a repetition on the second or third day there is evident albuminuria, the urine contains finely granular casts and occasionally renal epithelium easily recognisable. After boiling the kidneys and hardening them in alcohol, they exhibit changes corresponding with the degree in which they have suffered. These are sometimes of a transient character and take the form of patches of hyperæmia, minute hæmorrhages, swelling of the epithelium, and perhaps coagulated albumen in the capsules alone or in the urinary tubules as well, or there may be nothing remarkable, no albuminous coagula or none at least in the capsules. The same description holds good of albuminuria occurring after rapid heating, and also of the same symptom resulting from the injection of egg-albumen into the blood. Miss Bridges Adams (42), in a series of experiments under Cohnheim and Weigert's direction, noticed albuminuria as a constant phenomenon in six cases in which the injection was made, and in all of them without exception the boiling method failed to detect any albuminous secretion in the capsules. And yet albumen has certainly been present in the capsules, for, as Ribbert has shown, it really escapes from the glomerular vessels and from them alone under those circumstances, and may be seen in the capsules after coagulation. But that it must necessarily be visible under all circumstances is untrue, as the above-mentioned experiments strikingly show, and as might *à priori* have been expected. For in the normal condition the glomerular tuft completely fills the capsule, and is in such close contact with it that even "with a magnifying power of 800 diameters applied to very fine sections of a pig's kidney removed during life, and placed in a freezing mixture, we are able to distinguish only a scarcely measurable cup-shaped fissure between the capsular and the glomerular epithelium."[1] This

[1] S. W. Krause, 'Allg. u. mikrosk. Anat.,' 1876, S. 246.

fissure represents the space occupied by the minimal amount of transudation present at that moment, and a consideration of these facts will make it appear quite natural that the infinitesimally minute trace of albumen contained in this minimal transudation, should remain invisible even after coagulation. To obtain some approximate idea as to how far the minute traces of albumen with which we are dealing are within the limits of vision, supposing that they can be thus included, it is sufficient to take a simple estimate based on the well-known dimensions of a Malpighian corpuscle, and the thoroughly admissible supposition that the corpuscle is perfectly globular in form. The diameter of such a corpuscle, and therefore of the Bowman-Müller capsule, is on an average ·25 mm., its capacity is therefore $(\frac{4}{3} r^3 \pi)$ ·00818 cbmm. If we assume that this space is not, as is really the case, quite completely occupied by the glomerular vessels, but only to three-fourths of its extent, so that a complete fourth of the contents is taken up by the transudation, our supposition will have exceeded the most exaggerated demands. There is then at a given moment ·00204 cbmm. of transudation in a capsule, or, expressed in weight—for it is somewhat heavier than water—·00206 milligrammes. This transudation, as we have explained in a previous page, must be poorer in albumen than the poorest normal transudation, than, for example, the cerebro-spinal fluid. But if we here also advance the most exaggerated assumption, and estimate that the quantity of albumen contained in the fluid of the capsule is equal to that in the cerebro-spinal fluid at its highest amount (see p. 24), that is, 3 per 1000, we should have ·00000618 milligrm. of albumen in ·00206 milligrm. of transudation. That is the result of a calculation based upon figures exaggerated beyond all belief in order to obtain as large an amount of albumen as possible; and the most extravagant fancy would find a difficulty in supposing that this quantity, even when compressed into a spot, would be visible under the microscope, to say nothing of its being more or less diffused in the capsule, and therefore necessarily only partially exhibited on a transverse section.

As a matter of course the circumstances are different under abnormal conditions, when more albumen transudes,

when the transudation is stemmed in consequence of impediments to its escape and its albumen is increased owing to the absorption of water, and when, lastly, this stemmed-up, copiously albuminous, fluid compresses the glomerular vessels, and forces them away from the capsule. The albumen thus becomes visible, but by no means always in every capsule, even where they are all involved, though in varying degrees of intensity, in a diffuse process, but it very frequently happens that only more or less of them present traces of albumen, the remainder being apparently free from it, because the accumulation has not been sufficient to make itself visible. Very instructive in this respect is the condition which is produced after prolonged stagnation of urine, the result of applying a ligature to the ureters. Here at a certain period (see S. III) we find the urinary tubules and the capsules distended, and in several of the latter the glomerular vessels forced back by coagulated albumen, in others by a more or less broad and clear ring of fluid without any visible trace of coagulated albumen. This fluid must, however, be albuminous, and it does contain albumen (as appears on examination of the urine obtained immediately after the ligature has been removed from the ureters), and much more than there can possibly be in the normal transudation under the most favorable hypothesis. And as, notwithstanding this, it appears clear and transparent, we cannot expect, as a general rule, to see coagulated albumen, or anything more than a small clear ring, between the glomerulus and the capsule.[1]

It follows, therefore, that there is at present no reason for supposing that microscopical examination can furnish any proof of the presence or absence of albumen under normal

[1] The application of a ligature to the ureters of rabbits can be so easily and neatly effected, that any one may convince himself without any great difficulty of the correctness of the above statement. The best proof, however, is furnished by a repetition of Posner's own description of the appearances which the kidneys present after ligature of the ureters for two or four hours, after which we know, and Posner himself asserts, they are in a state of acute congestion, and red blood-corpuscles (?) are mixed with the secretion. "On microscopical examination in this initial stage, besides the excessive capillary hyperæmia and partial hæmorrhages, all that is seen is dilatation of the urinary tubules, but there is no coagulable exudation and no unequivocal casts."

conditions in the fluid yielded by the capsules. We must continue to maintain the assumption, which is supported by other considerations, that the transudation which exudes from the glomerular vessels is feebly albuminous in its normal state, and this supposition furnishes us with a means whereby we may explain the presence of the minute proportion of albumen in normal urine, and the occurrence of physiological albuminuria, in a manner at once complete and satisfactory (see p. 22, *et seq.*).

III. The Dependence of Albuminuria upon Alterations in the Blood-Pressure.

Proceeding from the supposition, which we have shown to be perfectly tenable, that the normal fluid in the Bowman-Müller's capsules is a transudation yielded by filtration, and as regards its constitution dependent upon the pressure of the blood, many experimenters have endeavoured to produce albuminuria by altering the general blood-pressure of the body, or the pressure in the vessels of the kidney. It is necessary that we should minutely discuss these attempts, on the one hand, because endeavours have been made to obtain conclusions therefrom as to the origin and secretion of the urine and the importance of the blood-pressure in the process, and secondly, because these experiments have been made to serve as a basis for the explanation of pathological processes. Unfortunately we find many difficulties in forming conclusions as to their value for the above purposes, and these we shall presently endeavour to point out. The conditions are not sufficiently simple to justify the expectation that the discovery of albumen in the urine would enable us to decide as to how far its production had been influenced by any possible change of pressure in the renal circulation. For, although we are correct in supposing that the transudation in the Bowman-Müller's capsules is subject to the laws governing filtration-pressure, we are by no means to conclude that the urine as a whole is placed under the same rules. The urine is, as I have already explained, to be compared to a stream fed by two sources, which differ from each other as

regards the quantity of water they yield and other conditions, and are governed by laws not altogether similar in character. The one source, the transudation from the glomerular vessels, certainly yields more water and contains a little albumen in addition to the ordinary constituents of all transudations; the other, the secretion of the glandular epithelium in the urinary tubules, is, perhaps, less abundant as regards quantity, is free from albumen, but loaded with the specific constituents of the secretion. The former is, as I have before said, subject to the laws of filtration, whereas the glandular secretion is, as we all know, governed by other influences. In the second place, the fact has been overlooked that, although with increased pressure the quantity of the filtrate becomes augmented, its composition becomes changed, and particularly that the proportion of albumen undergoes a relative diminution, and therefore that increased pressure renders its demonstration more difficult owing to the greater dilution of the urine. Thirdly, as has been fully recognised since Virchow's investigations (43), the vascular distribution in the kidney is so peculiar in its arrangements, and so different from that of other organs, that alterations of the general blood-pressure, or of the pressure in the principal vessels going to or leaving the kidney, can and must affect unequally the two sources concerned in the secretion of urine. Fourthly, as a result of certain changes in the blood-pressure, the nutrition of the walls of the vessels or other membranous structures, and also the epithelium, may become affected, and these may in consequence become abnormally permeable for albumen. Fifthly and lastly, the condition of the flow of lymph in the kidneys is also dependent upon the state of the blood-pressure, and is not without influence upon the composition of the urine. The fact must also be mentioned that, at all events, increase of pressure in the venous system is accompanied by alterations in the purely mechanical conditions of escape, as was shown by Ludwig some years ago (44).

These facts and considerations teach us how complicated the conditions are, and how difficult it is to estimate the results of experiments upon the changes in the blood-pressure in the kidneys. If we also consider that in certain series of

experiments, undesigned and yet potent conditions have been introduced,—conditions frequently overlooked, or their action erroneously interpreted,—we shall not be surprised that some of the experiments with regard to the influence of alterations in the blood-pressure have led to results mutually contradictory, while others, even where the results were congruent, as, for example, in venous congestion, have had very discordant explanations attached to them.

The conditions with regard to the increase of blood-pressure in the arteries are the simplest and plainest of all, yet the influence of this change upon the production of albuminuria has been made the subject of very different statements by the various investigators. It is true that many of the experiments have either been incorrectly performed, or they lead to a conclusion just the opposite to that which their performer deduced from them. If, taking our stand upon pure theory, we ponder over the views developed in the foregoing pages, and reflect upon the necessary consequences of the increase of arterial pressure within the kidneys, we find that the result must be a combination of the influence of the increase of pressure upon the transudation from the glomerular vessels, and of the influence upon the secretory elements of the uriniferous tubules. The condition of the transudation may be predicted with perfect certainty, it will, as has been already explained (see p. 28 *et seq.*), be more copious than under normal conditions, and its percentage of albumen will be diminished. The condition of the proper secreting elements of the kidney cannot be predicted with a like certainty, for they cannot be subjected to a separate experimental test, as is the case with the elements of other glands. Following, however, the analogy of other glands, especially the liver, we may assume that with the increase of arterial pressure (and of the rapidity of the current) the secretion will, at all events, be increased up to a certain point, and no albumen will appear therein. The result, as regards the urine, will be an increase in its quantity ; on the other hand, it will contain a still smaller percentage of albumen than is assumed to be normally present. If we take, as before, the percentage of albumen in the transudation as a, it will, under the assumed conditions, be $a-x$, and the

quantity of the secretion yielded by the epithelium, instead of n, will be rather $n+y$, so that now the urine as passed will contain only $\frac{a-x}{1+\frac{n+y}{100}}$ of albumen, always supposing that no absorption takes place from the urinary tubules.

It follows, therefore, that if the experiments be really successful, and free from all objection, we may expect to find less albumen than normal in the urine; but simultaneously with the increase of pressure, other conditions must prevail, which, without causing any other alteration, diminish the quantity of urine, and therefore raise its percentage of albumen, enabling us to demonstrate its presence.

Let us consider once more the experiments that have hitherto been made, and their results in particular. There are various methods which may be used, and for the most part have been used, for the purpose of increasing the pressure in the arteries in general, or in those of the kidneys only. The greatest increase of pressure in the aorta can be produced with ease and precision by electrical irritation of the cervical spinal cord, by producing dyspnœa, by poisonous doses of strychnia, digitalis, &c. All these means have been employed to produce albuminuria, and it may be asked, with what result? All observers agree in stating that invariably during the rise of the aortic pressure, and at its height, the flow of urine altogether ceases; subsequently, when with the falling pressure secretion is restored and is more copious than before, a (gradually diminishing) excretion of albumen takes place. In the first period there is a general spasm of the small arteries, which causes the pressure to rise in the great trunks. The small renal arteries, as Grützner (45) has shown, participate in this spasm, and the natural result is the exhaustion of the flow of urine; with the subsidence of the vascular spasm, as a matter of course, there is increase of pressure in the arteries and capillaries of the kidneys (as in other organs), and exceeding the original height in a degree corresponding to the previously abnormal increase in the aorta, until the dilatation of the vascular channels causes the normal conditions to be restored. In this period, in which the pressure and the rapidity are both increased, with active

vascular dilatation as a result, the increase in the secretion of urine is accompanied by appreciable albuminuria.[1] Can we now draw any conclusions therefrom with regard to the influence of the increased blood-pressure upon the albuminuria? At the first glance, the position of cause and effect seems so clear that this conclusion might be accepted without any hesitation. But there is one important fact of a contrary character, namely, the defective supply of arterial blood while the vessels are constricted. It makes no difference if, as is generally stated with regard to these experiments, the flow of urine altogether ceases during the period of vascular spasm, and the albuminuria consequently afterwards appears with the returning secretion of urine, for undoubtedly this phenomenon is to be referred to the previous period, unless hæmorrhage sets in. We shall see later on that, when the supply of arterial blood is considerably reduced, but not altogether cut off, the secretion of urine continues, and is accompanied by manifest albuminuria, the causes of which will then be described. The only question at present is to establish the fact that even the diminution of the supply of blood, which occurs in the first period, explains *per se* the albuminuria, and that, consequently, these experiments are of no force at all as arguments in favour of the influence of increase of the arterial pressure.

These experiments would have far greater demonstrative force, for or against the existence of this influence, were it possible so to perform them that this effect of pressure, and nothing else, came out as the resultant. The idea formerly was that the pressure in the aorta and other

[1] Litten (46), who has performed the above-mentioned experiments with the same result, especially with regard to the albuminuria, and was disposed to consider this latter as caused by the increase of the arterial pressure, explains this now as the consequence of the dilatation of the vessels and of the resulting retardation of the current of blood, and he finds in this a positive confirmation of Runeberg's views, which he had regarded as correct (see pp. 2 and 31). But inasmuch as the condition in question is that of an *active* dilatation, as a matter of course there can be no retardation of the current of blood, a fact which is also incidentally revealed by the increase in the quantity of urine. It is evident that other conditions prevail when the pressure is raised in consequence of impeded escape from the veins and *passive* vascular dilatation.

arteries could be easily raised by placing a ligature on peripheral arteries. Observations with the kymograph have, however, taught us that that idea is erroneous, and have shown that the organism sets in action compensatory arrangements whereby, even when large vascular districts are cut off from the circulation, the general blood-pressure is kept at the normal height. The pressure in the aorta can be greatly increased only by ligature of the carotids or, indeed, of one carotid, but, as Nawalichin (47) has shown, this is merely in consequence of the irritation thereby induced in the vaso-motor centre and of the general vascular spasm which ensues, just as takes place in the first series of experiments which we have already discussed. Without tying the carotids the pressure in the aorta can be certainly and considerably increased only by applying a ligature to the vessel itself below the diaphragm, but above the kidneys. But the obvious consequence of this is that the supply of blood to the kidneys is almost, although not altogether, cut off, and the experiment is therefore useless for our purposes. Ligature of the aorta alone, below the kidneys, causes no increase of pressure; Litten, indeed, found a slight increase on tying the cœliac and superior mesenteric arteries in addition to the aorta, but this gradually subsided. These facts will cause us to regard the changes which the urine may present after ligature of the aorta below the renal arteries, not as the consequences of the increased arterial pressure, but as due rather to the incidental injuries, unavoidable in so serious an operation, to the pressure to which the kidneys or their vessels have been subjected, and to laceration and injury of the splanchnic nerves, &c. Considerations of a similar character may be raised against those experiments, by which the blood-pressure can be really raised by placing a ligature on the aorta and other arteries. And a review of the results obtained by various investigators will confirm our scruples. G. H. Meyer, who was the first to tie the aorta in order to decide this question, found albumen in the urine passed afterwards, Robinson, on the contrary, found no albumen, Frerichs found traces of it in a few cases, but invariably considerable quantities when he removed a kidney in addition to tying the aorta. The same result was obtained

by Ph. Munk. Correnti found albumen after simply tying the aorta; H. Cohn also, but only exceptionally, after removal of one kidney; Litten, after tying the aorta alone, found no albumen, and he noticed the same result when both the cœliac and the superior mesenteric arteries were tied, slight increase of pressure being consequently induced. I myself have twice tied the aorta in rabbits, and subsequently found the urine to be albuminous, but I also convinced myself of the very serious character of the operation. To avoid injury Stokvis has applied compression, externally through the abdominal wall, to the aorta in rabbits below the kidneys, by means of a belt fitted with a screw; in two out of three cases the addition of nitric acid to the urine produced a slight cloudiness, which he did not consider to be albumen, but which could not, I think, have been anything else. No importance, however, is to be attached to these experiments, for if in reality only the aorta were compressed below the kidneys, we could not, as above stated, expect with any degree of certainty that the blood-pressure in that vessel would be raised. As a matter of fact, however, in these experiments, the pressure in the abdomen generally, and consequently the pressure to which the kidneys are subjected, must necessarily be increased, and must produce considerable congestion of those organs as a result, and we know that this condition is sufficient to cause albuminuria (48).

What, then, are the conclusions to be drawn from all these experiments? Without raising the pressure in the aorta (ligature of the aorta alone) one observer always finds albumen, another never finds it, a third discovers it occasionally, and traces of it only; by raising the pressure (ligature of the aorta and ligature of, or checking the flow through, other arteries), albumen is constantly found by one experimenter and not by another. My own opinion is, that that series of experiments, as a result of which no increase of pressure can be expected, really proves nothing; and that the albuminuria observed in connection therewith is to be ascribed to the above-mentioned injuries which are incidental to the operation. In those experiments which are attended with real increase of the arterial pressure, one

assertion contradicts the other, and the question still waits decision. At the same time, Litten's experiments, in which no albumen was found, must be allowed to possess the greatest force as demonstrations; for, though it is easy to understand how in such serious operations the urine becomes albuminous owing to incidental causes, we cannot readily imagine, if the increase of pressure *per se* caused albuminuria, how this could be accidentally masked, always supposing that proper methods were used for discovering the albumen, of which in this case there can be no doubt. If, therefore, Litten's experiments be regarded as demonstrative, the result thereof corresponds exactly with our hypothesis (see p. 42). If albumen is, as a general rule, not to be found in urine, increase of the arterial pressure, unless the quantity of urine be simultaneously diminished, will assuredly not cause any albumen to appear.

The experiments in which the renal nerves are divided have yielded results more in harmony with each other, mainly because in performing them it is difficult to avoid pressure or injury to the renal vessels. The latter causes can *per se* produce albuminuria, and this fact may explain why some observers, as in recent times von Wittich and Vulpian, have noticed it after dividing the nerves, while others, as M. Herrmann and Knoll, have observed this symptom only after injuries incidental to the operation (49). Considerable importance must be attached to these negative results, for the same reasons as in the experiments in which the aorta was tied. The same thing then occurs as after the division of the nerves, as a result of which the blood-pressure and the rapidity of the current in the kidney are increased, that is to say, we are not able to discover albumen, because in all these experiments the quantity of the urine is very considerably increased.

Greater results than those of all these experiments are, as already said, to be expected from a method which increases the arterial pressure, and that alone, and at the same time diminishes the water of the urine so that the discovery of the albumen is facilitated. Such a method we possess in the elevation of the bodily temperature. That this should raise the general blood-pressure can be *à priori* inferred from the well-known changes which take place in the blood-vessels

and in the circulation when the temperature of the body is raised.

The increased pulsations of the heart, the enlarged calibre of all the arteries, and their greater fulness, the pulsation of small vessels in which no pulse was previously felt, the red and injected appearance of all visible parts, in a word, all those well-known phenomena of "fluxion" as they occur locally or generally after the operation of warmth, are the unquestionable consequences and proofs of the increase of blood-pressure in the arteries (and of the rapidity of the current). At the same time, in the numerous and multiform experiments of a bygone and recent date, and performed with a view of measuring the pressure, but little heed has been paid to this condition, and thus it comes to pass that with regard to the direct estimation of the arterial pressure, when the general temperature is raised, we have one solitary incidental communication made by Paschutin (50) when he was investigating the lymphatic system in dogs. He found, as a matter of fact, that the pressure was considerably increased. In addition, J. Zadek (51), who performed experiments on men by Basch's method of indirectly measuring pressure, likewise found that the pressure in the arteries becomes abnormally high when the bodily temperature is increased. These statements are, however, inadequate, and I have therefore performed a great number of experiments on rabbits, in which the pressure in a carotid artery was measured while the surface of the body was somewhat rapidly warmed. The detailed account and a description of further experiments made at the same time do not belong to this part of the subject and will follow hereafter. I may here just say that, as a matter of fact, when the temperature of the body is raised within certain limits, the pressure in the carotids is increased, and possibly to an extraordinary degree. If, however, the temperature rises above a certain limit, the intense heat produces its well-known effect upon the heart, and the result is that the pressure falls rapidly and far below the normal degree, and death may ensue.

It is therefore an established fact that, by applying heat to the body, the blood-pressure, and indeed the general arterial pressure in the whole body, can be increased. There is

no cause for the suspicion that, as occurs in the methods previously discussed, the pressure in the aorta alone, or the pressure in certain portions, is increased at the expense of the pressure in other parts, or that this increase is preceded by a lowering of pressure with diminished supply of blood, but in this method from the very commencement, immediately the warming begins, the superficial and deep arteries and those of the viscera become dilated,—a fact in harmony with our expectations, and of which we may fully convince ourselves by examining any organ we choose and especially the kidneys.[1] The first condition for our experiment is therefore present in a manner which leaves nothing to be desired. There is no difficulty with regard to the fulfilment of the second condition, which is, that the quantity of the urine should be diminished. The condition is perfectly fulfilled provided that neither a bath nor a room saturated with moisture has been the means adopted for raising the temperature, for the increase of water given off by the skin and lungs causes the urine to become diminished in quantity. This fact is too well known to require me to allude to it any further.

All that we have to do, therefore, is simply to examine the urine for albumen, before and after heating the animal. I have performed these experiments upon a great number of rabbits.

As a heating apparatus we use a kind of oven or a drying-closet, made of copper-plate, and such as we find in chemical laboratories, with double walls all round for holding water, except at the side, where there is a door. The water is poured in through an orifice in the upper part. Another opening goes through both walls and reaches into the interior of the oven; this is designed to admit the thermometer which measures the temperature of the oven. This latter is of sufficient size to allow a large rabbit (and only such should be chosen for the experiment) to sit quite at ease in it. It stands upon a broad tripod, beneath which the heat is applied. Before placing the animal in the oven, its bladder should be emptied by com-

[1] It need hardly be said that the heating must be a gradual process, in order that the phenomena may appear in the way above described, and that on the other hand sudden exposure to intense heat acts upon the body as a violent irritant, and could only disturb the results of our experiments.

pressing it, and the temperature is to be taken in the rectum. As a general rule, in order to save time the oven was first slightly warmed, and after the introduction of the animal the temperature was more or less rapidly increased. In order to give sufficient air, the door was not quite closed, but left somewhat ajar. When the heat had been applied for a sufficient length of time, the temperature in the rectum was taken while the animal remained in the oven, but with the door open, or immediately after removal; then the bladder was emptied by pressure, and if no urine could be obtained, the animal was placed in a cage from which the urine could escape. Experiments made at the same time with a view of examining the urine as regards its correspondence with the measurements of pressure and its variations, are attended with some difficulty, which, however, may perhaps be overcome by using large animals, such as dogs. For various reasons, however, and for the most part extrinsic ones, I avoid experiments upon large dogs. I have made a few experiments with the view of measuring the pressure in small dogs, but which were not altogether satisfactory on account of the numerous unavoidable difficulties connected with the production of anæsthesia in dogs. And in connection with those few experiments in which it was sought to obtain urine from rabbits while estimating the pressure, there was this drawback that the excitement caused by handling the animal was a thing to be avoided; moreover, in the short interval, urine could not be obtained from the bladder for examination if the organ had been previously compressed. The secretion obtained from the ureters on one occasion was not free from an admixture of blood, which was attributable to the unavoidable irritation and injury of the mucous membrane.

The result in all cases was, the production of albuminuria when the bodily temperature had been increased by 1·5—3·0 C. with sufficient rapidity, or the heat continued for a sufficient length of time, and where this symptom was normally present, it became more marked.[1]

[1] I have already stated (p. 20) that even in animals the urine in the normal condition very often contains albumen. I found it in more than half the number of these I submitted to experiment, and even in those

If the temperature were very rapidly raised to an extreme degree, blood appeared in the urine, and in quantities which did not require the microscope for its detection. In addition to this, and even when no blood-corpuscles were visible, the urine often contained pale hyaline casts, and sometimes finely granular ones, in greater or less abundance.

That the excretion of albumen, or its excretion in an increased degree, is a direct result of the elevation of temperature, and not a secondary phenomenon manifesting itself while the temperature is falling, is demonstrated by two cases in which the animals passed water while in the hot oven, and a portion was caught for examination. This proved to be albuminous, while the urine shortly before had been found free from albumen.

We could *à priori* assume with a degree of probability almost amounting to certainty, that the capsules are the seat of this excretion of albumen. In order to prevent any doubt on this point, I examined the kidneys in many cases with this particular object, killing the animals by cutting their throats immediately after heating them, and then at once removing the kidneys and hardening them by boiling. In proportion to the intensity of the action, there were, in these cases as in those of poisoning by phosphorus (see p. 36), variously strongly marked changes in the kidneys; in those least affected there was only a very slight deposit of albumen, not visible in all the capsules, whereas in the more violent cases there was not only a marked deposit of albumen, but likewise hæmorrhages in many capsules and uriniferous tubules, whilst in others there was no abnormality, with the exception of more or less marked hyperæmia.

With regard to the strictly ultimate cause of this albuminuria, we could at once recognise therein a confirmation of the view already unfolded, that with the increased pressure more albumen escaped, and could be more easily found by reason of the simultaneously diminished excretion of water, if we were quite sure that the purely physical agent, the increased temperature, might not *per se*, modify the filtration of albumen in this direction. That this is so cannot be

recently brought to me and kept in the open air during the summer. It is true that the albumen was often discoverable only after careful examination.

asserted with our present knowledge. We know from the experiments of W. Schmidt (52) and Eckhard (53) that the filtration of saline and albuminous solutions through animal membranes goes on with increased rapidity as the temperature is raised, just as Poiseuille has found to be the case in filtration through glass capillary tubes, but unfortunately, we can draw no certain inference from Schmidt's experiments, which specially related to albuminous solutions, as to what proportion the albumen in the filtrate bore to that of the original fluid. If the percentage of albumen increased in the filtrate, this, as a matter of course, would alone suffice to explain the occurrence of albuminuria without any changes of pressure, whereas the latter alone entered into our considerations. I see, therefore, in these heating experiments, no irrefragable proof of the dependence of albuminuria upon an increase of blood-pressure, but certainly a support to this view, and one which taken in connection with other experiments pointing in the same direction give to it a weight by no means inconsiderable. In any case, these experiments support the theory, according to which filtration takes place in the capsules.

There are, however, sundry other conditions which more or less decidedly increase the blood-pressure in the aortic system, and serve as a test for our view—the most important of these is muscular action. Its influence in this respect has been so constantly observed since the introduction of the kymograph by Ludwig that there can be no doubt whatever in regard to it. Its influence upon the production of albuminuria has not, however, so far as I know, been experimentally investigated, but Litten in describing his above-mentioned experiments states that albumen often appeared in the urine as a consequence of the muscular action of the dogs while they were fastened, whereas the urine previously obtained through a catheter was free from albumen. On the other hand, in healthy men frequently after intense muscular exertion, in parturient women as a consequence of difficult labour (without any previous disorder) albumen has been found in the urine (see p. 19), and also in pathological conditions, in convulsions of all kinds, but particularly in epilepsy, albuminous urine has been very

frequently observed, and its appearance has been often connected with the extraordinary muscular exertion and the accompanying increased arterial pressure.[1] That the pressure in the human subject is increased by muscular exertion may be confidently assumed from the analogous effects observed in experiments on animals, and from the condition of the organs of circulation, resembling as it does that which is produced by raising the temperature (see p. 48), and, besides this, positive proof is furnished by the experiments of Zadek, which have been already referred to. He found that active muscular exertion (running, for example) was accompanied by an increase of the arterial pressure by about one-fourth of the normal degree. But in addition to this, in all muscular exertion of any degree of intensity, as daily experience teaches us, and as countless experiments have proved beyond doubt, the escape of water through the skin and lungs in the form of perspiration and vapour is enormously increased, and as a result the urine contains less water; we have, therefore, a perfect combination of the two conditions which we have laid down at the commencement as essential for distinguishing that form of albuminuria dependent upon blood-pressure, that is to say, increase of the arterial pressure and simultaneous diminution of the water of the secretion, and these conditions are fulfilled under the circumstances before us as decidedly as, or even more so than, when the temperature is raised by experiment. For if the latter could be supposed to be open to any slight objection as to its validity as a demonstration, nothing of this kind can arise with respect to muscular exertion, and I cannot conceive of any well-founded hesitation in accepting the explanation which I have given. But for all this, the attempt has been made to explain the process in another way, and this has been done from the standpoint of Runeberg's theory by Edlefsen, who has been followed by Runeberg himself. Confiding in the correctness of this theory, which assumes that the escape of albumen is due to a diminution and not to an increase of the normal pressure in

[1] To meet any possible objection it may here be remarked that in the muscular exertion of the healthy subjects, and also in the majority of the pathological cases, there was no symptom of dyspnœa, or risk of suffocation

the vessels, Edlefsen attempts to prove that during muscular exertion the arterial pressure in the kidneys falls below the normal, and he founds his view upon J. Ranke's (54) observations on the subject of the interchange of activity among the organs of the body. According to these observations, during tetanus, the muscles contain more blood, the other organs, and especially the glands, less blood than when the muscles are at rest. But such a conclusion is most assuredly not justified by Ranke's observations, for the conditions under which these were made were fundamentally different from those associated with the muscular exertion of healthy subjects, and those which obtain in the majority of convulsive attacks. Ranke caused tetanus of all the muscles by administering poisonous doses of strychnia or irritating the spinal cord; or tetanus of single groups of muscles by electrically irritating the nerve trunk of one extremity. In the first form, causing tetanus by acting on the spinal cord, a violent contraction of all the small arteries takes place—a fact to which we have incidentally alluded (see p. 42), and this is least marked in the muscles, the arteries of which, according to Hafiz (55), are far behind those of the skin and the abdominal organs as regards both the degree and also the duration of the contraction.[1] It was therefore that Ranke noticed that little or no blood escaped from the incised vessels of the skin in tetanus, whereas the muscles were full of blood, and bled freely on incision. This fact alone would suffice to demonstrate the difference between the conditions in Ranke's experiments and those which obtain in ordinary muscular exertion, even of the most severe character. But in the case before us, are the vessels of the skin abnormally empty? On the contrary, there is visible and marked congestion, that is, an increased supply of blood to the skin, and not to the skin alone, but doubtless to other organs, such as the lungs, brain, &c. The cause of this fluxion, at least an important if not the only cause, is the increase of the bodily temperature which is associated with all muscular exertion, and therefore the conditions, so far as we are concerned with

[1] The albuminuria which is sometimes observed in connection with tetanic convulsions probably depends upon this intense constriction of the renal vessels, as explained above (see p. 42).

them, are in all respects similar to those which obtain when the temperature is raised by simple exposure to heat. Can there be any doubt that the kidneys participate in the general increase of temperature caused by muscular exertion, and can we possibly conceive that, notwithstanding the fluxion to these organs and the rise in temperature, the renal vessels should be diminished in calibre and receive a smaller supply of blood? We must reject all previous experience before we can entertain such a suggestion. As far as regards, however, the tetanic irritation of nerve-trunks by means of electricity, we must allow that Heidenhain is correct in the objection he raises, to the conclusions drawn from these experiments, with regard to the activity of glands in muscular exertion, his belief being that the abdominal vessels probably become contracted in a reflex manner, just as occurs when the sensory nerves are violently irritated (56).

The diminution in the quantity of urine and its concentrated condition, which follow muscular exertion, are brought forward by Runeberg (57) as proofs that the pressure is diminished in the renal vessels. We are exempted from any controversy with regard to this view, inasmuch as we have given an obvious and very simple reason why the urine becomes less watery after muscular exertion.

We may in the last place specify the process of digestion, and certain poisons, as factors by which the arterial blood-pressure can be more or less positively raised, or is perhaps regularly increased. With regard to the former of these, certainly I do not know of any experiments in which the pressure has been directly measured, but Zadek in his researches, has indirectly observed that the pressure is increased. Besides this, however, the existence of such increase might, with some probability, be inferred from the fact long since observed by Vierordt and Aberle that the size of the arteries is increased after taking food (58); and also, that during digestion certain changes take place in the circulation which, at least faintly resemble the condition which follows muscular exertion and exposure to heat, and have therefore been designated as "digestion-fever." It is true that this increase of pressure does not appear to be very considerable as a general rule;

according to Zadek it amounts to from one-twelfth to one-sixth of the normal degree. It is therefore doubtful whether this increase can exercise any appreciable influence upon the transudation of albumen. Moreover, the albuminuria often observed during digestion cannot be attributed to increased pressure, for, as we know, the urine is increased in quantity while the process is going on. This albuminuria of digestion can be satisfactorily explained in another way, as will appear later on. (See V.)

No methodical experiments have yet been made with the various poisons which raise the pressure in the aorta, such as, in addition to those previously mentioned (see p. 42) nicotine, picrotoxin, &c. ; only incidentally has albumen been observed in the urine during the employment of one or other of these remedies. For our purpose we can scarcely expect to obtain any definite explanations from such experiments as these, for when poisons of this kind are administed very different conditions co-operate, which make it difficult to connect causes and effects together. With regard in particular to the increase of pressure and its connection with albuminuria, we must take into consideration the fact that the increase depends upon a narrowing of the vessels when most of these substances are administered, and therefore proves just as little as other experiments based on the same process (see p. 42 *et seq.*) ; and likewise that these drugs often cause spasm of various groups of muscles, and exercise a disturbing influence upon the nutrition of the tissues, &c.

A review of the total results of all these numerous experiments, having for their object the production of albuminuria by increasing the pressure in the aorta, produces the impression that the majority of the experiments have failed, and that the labour devoted to them has been, for the most part, expended in vain. The few trustworthy experiments performed with unequivocal results, and facts of a like import, and in harmony with the experiments observed, in the human subject, appear to justify the following conclusion, viz. that the increased arterial pressure in the kidneys *per se* produces, or may produce, albuminuria, if by the removal of water through other channels, the urine is simultaneously decreased in quantity below the normal amount.

In venous congestion of the kidneys the processes are vastly more complicated and difficult to fathom. It is true that the experimental investigations on this subject have shown, with a mutual conformity otherwise rare, that apart from other changes, the urine under these circumstances always becomes albuminous, and with this result the experiences of renal congestion in the human subject seem to accord, so that there has been no hesitation in interpreting in the same sense all the experiments having for their object the arrest of the venous blood; and in regarding all of them, like the clinical examples, as of equal value. If, however, we look more closely into the matter, we find that there are very important differences among the experiments, and to a greater extent between them and the clinical examples, in spite of the fact that a certain similarity exists. With regard to the clinical examples, we do not require much experience to know that, in the first place, with the exception of those extremely rare cases which have scarcely any clinical significance, the phenomena of congestion in the human subject are induced by conditions which lower the pressure in the aorta, and that in the proportion in which this takes place the congestion becomes increased; and in the second place, that the conditions generally become developed very slowly, that is to say, in the course of many weeks, or at least days, and hardly ever in the course of a few hours or minutes. In the experimental investigations performed for the purpose of explaining the conditions in the human subject, there is much that contravenes the above experiences. In order to produce congestion, the preference has been given to experiments in which the renal vein is tied, and these have been used for purposes of comparison, although inasmuch as the arterial supply to the kidneys continues unchanged, the circumstances are the opposite of those of the pathological conditions of the human subject. Moreover, in the majority of cases, and especially in recent times, when these questions have assumed considerable prominence, the effects of the various operations regarded as necessary have neither been gradually induced nor allowed to continue for a brief period, but suddenness and extreme results have been aimed at, and thus, for example, the move-

ment of the blood has been completely arrested during many hours, and indeed, for days. As a matter of fact, the final result of all such experiments in which, however much the conditions are changed, one at last is always attained—the arrest of the circulation and of the renewal of the blood; I say, the final result of all such experiments is, as could be easily foreseen, always the same, namely, the escape of albumen and blood throughout the kidneys, and these pass into the urine when the secretion is again restored. But that under the various conditions which induce congestion, the phenomena should develop themselves in an unequal degree might certainly *à priori* be regarded as more probable than the contrary; it is also certain that these differences could not be recognised if the changes were investigated only after they had become very far advanced, and the mischief had thoroughly and universally affected all the various elements of the kidneys; and in the last place it is no less certain that the course taken by the phenomena may be very different according as the disturbance is a sudden one or of a slow and gradual character. We know that all the organs, and the kidneys are no exception, are very sensitive to disturbances of the circulation, but notwithstanding this fact, when the disturbance is of a gradual character, they accomodate themselves very well to the altered conditions, and within certain limits can discharge their functions in a manner approximatively normal. A consideration of all these facts will prevent us from expecting, on the one hand, any uniform course of the phenomena in the experiments on congestion, and, on the other, any collective and individual coincidences between the clinical appearances of renal congestion and those experimentally induced.

If the escape of venous blood be completely prevented by tying the renal vein, or by any other plan which does not at the same time arrest the action of the heart, inasmuch as blood will be supplied as freely as before, there must necessarily result the fullest imaginable engorgement of the organ with extravasations of blood due to lacerations of the smallest vessels, and the only limit to this engorgement will be the distensibility of the renal capsule. In rabbits, for example, this limit appears to be reached when the escape of blood

has been prevented for from half-an-hour to an hour. The kidney is then enormously swollen—to double its normal size or more—and in addition to extravasations varying in size and number, its entire substance, cortical and medullary, is turgid with blood. After the organs have been hardened by boiling, according to Posner's method, and then coloured, microscopical examination shows that coagulated albumen and numerous red blood-corpuscles are deposited in the capsules and in the uriniferous tubules. The same appearances are found, according to Posner, when the inferior vena cava and not the renal vein is tied, and the same holds good, particularly with regard to the excretion of albumen (to anticipate further statements on this head), in every form of congestion due to other causes, provided only that the condition be of sufficient intensity and duration, stopping short, however, of causing the engorgement and extravasation of blood to attain the same degree. During the existence of such intense congestion, the function of the kidney is almost, if not altogether, in abeyance. In experimenting upon rabbits I could not succeed in obtaining any trace of secretion from the ureter of that side, if, before applying the ligature, the tube had been emptied of its contents. Cohnheim (59) states that immediately after the vein is tied a bloody liquid, highly albuminous, may at first distill from the ureter; this gradually but decidedly diminishes, until at last the secretion entirely ceases. When the ligature is removed, supposing that it has not been retained for too long a time, the secretion is restored, and the fluid, as a matter of course, contains blood and a considerable quantity of albumen. How far this fluid, containing as it does so much albumen and blood, deserves to be called urine, is a question which may be left undecided.

An experiment of this kind can, as I have already said, teach us nothing with regard to the order of sequence of the process of the development of the albuminuria, especially upon what it depends. The production of such an intense congestion injures more or less all the elements concerned, the glomerular vessels, the interstitial vessels, the various epithelia, and possibly other constituents of the tissue. With regard especially to the tubular epithelium, it is true that

no marked changes can be possitively detected under the microscope, but this is clearly no proof that the cells have escaped all injury, for the glomerular vessels appear unchanged, notwithstanding the fact that blood and albumen have escaped from them in appreciable quantities.

The appearances are of a different and more simple character when the flow through the vein is checked for a very short period only, the obstruction being removed before the symptoms have reached their acme. We are indebted to Ludwig, whose studies on the secretion of urine led him to try the effect of these very brief interruptions to the venous outflow, for a knowledge of the very important fact that the closure of the renal vein (the arterial supply continuing unchanged) causes the uriniferous tubules of the pyramids and medullary substance to be compressed by the much dilated veins surrounding them, even to the extent of producing complete closure, so that the flow of urine may be interrupted, to be immediately renewed when the blood is allowed to flow. In such a kidney, removed from the body immediately after ligature of the vein, all that can be recognised, both with and without the microscope, is great dilatation of the numerous vessels of the renal medulla, but no excretion of albumen, none at all events in the Bowman-Müller's capsules. If the vein is allowed to remain occluded for a somewhat longer period, for eight to twelve or at most fifteen minutes, the changes will be found to have advanced a step further—there is now distinct excretion of albumen in the kidney, but this has taken place exclusively, or very nearly so, in the uriniferous tubules, particularly of the medullary substance, in the collecting tubes, but not in the capsules.

The appearances are very beautiful and distinct on sections, doubly coloured with picro-carmine, of the renal tissue hardened by boiling with or without subsequent treatment in alcohol. Everywhere within the tubuli we see the tiny coagula of albumen with shrivelled blood-corpuscles, whereas in the capsules we look in vain for albuminous deposits; perhaps here and there an isolated specimen may be found, if the experiment has been somewhat more prolonged.[1] If the ligature be retained for more than the brief interval

[1] See fig. 1.

mentioned above, the conditions more and more closely approximate to those already described, and the excretion of albumen within the capsules generally is a manifest phenomenon. At the same time, it is important to notice that the epithelial cells of the tubules blocked-up by the masses of albumen are, like the epithelium in general so far as can be recognised, in a perfect state of preservation. When the interruption to the circulation is of such a brief character, no detachment of cells from the basement membrane can be made out, but if the congestion lasts a little longer, the epithelium is seen to be removed from its bed by a layer of coagulated albumen.

No other conclusion can be drawn from these experiments than that the medullary substance of the kidney is that portion which is primarily and most seriously affected when the renal vein is occluded, and the flow of blood through the artery allowed to continue; and that the abnormal excretion of albumen first occurs in the uriniferous tubules of this portion, the visible escape of albumen into the capsules being a subsequent phenomenon. I do not know of any other way of interpreting the appearances before us, for no one can be expected to assume that the albumen is excreted into the capsules, and drains away into the tubules in the course of a few minutes, and to such an extent that nothing beyond a mere trace, if even so much as this, remains behind in the capsules. There is nothing extraordinary in the fact that this conclusion has hitherto not been recognised, or that it should have been repudiated as illegitimate by experimenters, for as I have already said, the experiments hitnerto made have not been properly performed, at least with regard to our present purpose.[1]

This explanation of the origin of the excretion of albumen when the renal vein is completely occluded for a brief period, during which some amount of circulation is probably kept up by other veins, can also be applied to explain the same

[1] I have shown in a former treatise (*l. c.*) that the process of the excretion of albumen, when the flow through the vein is interrupted, goes on as above described, but I then made no distinction between occlusion of the vein, without any change in the arterial pressure, and the same condition accompanied by a reduction of that pressure.

phenomenon occurring when the renal vein is only partially occluded or the inferior vena cava is tied,—in either case the escape of venous blood not being interrupted for too long a period. From some statements of Weissgerber and Perls (60), in whose experiments the flow through the vein was thus checked, but for too long an interval for our purpose, we may gather that the excretion of albumen took place primarily and in the most marked degree in the pyramidal portion and medulla of the kidney. For they found the albuminous coagula, which they described as "hyaline cylinders," chiefly and primarily in the pyramids and looped tubules, but more rarely and only sparsely in the convoluted portions.

A second method of producing congestion consists in cutting off, or reducing, the supply of arterial blood. After a ligature has been applied to the artery for several hours, the macroscopic and microscopic appearances presented by the kidney are, as already mentioned, but little different from those observed when the vein has been similarly treated for an equal period; the kidney, however, is less swollen, and the engorgement of the medulla as contrasted with that of the cortical portion is more conspicuous; there is no appreciable difference with regard to the excretion of albumen. It is no wonder that those who performed experiments of this kind, discovered no sort of difference between the processes of congestion induced in either manner. But the state of the case is different, if the interruption, this time of the arterial supply, is removed after a short interval, say eight to ten or twelve minutes. Then we see a considerable excretion of albumen, but in the capsules alone, and only after a longer interval in the urinary tubules as well, and then only in a very inferior degree, altogether different therefore from what takes place when a ligature is kept on the vein for a short time. A similar result will in all probability be induced, if the artery is not quite closed, but its calibre very much narrowed for a somewhat longer period, as has been done by M. Herrmann and von Overbeck, (61) and resulting in the production, according to their observations, of albuminous urine, while the afflux of blood continued, though in a reduced quantity, the urine itself undergoing a very considerable diminution before the albumen appeared. I

have not made any experiments with regard to the first appearance of albumen and its distribution under these circumstances.

With regard to the condition of the urine, the fact is not without significance that, contrary to what happens after the vein is completely occluded, viz., the almost invariable appearance of blood in the secretion, this, in the case before us, is only an exceptional phenomenon. Stokvis discovered albumen, but no trace of blood, in the first specimens of urine passed after the circulation in the renal artery had been completely interrupted for a prolonged period. The albumen under these circumstances cannot have been yielded by the remaining healthy kidney, for, as Frerichs, Rosenstein and others have shewn (62) the complete removal of one kidney is not followed by albuminuria. We must, in preference, assume that, when the principal artery of the kidney is tied, the circulation is partially maintained by means of certain small arteries, and that a secretion of urine, certainly abnormal in its characters, goes on. To this category must be referred those instances in which, in consequence of a morbid constriction of the small arteries, the supply of blood is checked and albuminuria results—conditions which have been observed in the experiments previously alluded to (see p. 42) in which the irritation of the spinal cord, the poisonous effects of strychnia, dyspnœa, &c., were produced—and likewise the albuminuria observed by Cl. Bernard (63), when he irritated the peripheral portions of the divided renal nerves. We may fairly assume that, in all these instances, there was an abnormal escape of albumen into the capsule while the spasm of the vessels continued, and that it was washed onwards by the subsequently restored current of blood. Blood may appear in the urine passed under these latter circumstances, just as it may in that which is secreted when the circulation is restored in the artery after complete interruption, for at this period the blood passes under abnormally high pressure through vessels more or less damaged as regards their nutrition, in consequence of the previously defective supply of blood,—conditions under which hæmorrhages are prone to occur.

In connection with the above details we must, in conclu-

sion, allude to those experiments of tying the ureter, which have likewise been adduced in order to explain the alterations of pressure in the kidney. They may be legitimately used for this purpose, provided that the ligature be not retained for too long a period, for in that case the results will tend to produce further obscurity instead of contributing towards an explanation. If the ligature be retained for a very long period, several hours or more, Posner states that the same changes will be produced as are observed after a prolonged occlusion of the vein or artery, viz., marked accumulation of blood, with rupture of vessels here and there, and later on (according to Posner, after the ligature has been retained for three days) albumen in the capsules and tubuli. There is therefore either no difference at all, or only a very slight one, between this form of albuminuria and others produced in other ways, so that Posner in the case before us can discover nothing more than the consequences of acute congestion. The circumstances however, are not exactly similar. In the first place after ligature of the ureter, the blood continues to circulate for at least a considerable time, perhaps not in an altogether normal manner, but without indications of much disturbance, a fact which can be demonstrated by opening the vein. It has been supposed that the retention of the secretion in the urinary tubules would check the flow through the vein, just as obstruction of the latter checks the flow of urine, but this, as Runeberg justly remarks, is a purely arbitrary assumption (64). Certainly no comparison can be drawn between the enormous increase of pressure, the result of occlusion of the vein, and the pressure caused by the confined urine, and the uriniferous tubules are very much less distensible than the veins. Granted therefore that the confined urine may possibly directly obstruct the escape of blood, the obstacle is certainly not a considerable one and its action is very far indeed behind that of the congestion due to occlusion of the vein.

If the ureter be kept closed only for a shorter period, ten to fifteen minutes, the principal change, and, indeed, the only one when the period is very brief, is marked distension of the uriniferous tubules, to such an extent that even the convoluted portions present a wide clear lumen, filled with fluid,

and secondly, an enormous dilatation of the lymph-spaces (œdema) especially in the medullary portion. The intervals, generally hardly recognisable, between the urinary tubules and in the neighbourhood of the vessels, appear as wide clefts; the adventitia of the small arteries is enlarged to thrice or more times its normal breadth, and its nuclei in consequence are separated from each other by considerable intervals. On sections, prepared as already described, the appearances are very remarkable indeed, and we also noticed that the interior of these lymph-spaces is often filled up with a very finely-granular mass (coagulated albumen).[1] Besides this there is congestion, though not of a very marked character, of the medullary veins. When the ligature is retained for a somewhat longer period, we find a decided excretion of albumen in the capsules as in the uriniferous tubules. Whether this occurs first in the former or in the latter structures I have not been able to decide, but on the whole the albuminous excretion appears to me to preponderate in the capsules, even when the flow of urine is obstructed for the briefest intervals, so that its appearance is probably earlier and its quantity greater in these portions of the kidney than in the uriniferous tubules.[2]

The urine secreted after removal of the ligature contains more or less albumen, according to the length of the interval during which the flow of urine has been obstructed. It also contains blood, but always in very small quantity, even when the congestion has been maintained for several days (65). In my experiments, when the ligature had been retained for brief intervals, there was scarcely any trace of blood-corpuscles visible under the microscope.

These results of all these experiments may therefore be summed up by saying that the various forms of congestion correspond with each other in so far that they all produce

[1] See fig. 2.

[2] Posner states that only after three days was he able to discover marked albuminous engorgement of the capsules and tubules. Perhaps the cause of this later appearance, as compared with my experiments, is to be found in the fact that he tied the ureter close to the bladder, whereas I tied it near the kidney. The ureter is very distensible, and, therefore, more time will be required for it to become distended throughout to such a degree as to cause the obstruction to be felt in the kidney.

albuminuria, but that differences exist not only as regards the degree, but also as regards the place in which the excretion of albumen occurs. Only after a distinct interval, varying according to the method of experimenting, do these differences become effaced, especially so far as the deposition of albumen is concerned, uniform or nearly uniform conditions are then developed in the kidney, all the tissues becoming gradually involved. The albumen escapes most rapidly and abundantly when the vein is occluded, and the escape of blood in considerable quantity is also a regular phenomenon; cutting off the arterial supply would seem to come next as regards these effects, while confining the urine by tying the ureter occupies the last place.

What are the causes of the differences in the development of the albuminuria and other changes in the kidney, in these various forms of congestion? In estimating the processes, we may be guided by the changes of pressure in the blood-vessels and lymphatics, and the disturbances of nutrition which are invariably associated with certain degrees of these changes, since in all cases of congestion, however produced, there is a retardation of the current of blood, with disturbance of nutrition as a constant accompaniment as time goes on. These two factors, the retardation of the current and the disturbance of nutrition, must therefore not be separated from each other. It must also be remembered that in the production of urine two factors co-operate, viz. the glomerular vessels which are governed by the laws of filtration, so long as their nutrition is unaffected, and the glandular epithelium proper, which is subject to influences of a different character, and only partially understood.

The process takes the simplest form when the arterial supply is interrupted, that is, when the congestion is the result of ischæmia. The natural consequence of this interruption is that the blood-pressure in both capillary systems of the kidney falls below the normal. How the filtration in the capsules will be thereby affected can be stated with certainty; there will be a diminution in the quantity of the filtrate, but an increase relatively in the amount of albumen it contains (compare p. 28); and this will be decidedly the first direct effect of the diminution of the pressure, and one

which must occur immediately, before any possible disturbance of nutrition can take place, provided that any pressure exists and any filtration goes on. The consequences as affecting the glandular epithelium of the kidney can be estimated only according to the analogy of other glands in which secretion and not merely filtration takes place. It is, however, certain that every secretion becomes more scanty as the blood-pressure diminishes. Whether, in cases of simple diminution of blood-pressure, albumen, previously absent, passes into the secretion, has, so far as I know, not as yet been experimentally determined with regard to the liver, the only gland with a non-albuminous secretion suitable for an experiment of this kind. I should, however, think it not improbable, considering the results obtained by occluding an artery for brief intervals and which have been described in a previous page (see p. 59). The result would be different if the congestion were of long duration. Examination of the kidney in this case furnishes no positive explanation, for deposits of albumen are found both in the capsules and in the uriniferous tubules, but this appearance is no irrefragable proof that the albumen is derived from the interstitial capillaries, for it may have escaped from the capsules into the tubules. On the other hand, from the condition of the biliary secretion in the human subject, after prolonged congestion, it may be allowable to draw conclusions as to the secretion of the renal epithelium in similar circumstances. In connection with this, much importance is attached to Frerich's statement (66) that albumen has been found in the bile in several cases of passive hyperæmia of the liver. It proves that even in this form of congestion, besides the glomerular system of vessels, the secretory apparatus of the kidney may also yield albumen.

But however this may be, the combined results at any rate of the diminution of pressure upon the two sources which participate in the production of urine, must be a considerable diminution in the quantity of that fluid and an increase in the percentage of the albumen it contains. The percentage of albumen in the fluid yielded by the capsule will now therefore be $a+x$ instead of a; the quantity of the epithelial secretion will be represented by $n-y$ and therefore the percentage in the

urine when the arterial supply is diminished will be $\frac{a+x}{1+\frac{n-y}{100}}$ A.

comparison with the formulæ previously given (see pp. 33 and 42), for the normal and for the increase of arterial pressure, makes the difference sufficiently obvious. If in this case albumen really appears in the true glandular secretion, we might naturally expect an increased quantity in the urine.

When the ureter is closed, the action which the confined contents of the uriniferous tubules exercise upon the glomerular vessels and the secreting epithelium is the factor which determines the result. Runeberg has set forth in a proper light the consequences as they affect the former portions; he states that the stagnation of the urine causes an increase of the pressure upon the external wall of the glomerular vessels, that the filtration-pressure—that is, the difference between the external and internal pressure—is decreased, always supposing that the current of blood remains unchanged, or at least that there is no increase within the glomerular vessels. That this supposition is perfectly right may be positively assumed only with reference to the first beginnings of the process. As we have already explained (see p. 63), when the ligature is retained on the ureter for a short time only, the circulation is certainly not completely interrupted, and the obstruction which the distended uriniferous tubules cause to the blood-vessels can at first be only very slight, so long as there is no great accumulation of fluid confined within the excretory tubes of the renal medulla.[1] A diminution of the filtration-pressure may therefore be admitted to exist in the earliest stage, and the evident deposition of albumen in the Bowman-Müller's capsules is capable of explanation in accordance with the descriptions already given. It is difficult, however, to make any further statement with regard to the conditions and circumstances of the pressure as the process goes on. It is possible that the blood-pressure within the glomerular vessels may be increased to an extent exceeding that of the external

[1] Even when there is prolonged and complete occlusion of the ureter, circulation of blood to a sufficient extent may continue, as clinical observation shows. A case of this kind, interesting also in other respects, has been recently reported by Schwengers and Leichtenstern (67).

pressure to which they are subjected ; in that case the filtration would become almost normal ; or if the preponderance of pressure within the vessels increased, the resulting filtrate would contain more water and would be poorer in albumen. There is also the possibility that, the secretion by the glandular epithelium being continuous and actively carried on, the predominance of pressure might become so decided in the uriniferous tubules that the fluid of the capsules might take a backward course, and that absorption might go on until the internal and external pressures became equalised.

There is no doubt that the secretory epithelium of the uriniferous tubules continues to possess and exhibit its secretory activity after the ureter is occluded. That this is so is positively shown by the increasing fullness of the uriniferous tubules, the great dilatation of the lymphatic channels depending upon the pouring into them of secretion not removed in the ordinary way, and by the behaviour of other glands under similar circumstances. It is, however, another question whether the secretion is altogether normal in character, and in particular whether it is free from albumen as is normally the case. The liver is the only organ which could be used to assist in deciding this question, but, so far as I know, no investigations have been made upon its secretion after tying its excretory ducts. But the albuminous deposit which I found in the uriniferous tubules after a ligature had been kept on the ureter for a brief interval (see p. 63), it being impossible that the albumen can have got there by escaping from the capsules, is evidence in favour of the supposition that the real secretion of the epithelium contains albumen, as might be expected with regard to every œdematous gland. It is, therefore, highly probable that both the glomerular vessels and the interstitial vascular and lymphatic systems participate in the production of the albuminuria that ensues after the impediment is removed.

When the escape of venous blood is prevented without interfering with the arterial supply, the circumstances, first and foremost, with regard to the blood-pressure, are very different from those which obtain in the other two kinds of congestion, and especially in that caused by tying the artery. When the supply is cut off, the pressure in the capillaries

falls, as we have said, below the normal; if, when the occlusion is complete, compensation takes place by a retrograde movement of the blood from the spot where the renal vein opens into the inferior vena cava, the pressure in the radicles of the renal vein will become equal to that in the vena cava, and therefore as a matter of course always lower than the normal degree. The case is, of course, different when the vein is occluded, for then the pressure must reach an abnormal height, the degree varying with the degree of completeness of the closure. In consequence, however, of the peculiarity involved in the fact that the majority of the branches of the renal artery break up into the glomerular capillaries before passing into the interstitial capillary system, the increase of pressure in the various portions of the vascular system must differ in amount (68). It will be most marked and its occurrence earliest in the interstitial capillary system; it will appear later and in a less degree in the glomerular vessels. The production of œdema, here as everywhere else, necessarily follows as the second direct effect of occlusion of the vein with unimpeded arterial supply. And thus it is that this form differs from the ischæmic congestion, which *per se* does not immediately produce œdema, but which, only after a much longer period, and when in consequence of the deficient supply of blood the nutrition of the tissues has become affected, may be the indirect cause of a dropsical swelling. Thirdly, and lastly, as I have already more than once mentioned, the excretory portions of the uriniferous tubules, especially in the medullary substance, are compressed by the congested veins and stagnation of urine is the result.

These three sets of consequences blend with each other and their mutual influence is to some extent contrary, so that it is difficult to separate their effects, and to determine separately the share which each has in the disturbances they jointly produce. The stagnation of urine in particular counteracts to a certain extent the increase of pressure, as already explained. If we therefore leave this complication out of the question, we shall have less difficulty in estimating the effects of tying the vein upon the two factors concerned in the production of urine, always of course supposing that

the discharge through the vein is not entirely checked, for if it be, the function of the kidney is rapidly extinguished (see p. 60). Cohnheim's researches upon venous congestion (under conditions, as in the case before us, of unimpeded arterial supply), justify the conclusion that in the parts in which transudation occurs, the glomerular vascular system, there will be an escape of fluid, in quantity exceeding the normal and containing blood. Both of these, the quantity of fluid and the blood, are in direct proportion to the degree in which the pressure of congestion exceeds the normal amount; and the blood especially is the characteristic which distinguishes the transudation of congestion from that which is normal, and likewise from that yielded by the kidney when only the arterial pressure is increased; and whether the blood-corpuscles escape by diapedesis, or in consequence of rupture of vessels, is for our present purpose a question of no importance.

The condition of the secretory apparatus may also be determined with some degree of certainty, although in this case a comparison with the liver is again wanting. For, so far as I am aware, no observations have been made with regard to any changes which may occur in the bile after ligature of the hepatic vein. But we may regard it as certain that an œdematous gland—and with such we are certainly dealing at present—not only secretes more copiously, but also allows albumen (and probably also blood) to pass abnormally into its secretion, as a matter of course from the interstitial vessels. This is also proved by the discovery of albumen between the epithelium and the membrana propria, an appearance which, in this form of congestion, is speedily produced at a much earlier period than in the other forms in which it may occur after a longer interval. If, however, this hypothesis be rejected, and the albumen in congestion be supposed to escape from a source other than the glomerular vessels, it would then be necessary to interpret these appearances by supposing that the epithelium first became detached, and then that albumen drained from the capsules between the epithelium and the membrana propria. It is, however, easy to foresee the relation that would then exist between the basement membrane which had lost its epithelium and the

lymph-current in which it is bathed. This thoroughly forced explanation is therefore discarded.

An abnormally copious secretion yielded by the interstitial vascular apparatus, containing albumen, and possibly blood as well, will therefore be added to the already abnormally copious and blood-containing transudation of the glomerular vessels, and the collective results of the venous occlusion will consequently be the production of urine abnormally copious in quantity and containing albumen and blood. This effect will, however, in part, be weakened by the confinement of the urine within the tubules, which is associated with the occlusion of the vein, and which counteracts the pressure in the glomerular vessels. We have already seen that in these vessels the increase of pressure *per se* is less than in the interstitial capillaries, and is also later in appearing, and it follows that the effect of occlusion of the vein upon the glomerular vascular system will be much less and occur later than the same effect upon the interstitial vascular system and the uriniferous tubules.

So far as any knowledge can be obtained from experiment and clinical observation, they confirm in the most striking manner the correctness of these explanations. It is certainly true that experiments in which the occlusion of the vein is complete and continued for several hours furnish no explanation at all, or none of any use for our purpose. Under such circumstances the function of the kidney scarcely exists, and extravasations of blood and albumen are the general appearances on anatomical investigation. On the other hand, our experiments, in which the vein has been tied only for brief intervals, have really shown that the first effect is produced in the sphere of the interstitial capillaries and the uriniferous tubules (see p. 60).

As regards the condition of the urine, it is not easy to come to any decision in experiments of such brief duration, for the secretion soon becomes abolished when the closure is complete. In the experiments already referred to of Weissgerber and Perls, in which the vein was completely occluded, but for a longer interval, the special feature was the appearance of albumen in the collecting tubes and loops, and only in a portion of the cases in the convoluted tubes as well.

In five cases it is twice mentioned that the urine contained blood, so that these may be regarded as confirmatory of our explanations. These latter are also supported in a very marked manner by Bartels' case, already referred to (see p. 26), and in many respects so very interesting, of thrombosis of the inferior vena cava occurring in a robust man, a case presenting precisely the same conditions with those in Weissgerber's and Perls' experiments, viz. obstructed venous outflow with unimpeded arterial supply, and this, moreover, is the only known case of the kind in which any reference is made to the quantity of the urinary secretion. "In this case the result has been," to quote Bartels' own words, "not only a profuse secretion of urine, but also the escape of albumen, and even of considerable quantities of blood into the urine, probably in consequence of rupture of several vascular tufts of the glomeruli." If an obstacle to the venous outflow, limited to one renal vein or to the inferior vena cava, is formed in the human subject, the conditions under which this generally occurs do not permit of any comparison with venous occlusion produced by experiment, since the arterial pressure is simultaneously diminished. This is what occurs, for example, in the most common clinical thrombosis of the renal vein of new-born children, for this is always of marantic origin. But even in the very rare cases as well, in which the vein is occluded by tumours, generally of a malignant character, other complications being likewise present, the arterial pressure is usually in a similar condition. Corresponding with this, there is in these cases a difference as regards the quantity of the renal secretion. According to the rapidity of the development and growth of the obstacle the urine invariably becomes more or less scanty, and at the same time contains much albumen and blood.

The admixture of blood distinguishes this urine from that which usually and frequently occurs in congestion in the human subject, this condition of the kidney being the result of diminished cardiac activity. The conditions are different from those of artificial venous occlusion, and so likewise are the consequences as regards the renal secretion. The statement so frequently made to the effect that the ordinary urine of congestion in the human subject

contains blood and abundance of albumen, is not at all in accordance with facts, and there is, by no means, the most perfect harmony between clinical observations and the experimental venous occlusion, as is generally pretended. For these reasons it will not be superfluous to describe somewhat minutely the urine of congestion, as it daily comes before us in disease of the circulatory or respiratory organs, and such a description will be useful for another reason, viz. because certain changes in the urine, especially with reference to the proportionate amount of its several constituents, and which may be of importance in the explanation of the processes going on in the kidney, can be determined in a satisfactory manner only in the human subject. The first and most constant phenomenon is the diminution of the water, and therefore of the quantity of urine, this being determined by the amount of water present. There is little known with regard to the proportionate amounts of the solid constituents, but the total quantity of these is not diminished in the same proportion as the water. On this account the specific gravity of the urine is increased, its colour becomes deeper, and there is a tendency to the formation of sediments, these being due to the fact that the urates, which are not very soluble in cold water, and uric acid itself, are readily precipitated during cooling from urine which contains less than the normal amount of water. It is, however, more than probable that the solid constituents, or at all events a portion of them, are excreted in diminished quantities, although their diminution does not proceed *pari passu* with that of the water, and they may therefore be relatively increased with reference to that fluid. The urea at least, according to my observations, is always absolutely diminished in quantity, notwithstanding the relative increase, which may amount to three or more per cent.[1] The condition of the uric acid is more difficult to estimate, inasmuch as the respiratory troubles almost always present in this form of renal congestion, give rise to complications which apparently exert

[1] The absolute diminution of the urea can partly be explained by the fact that less nourishment is taken and absorbed, and partly also by the consideration that the urea finds its way into any dropsical effusions that may be present, and into the discharges from the bowels.

some influence on the formation of uric acid in the system (69). At the same time that the water becomes diminished, or shortly afterwards, albumen is often to be found in the urine, but always in small quantity, provided that the condition is that of congestion and nothing more. It would seem that this statement cannot be too often repeated. It is true that when the congestion is of long duration and of great intensity, the quantity of albumen is much increased at a later period, but the consecutive phenomena of the stagnation of blood and lymph (inflammatory processes and conditions of induration, nephritis from passive congestion), have by that time invariably made their appearance. Blood is—and this is a fact which must be repeatedly insisted upon—scarcely ever present in macroscopic quantities, and even with the microscope it is only in a minority of cases that we are able to detect isolated red blood-corpuscles, and generally a few colourless cells. It appears to be doubtful whether, in such cases, this slight admixture of blood has its origin always in the renal parenchyma, rather than from the mucous membrane of the bladder, ureter, or pelvis of the kidney; for, as a general rule, the mucous membrane of these parts is the seat of more or less marked hyperæmia which, as in other mucous membranes, may give rise to more or less considerable hæmorrhage. Pale (hyaline) casts are often found associated with the albumen.

We have thus given the details of the ordinary kind of renal congestion with which every physician is familiar. Another circumstance, equally well known, may be added, viz. that so soon as the heart recovers its power, the urine very rapidly returns to its normal condition. I need only refer to the action of digitalis in cases of mitral deficiency. In its origin and the way in which it becomes developed, this form of renal congestion obviously most resembles that which is induced when the arterial supply is cut off for a brief interval or in an incomplete manner, for in both cases diminution of the arterial pressure within the kidney is the first thing that happens. In the experiment certainly, in order to produce the most striking phenomenon in the brief period of observation, the arterial pressure is very considerably reduced, or perhaps altogether abolished, a condition not belonging to the pathological examples. The

case before us, however, differs from the experiment in which the artery is tied, in the fact that an obstacle is introduced in the direction of the current of venous blood, the pulmonary vessels and the right side of the heart being engorged with blood. The effect, therefore, with regard to the difference of tension between arteries and veins, which is the main point in the question, may be exactly similar: in the experiment, in consequence of the marked diminution of the arterial pressure, the flow through the vein being unimpeded: in the pathological conditions, in consequence of a smaller diminution of the arterial pressure, but with a co-existing impediment to the escape of blood through the vein. It comes to pass, therefore, that there is an anatomical resemblance in the main feature between the kidney in the ordinary state of congestion and in that due to cutting off the arterial supply, any difference being due only to the fact that in the human subject death does not result as soon as the congestion begins, and that this latter condition is of longer duration, time being thus afforded for the development of those consequences of stagnation which have been already mentioned. There is seldom an opportunity, except in cases of sudden suffocation, to examine kidneys in a marked state of venous congestion of recent origin, and in which, when death took place, the congestion had rapidly advanced so far as to produce albuminuria. In the few cases which have come under my notice, and which to some extent coincided with the requirements, a very moderate increase in the size of the kidney was the only thing to be observed; but this did not even approximately resemble that enormous increase which took place in a rabbit's kidney after tying the vein, and never even once reached the degree attained in cases of recent inflammatory swelling of the kidney. The congestion presented the usual appearances, being most marked in the pyramidal portion in which veins are numerous, but less prominent in the Bowman-Müller's capsules. Under the microscope a deposit of albumen was not always to be found, at least not in every section; where, however, it was present, it was contained almost invariably in the capsules alone.

The results, therefore, of clinical observation coincide, in a manner practically complete, with what follows when the

arterial supply is artificially cut off, and are most satisfactorily explained by those processes which are induced by this experiment, as above described (see p. 63). These are, the great diminution of the water as the aggregate result of decrease in pressure upon the filtering and secreting apparatus, the diminution of the specific constituents of urine as represented by urea, the small proportion of albumen present, the total absence of blood, or its presence in very minute quantities. Considering the experimental and clinical facts, it is not necessary to discuss the influence of an alteration in the nutrition of the epithelium and vascular walls upon the commencement of the process, even when the arterial supply is completely cut off, and it is still less necessary to do so when the supply is reduced but not entirely abolished. M. Herrmann as well as v. Overbeck noticed the appearance of albumen in the urine a few seconds after they had tied the renal artery, and they also observed that it disappeared after a very short interval, even within half-an-hour. It has been already mentioned that the circumstances are often very similar in the human subject, and that the urine changes, so to speak, at once when the circulation becomes regulated, being more abundant in quantity, and exhibiting no trace of albumen. It would be necessary to magnify considerably our notion of disturbed nutrition and to regard every disorder of the circulation as coming within this category, if in cases like these we attempted to attribute the rapidly appearing and transient albuminuria to deranged nutrition of the elements of the tissues. It is, moreover, difficult to reconcile the scanty excretion of albumen and the very slight admixture, if any, of blood in the urine, with the assumption that there is any disorder of nutrition in the glomerular vessels or their epithelial investment.

It would, however, be improper to deny that, as time goes on, disturbances of nutrition become a prominent feature in the congestion we are now discussing. It is certainly difficult or even impossible to specify the degree to which the arterial supply must be reduced in order to cause nutritional disturbances, and therefore in forming an estimate of this influence considerable latitude must be allowed to individual discretion. According to the stand-point which an

observer takes up with regard to the effect of mere changes of pressure, will he be inclined to claim the right of laying stress upon the influence of derangement of nutrition, at an early or late period, for the purpose of explaining the disturbance of function. It is only in estimates of extreme conditions that certainty can be attained, and we shall not go wrong in laying down the two following propositions: First, that no considerable disturbance of the nutrition of the tissues can take place if the supply of blood be interrupted for a very brief period only, say for a few seconds, and during no portion whatever of the time in a complete manner; or if a retardation of the circulation be moderate in extent and slow and gradual in its development, so that, as already mentioned (page 58), the tissues accommodate themselves to the diminished blood-supply. Secondly, that if the circulation be completely interrupted for several hours or days, severe disturbance of the nutrition of the tissue must necessarily ensue.

The third form of renal congestion, viz. that which is due to impeded escape of urine, occurs in the human subject, though less frequently than the other forms. A comparison, however, cannot be so freely instituted between the symptoms at the bedside in these cases and those produced by experiment, because for the most part only one kidney is affected, so that the urine, secreted by the healthy kidney alone, either presents nothing abnormal, or, if, the discharge from the affected kidney be not completely suspended, the result is a mixture of normal with abnormal urine. Another fact which affects the comparison is that the obstacles to escape of urine are usually of such a character that abnormal admixtures, *e.g.* albumen or blood, become superadded to the fluid after it has left the kidney. Instances of this kind are, among others, the obstacles due to concretions which irritate the mucous membrane, tumours in the urinary passages, &c. We have also no knowledge of the anatomical appearances of the human kidney from the very first stages of the process involving complete stagnation of urine, which can be compared with the result of tying the ureter; we likewise know nothing as to the part in which albumen is deposited in these kidneys.

IV.—Albuminuria as Dependent Upon Degeneration of the Renal Epithelium.

Of the tissue-elements, other than those of the vascular system, which compose the renal parenchyma, the epithelium alone remains for consideration in our study of albuminuria. The most important are the epithelial cells of the uriniferous tubules, next come those of vascular tufts, and in the third place, though these are not so directly concerned, the epithelial cells which line the internal surface of the Bowman-Müller's capsules. In former times the epithelial cells were credited with the possession of a considerable amount of influence in the causation of albuminuria, but this was attributed only to those of the first class, the existence of the others not being recognised. Two principal ideas prevailed on this subject. It was supposed that in morbid states of the epithelium, the albumen which normally transuded through the glomerular vessels, to be taken up and assimilated by the epithelium, no longer underwent this process, but escaped by the urinary tubules; or, secondly, that the albumen passed into these channels from the blood-vessels by which they are surrounded, because the epithelial cells having undergone nutritive derangement, were unable to discharge their normal function of preventing such escape from taking place. Modern theories pay little attention to this question, but they usually cut the matter short by asserting that in fatty or other degeneration of the epithelium, as in phosphorus-poisoning, in severe anæmia, and severe febrile infectious diseases, no albumen is found in the urine, and therefore the epithelium can have no share in the production of albuminuria. And yet these very theories are based upon the doctrine that the epithelium prevents the escape of albumen from the blood, and that normal urine is therefore free from this constituent. The contradiction stares us in the face! At all events those who assume that the albumen is retained in the blood by the agency of the epithelial investment of the glomerular vessels, must allow the possibility of albuminuria being caused by derangement of

the nutrition or destruction of this epithelium in particular. The other epithelial cells, especially those of the uriniferous tubules, of the proper glandular elements, are either not mentioned at all, or else they are expressly alluded to as altogether unconcerned in the production of albuminuria.

It is well known that some of the secretions proper, that is, the sole products of really secreting glandular epithelium, are albuminous, while others are free from that constituent. The products of the former, which are the more numerous class, owe their albumen to the constant metamorphosis and separation of the cells which mix with the secretion; the other glands yield a product which is non-albuminous, because according to the general and almost sacred theory, their epithelium does not decay under normal conditions, and their products consist only of definite matters which they take up from the vessels or prepare within themselves, but not of albumen. Whether these epithelial cells really continue without any change during the whole period of life, or whether they undergo an imperceptible metamorphosis, similar to that which is constantly taking place in the organism as a whole, are questions with which we are not concerned at present. It is enough for our purpose that the normal function of its epithelial cells is assigned as the reason for the absence of albumen in the secretion of the liver, the most prominent of the second class of glands, and likewise in the secretions of the perspiratory and lachrymal glands, supposing it to be the case that these latter secretions in their pure state are really non-albuminous. If the urine be regarded simply as a true non-albuminous glandular secretion, the epithelium must be credited with the function of preventing the escape of albumen from the blood; but if my view be adopted, and the urine be regarded as a mixture of a transudation with a glandular secretion (see p. 33), the latter at least being the produce of the epithelium of the uriniferous tubules must for the same reason be considered to be non-albuminous, whatever view may be entertained as to the presence or absence of albumen in the transudation from the glomerular vessels. The conclusion is forced upon us that, when their nutrition and functions are disturbed, or when the epithelial cells of the uriniferous tubules are in a

state of complete decay, albumen will escape from the blood and lymph and show itself in the urine hitherto apparently non-albuminous, that is, that albuminuria will become developed. Observations which are alleged to prove the contrary must be based upon error or defective investigation, for if not, all our doctrines with regard to specific glandular secretion must be thrown to the winds.

Fortunately, there is no such discord between observation and theory in the case before us. It is well known that fatty degeneration of the epithelium, of the most intense character, is induced in acute poisoning by phosphorus and with arsenic acid, and a few other toxic agents, and phosphorus-poisoning, the type of this class, is adduced as evidence against the theory of the participation of the epithelium in albuminuria, because little or no albumen appeared in the urine in cases of this kind. So far as I see, this entirely erroneous view owes its existence to the statements of Stokvis and Kohts (70), who, as a result of their experiments with this poison, felt bound to adopt the conclusion that no connection existed between albuminuria and fatty degeneration, whereas up to that time the observations of Ph. Munk and Leyden (71) had been taken as evidence that albuminuria is an almost constant symptom of poisoning by phosphorus. But in reality the experiments of Stokvis and Kohts do not contradict the hypothesis of a connection between albuminuria and phosphorus-poisoning. As far as Stokvis's experiments are concerned, in the first place he employed as tests nitric acid or acetic acid and boiling, all of which were at that time considered to be sufficient for demonstrating the presence of albumen, though now we know that such is by no means the case (see p. 13 and 14). In the second place, even with these not very delicate tests, he discovered the presence of albuminous substances, viz. serum-albumen and hemi-albumose (propeptone), in three out of his four experiments, and in the fourth experiment the attempt at poisoning appears to have been altogether unsuccessful.[1] With regard to the hemi-albumose,

[1] In the notes of his experiments, published by Stokvis, we find as follows: —Exp. 1. A rabbit poisoned by phosphorus. Death on the second day. The urine during this time without any trace of albumen. The bladder

these experiments permit us to infer its presence, or at least they do not prove that it was absent, inasmuch as the addition of acids caused a precipitate or cloudiness which disappeared on heating, and which Stokvis, without further testing, referred to the presence of fat or of fatty acids. But since we now know that peptone-like substances frequently occur in the urine in cases of phosphorus-poisoning, there is quite as much probability in the former assumption, viz. that they were present in these experiments, as that the reactions manifested were due to the presence of fat.

Kohts has performed eight experiments with poisonous doses of phosphorus—six on dogs, two on rabbits. With the exception of the case of one dog, which died suddenly on the second day, and one rabbit, the reports of all the other experiments state that, after the poisoning took place, albumen invariably appeared once or several times in small quantity, and that in one rabbit the quantity was considerable. No mention is made of the methods used for its detection, but we may suppose that the ordinary means were employed, viz. boiling and nitric acid. It follows that the sentence with which Kohts concludes his report, that "no albuminuria appeared in any of the animals experimented on, with the exception of one rabbit," directly contradicts his own observations.

Investigations on this subject have been very carefully

contains clear, acid urine, which is rendered slightly turbid on the addition of nitric acid, excess of which causes the turbidity to disappear. The same degree of cloudiness is obtained when the urine is heated with a little acetic acid. This can be only serum-albumen, which Stokvis's own experiments show to be soluble in excess of nitric acid, in cold urine. Exp. 2. A dog poisoned with phosphorus. Death on the fifth day. The urine on the last day containing biliary colouring matter, and rendered turbid by nitric and acetic acids, but not by heating; urine found in the bladder exhibiting the same reactions. Exp. 3. A dog poisoned with arsenious acid. Death on the fourth day. Urine of the last two days turbid on addition of nitric and acetic acids; the turbidity disappears on heating, and does not return on the addition of sulphate of soda; urine in the bladder gives similar reactions. Exp. 4. Dog poisoned with arsenious acid; killed on the eleventh day, though then appearing pretty well (!) The report on the urine simply states that there was no trace of albumen, but only a few epithelial cells in a state of fatty degeneration. Nothing found *post mortem*, but very slight (!) fatty degeneration of the organs.

made by Schultzen and Riess (72). In six dogs poisoned with phosphorus, they found more or less albumen in the urine in every case.

The statements, however, of Stokvis and Kohts, cast some doubt upon the subject, especially with regard to the appearance of albuminuria in rabbits, and I therefore performed four experiments with phosphorus upon as many animals of this kind, all previously healthy and vigorous. A solution of phosphorus in oil was injected under the skin. Albuminuria was the result in all four cases, or where there were previous evidences of albumen (see page 20, *note*) its quantity was found to be increased. I append brief details of these experiments.

I. March 20, 1880.—A large vigorous rabbit, fed on oats. Urine passed on compressing the bladder, acid, clear on filtration. The addition of acetic acid and of ferrocyanide of potassium causes a slight cloudiness; the same appearance results on adding acetic acid and concentrated solution of sulphate of soda to the cold urine, the cloudiness remains on boiling. Nitric acid produces a slight cloudiness. On subsequent boiling, the colour becomes so deep that it cannot be ascertained whether the cloudiness has disappeared or not. Three-quarters of a cubic centimeter of a solution of phosphorus in olive oil (1 to 80) are injected beneath the skin.

March 21.—The rabbit is pretty lively. Its urine is acid, clear on filtration, and gives a decided precipitate with acetic acid and ferrocyanide of potassium; with acetic acid and sulphate of soda, marked cloudiness, increased by heat; with nitric acid, marked cloudiness, and on subsequent boiling the urine becomes very dark and a precipitate gradually falls. Another injection as before.

March 23.—The rabbit died early this morning. The urine previously voided gives same reactions as yesterday. Under the microscope, sediment is found to contain very finely granular casts, and a few renal epithelial cells in a fair state of preservation.

II. March 23, 1880.—A large rabbit, fed on oats. Urine acid, clear on filtration; yields a doubtful trace of albumen with the three tests above-mentioned. Injection of half a cubic centimeter of the phosphorised oil.

March 24.—The rabbit's bladder contains a large quantity

of urine, when as recently passed has an acid reaction and is found to be pretty strongly albuminous with all the three tests. Only a few finely granular casts in the sediment. A portion boiled with acetic acid and sulphate of soda, and a precipitate formed. The filtrate gives no peptone- (or biuret-) reaction. Rather more than half a cubic centimeter of the phosphorised oil injected.

March 25.—Urine copious, paler than yesterday, acid, contains more albumen than before as shown by the three tests. No peptone-reaction in the precipitated urine. The sediment contains a good many coarsely granular casts and a few renal epithelial cells in a state of fatty degeneration. No injection.

March 26.—Death early this morning. The urine previously voided is very pale, acid, and very highly albuminous, as shown by all three tests. The sediment contains coarsely granular short casts and isolated epithelial cells in a state of disintegration.

III. March 28.—A very large rabbit, fed upon oats and turnips. Urine abundant and acid, clear on filtration; no cloudiness with all the three tests for albumen. Injection of half a cubic centimeter of phosphorised oil.

March 29.—Urine acid. Acetic acid and ferro-cyanide of potassium added to the clear filtrate produce slight cloudiness; the same result on boiling with acetic acid and sulphate of soda. Nitric acid alone causes no decided turbidity; boiling causes the urine to become so dark that nothing can be recognised. Injection of half a cubic centimeter of phosphorised oil.

March 30.—Urine acid, yields a clear filtrate which is shown to be decidedly albuminous by the two first-mentioned tests; nitric acid causes slight cloudiness, which seems to become more marked on boiling, so far as can be made out in the dark discolouration which appears in the urine.

March 31.—Rabbit found dead. Urine by mistake not examined.

IV. April 2.—A medium-sized rabbit, kept in a cage since the day before yesterday, and fed upon oats and turnips. The urine obtained by compressing the bladder is acid, somewhat gelatinous, semi-fluid, but yields a clear

filtrate in which a slight cloudiness after long standing is produced by acetic acid and ferrocyanide of potassium alone; the other two reagents cause no alteration.

April 3.—Urine clear, acid; the clear filtrate free from albumen with all three tests. Three or four drops of phosphorised oil injected.

April 4.—Urine acid; acetic acid and ferrocyanide of potassium added to the clear filtrate cause slight cloudiness; no effect with other two tests. Half a cubic centimeter of phosphorised oil injected.

April 5.—Urine acid; the filtrate slightly albuminous with all three tests.

April 6.—Urine acid; the filtrate highly albuminous with all three tests. The rabbit died this evening.

The condition of the kidneys, which in the first two cases exhibited macroscopic fatty degeneration of their parenchyma at various points, has been already briefly alluded to in the discussion of the question as to how far albuminous excretions in the kidneys can be detected under the microscope (see page 36). The kidneys were first examined just as taken from the body, and subsequently one half was hardened by boiling and the other in strong alcohol. In the two first cases, in addition to hæmorrhage in several capsules and uriniferous tubules, we could distinguish deposits of coagulated albumen, some finely granular, others rather hyaline, in the tubules and capsules; in the third and fourth cases, on the other hand, besides slight fatty degeneration of the epithelium, there was only very moderate hyperæmia of the cortical substance, but no sign of albuminous excretion in the capsules or the uriniferous tubules could be recognised.

The perfectly harmonious results of these four experiments rendered it unnecessary to pursue the subject any further in this way, for I regard the conclusion to be deduced as absolutely positive, viz. that albuminuria is produced in rabbits as well as in dogs by very small doses of phosphorus, which disturb the general health only to an almost imperceptible degree, and, with the exception of fatty degeneration of the elements of the tissues, cause no remarkable changes in the kidneys; certainly no visible evidences

of inflammation. That the albuminous excretion in the first two cases was visible in the capsules, and not merely in the uriniferous tubules, is a proof that phosphorus, as we might *à priori* expect, acts injuriously, not merely upon the epithelium of the uriniferous tubules, but upon that of the glomerular tufts as well, and possibly upon these vessels themselves. The last supposition is borne out by the fact that effusions of blood appeared in the capsules. No fatty degeneration within these parts could be detected under the microscope, because the poisonous doses of phosphorus were very small, but in one case of poisoning by this agent in a girl, I noticed that the glomerular vessels were in a state of profound fatty degeneration.

The clinical observations of cases of phosphorus-poisoning are in complete accordance with the experimental results, and it therefore appears extraordinary that the absence of albuminuria should be described as a normal occurrence in poisoning of this kind. The truth is that albuminuria is the rule, and its absence the exception in such cases. In 1864, at a time, therefore, when phosphorus-poisoning first began to attract general attention, Meischner found albuminuria seven times in a collection of ten cases, and in a recent treatise Hessler tells us that he found this symptom twelve times in fifteen cases (73). We are justified in supposing that in the cases in which it was absent, its non-detection was possibly due to the fact that only the usual test, viz. boiling, was tried, and the more so, since in phosphorus-poisoning, as I have already mentioned, peptone-like bodies appear in the urine (see pp. 8 and 81). It almost appears as if the albuminuria in these cases had been disregarded for the sake of certain theories, or, possibly, the cause of the disregard may be found in the fact that the quantity of albumen was so insignificant. But to my mind this is a proof that the fatty degeneration of the epithelium, and especially that of the uriniferous tubules, as the anatomical examination shows, is the real cause of the albuminuria; and that this is not due to other complications, such as acute inflammatory processes, for as a result of these latter, the urine always contains a very considerable quantity of albumen. On the other hand, in mere fatty degeneration of the epithelium, it

may easily be supposed that but little albumen is allowed to escape abnormally into the secretion, for there is always the basement membrane to be traversed, and more than this, the epithelial cells do not all at once completely lose their functions. The secretion of the liver would furnish the desired analogy, but, unfortunately, we have no definite investigations with regard to its condition. Many years ago two statements were made, from which, if they were sufficiently minute, we could conclude that albumen is really found in the secretion when the glandular epithelium is in a state of fatty degeneration. Thus Thénard states that he found albumen in the bile in five out of six cases of "fatty liver," and Lehmann makes the same statement with regard to two cases of "fatty granular liver" (74). But even without these statements, what we have previously adduced definitely proves that albuminuria occurs in fatty degeneration of the renal epithelium following phosphorus-poisoning, and this latter should certainly no longer be adduced as evidence against the dependence of albuminuria upon fatty degeneration of the epithelium, for it is evidence of an opposite character. It is not denied that other factors may co-operate in the production of albuminuria in cases of phosphorus-poisoning.

Almost equally indefensible is the statement that the absence of albuminuria in various conditions of anæmia is evidence against the connection of the former symptom with fatty degeneration of the epithelium. It is certainly true that no albumen can be found in the urine in many cases of anæmia, but it is also indisputable that fatty degeneration of the renal epithelium does not accompany every form of anæmia, even when of a very severe type—I need only refer to chlorosis. There are in particular certain pernicious forms of anæmia which lead to fatty degeneration, but the insignificant amount of albumen excreted in these cases has caused its appearance to be disregarded. But albuminuria though trifling is a real symptom, and as deserving of notice as the albuminuria of congestion, which is likewise trifling as a general rule. The amount of significance to be attached to the "absence of albuminuria" in pernicious anæmia is shown by the single circumstance that, while from the stand-

point of one observer peculiar emphasis is laid upon the fact, another regards the occurrence of albuminuria in "progressive pernicious anæmia" as evidence in support of his own different view (75). As a matter of fact albuminuria occurs sufficiently often in severe forms of anæmia, in which fatty degeneration of the epithelium may fairly be supposed to exist or is found after death; and if some regard it as a rare phenomenon, a more uncommon one than my experience compels me to consider it, this difference is probably due to the fact that in such cases the quantity of albumen contained in the urine is certainly inconsiderable, provided that other morbid conditions are absent, and is often enough overlooked when the customary but not very reliable test by boiling is applied. And if the albuminuria is not an invariable occurrence, and even fails to appear in the majority of cases, we are not justified in concluding that the condition of the epithelium is of no importance; for the extent to which its function is disturbed, and whether this impairment has reached such a stage as to become manifest and capable of demonstration, are doubtless dependent upon the intensity of the degeneration. We must abstain from drawing general conclusions as to the importance, or unimportance, of disturbances which perhaps occur with a certain amount of regularity, but differ as regards their intensity and therefore, perhaps, as regards their importance. It is a well-known fact, for example, that fatty degeneration of the epithelium of the gastric glands is a regular occurrence in phosphorus-poisoning, and this is observed even when the drug is not administered by the stomach; and yet the evidences of disorder in this organ are often altogether inconsiderable, or at least in no way marked, especially when the poison is introduced into the system in some other way. Are we then justified in characterising the condition of the peptic glands and of their epithelium as of no consequence so far as the function of the stomach is concerned; but must we not rather assume that in such cases the degeneration is not sufficiently intense for the production of manifest disorder? We are compelled to adopt this assumption, because we know from other sources how important are the peptic glands for the discharge of the functions of the stomach. The case is a parallel

one with regard to the epithelium of the uriniferous tubules. We ascribe to these cells, and to these alone, the power and the task of preventing the albumen of the blood in the interstitial vessels, and of the lymph, from escaping into the uriniferous tubules, and we must therefore infer that when their degeneration, fatty or otherwise, has reached a certain degree, the fulfilment of their task will become impossible. Certainly, it is said, when they are in a state of fatty degeneration, but not in that of fatty infiltration! It would scarcely be necessary to advert to the difference between these two conditions, were it not for the fact that the physiological occurrence of epithelial cells, containing fat, in the kidneys of several animals, *e.g.* dogs and cats, has been referred to as evidence of the insignificance of fatty changes (76). But in these instances the condition is that of fatty infiltration, and no demonstration is requisite to show that the absorption of fat by otherwise healthy cells is one thing and the conversion of the substance of the cell into fat is quite another thing, and that the fatty changes in the two cases are of entirely different import as regards the function of the cell. It may be remarked that a "physiological albuminuria" often enough occurs in dogs and cats; this, however, has nothing whatever to do with any fatty contents of the renal epithelium, but is dependent upon other causes (see note, p. 20). Fatty degeneration of the renal epithelium occurs in animals after long-continued exposure to excessive heat. The urine, however, becomes albuminous very soon after the heat is applied, long before any fatty degeneration can be demonstrated, and, as before shown, this result is due to other causes (see p. 47). When, however, in the further course of such exposure, albumen occurs in the urine—a fact of which there can scarcely be any doubt, though I have no knowledge of this subject—a share at least in its causation must be attributed to the degeneration of the epithelium, and the more so since that cause to which the appearance of albuminuria at the commencement of the exposure to heat must be ascribed, viz. the increased blood-pressure, ceases to operate with the continuance of the abnormally high temperature and of its deleterious effects upon the muscular tissue of the heart.

Another kind of degeneration of the epithelium, the parenchymatous degeneration, is a well-known phenomenon in febrile infectious diseases. It is also well known that these diseases are characterised by the presence of albuminuria, to which the epithet "febrile" has been attached, and there is a point of resemblance between it and the albuminuria which occurs in phosphorus-poisoning and pernicious anæmia, namely this, that though as a general rule it is more marked than in these latter conditions, it is usually not a prominent symptom, especially when compared with the albuminuria of nephritis or of amyloid degeneration of the kidneys. On this account it was unknown or disregarded in former times, and only recently has it been considered worthy of consideration, but the instances of its occurrence become more and more numerous the greater the care bestowed upon its discovery. Whereas a few years ago the term "febrile albuminuria" was scarcely heard of, the condition is now universally regarded as an ordinary symptom. It is, however, difficult to estimate the share taken by the parenchymatous degeneration of the epithelium in the causation of this albuminuria, because a series of other conditions invariably co-operate therein, and to which, even more than to the degeneration of the epithelium, a certain influence must be ascribed, as will be shown in a subsequent page (see Section VI).

It remains now to allude to a peculiar form of degeneration of the epithelium, viz. the coagulative necrosis, types of which can be artificially produced by the administration of certain poisons, especially chromic acid, and likewise by petroleum, croton oil, and cantharidin. Albuminuria speedily follows the administration of these poisons, as Gergen has found in experiments with chromic acid. Lassar has discovered a marked peculiarity with regard to petroleum poisoning, viz. that a stage of propeptonuria precedes the excretion of the albumen which is coagulable by heat (serum-albumin, and globulin), that is, albuminuria proper (see page 10). Weigert (Kabierske) states that in animals poisoned with chromic acid salts, after some hours interval, only the epithelium of the convoluted uriniferous tubules is involved in the degeneration, whereas the glomerular vessels, the interstitial tissue, and the epithelium of the straight tubes remain intact.

A similar description is given by Lassar of the condition of the kidneys in poisoning by petroleum. This author and Kabierske have experimented with injections of indigo-sulphate of soda, which, when suitable methods are adopted, as Heidenhain has shown, is well known to be excreted only by the epithelium of the uriniferous tubules; and they were able to convince themselves of the destruction of these cells as a result of the administration of chromic acid and petroleum, and that all the other elements of the tissue remained apparently unaffected. Nevertheless, it must be assumed that the mischief is not confined exclusively to those epithelial cells and to the proper secretory apparatus, for, according to Posner, Voorhoeve, and Kabierske, the microscopical examination of kidneys after the administration of chromic acid shows that albumen is deposited not merely in the uriniferous tubules, where it has separated the epithelial detritus from the basement membrane, but in the Bowman-Müller's capsules as well. And a similar description is given by Browicz and Voorhoeve in cases of poisoning with cantharidin, but with this addition, that inflammatory changes were also indicated. In this instance, therefore, as in phosphorus-poisoning, it would appear that the albumen is derived partly from the interstitial vascular system and partly from the glomerular vessels, which, together with their epithelial investment, have likewise become affected (though to an extent not perceptible under the microscope), since in these cases there are manifest extravasations of blood in the capsules and uriniferous tubules (77).

The conclusion to be drawn from the above statements is that albuminuria occurs with a certain amount of regularity in all forms of degeneration of the epithelium, and especially of that of the convoluted tubules, and likewise that, under the same circumstances, the appearance of peptonuria and of propeptonuria takes place, a symptom which, as Lassar has shown, (and as other experiences prove to be not improbable, see page 12), may pass into albuminuria proper, though the presence of these substances is liable to be overlooked when the ordinary methods of investigation are employed. Whether it will henceforth be regarded as proved that the degeneration of the epithelium alone, in the sense in which we have explained it, is or is

not the cause of the escape of albumen, will depend entirely upon the degree of credit which is attached to the evidence yielded by the microscope. He who steadfastly refuses to recognise any lesion unless it can be demonstrated under the microscope, will be forced to regard the connection between albuminuria and epithelial degeneration as proved beyond doubt, for the appearance in question is all that the most expert microscopists have been able to discover. I for my part do not adopt this stand-point, which is based upon an over-estimate of the capacity of the microscope, as is also evidenced in the attempt to procure microscopical demonstration of the albumen contained in the normal transudation of the capsules (see page 35, *et seq.*). I am, therefore, very far from wishing to discover in all the cases which have been adduced, a rigid proof of the dependence of albuminuria upon degeneration of the epithelium, but I see in them evidence of a very strong character against the assertion that the condition of the epithelium of the uriniferous tubules is a matter of no importance with reference to albuminuria. There is still less evidence in favour of this assertion. On the other hand, so low an estimate will hardly be formed of the value of microscopical investigation as to cause it to be rejected as altogether worthless. In the albuminuria in question, the microscope shows us intense degeneration of the uriniferous tubules as a constant appearance, but no other striking alteration. Neither more nor less can be detected. It is therefore extremely probable, as a result of this investigation, that a connection exists between the two conditions, and that at least a considerable share in the causation of the albuminuria is to be attributed to the destruction of the epithelium. This conclusion is converted into a certainty by the fact that these observations only confirm the proposition which the theory of glandular secretion, and of the function of glandular epithelium, has laid down by anticipation as a necessary postulate, namely, that when these epithelial cells are destroyed, the obstacle to the escape of albumen from the interstitial vessels is broken through.

A peculiarity exhibited by the albuminuria which is connected with the various forms of epithelial degeneration becomes intelligible from this point of view. In fatty degene-

ration, as in phosphorus-poisoning, or in pernicious anæmia, the excretion of coagulable albumen is, as I have already stated, as a general rule only slight; on the other hand, in the coagulative necrosis due to chromic acid salts or petroleum, the urine contains large quantities of albumen. In the former instances the degeneration is the result of the conversion into fat of the protoplasm of the cells, which, however, usually remain connected with each other and occupy the same position when death occurs; in the latter case, as Weigert expresses it, destruction goes on rapidly and the cells become detached to a considerable extent. It is evident that in this last-named lesion, the rapid destruction of the entire epithelial investment must be followed by total, or almost total, abolition of function, and the disturbance at any rate must be far more serious than in that slighter form of degeneration in which, so to speak, the obstacle to the escape of albumen is certainly damaged, but is not entirely removed. This at least is the rule, for it is perfectly clear that even fatty degeneration may finally lead to complete disintegration and detachment of cells, and consequently in exceptional cases to a more intense degree of albuminuria, just as in such cases the detachment of these cells in a state of fatty degeneration and their passage into the urine, simultaneously with the albumen, will induce a form of chyluria—a chyluria the result of phosphorus-poisoning, which has really come under observation (78).

We are thus led to consider the possible occurrence of a form of albuminuria, as a simple result of the passage into the urine of detached epithelial cells belonging to any portion of the renal parenchyma. For some years past, I have drawn attention to this possibility, and it must, I think, *à priori*, be admitted, without any scruple, that when epithelium becomes detached, it is not merely the fixed and therefore undissolved albumen contained in the substance of the cells which finds its way into the urine; for this is a matter of course, unless all the cells separately and collectively were converted into fat; but a portion of the protoplasm, in a state of disintegration and passing into a fluid state, may become dissolved in the neighbouring current (79). There can be no dispute on this point, and

no dispute has arisen, but a process of this kind has been thought unworthy of consideration, as appears from the notion expressed by Bartels (80) to the effect that these admixtures of albumen can amount only to very minute quantities, a notion which, as in phosphorus-poisoning and other forms of albuminuria, has led to the error of totally disregarding this symptom. But even so slight an albuminuria as this may be, is a real albuminuria; and it is the more remarkable that it has attracted no sort of attention, when it seems as though sufficient consideration could not be attached to the excretion of albumen in cases of renal venous congestion, amounting as it does (and as Bartels himself so justly remarks) "rarely to more than 0·1 per cent." of the urine. If an excretion of albumen, amounting within twenty-four hours at most to half-a-gramme or a gramme, as is common in renal venous congestion, be not so minute as to be unworthy of consideration, it follows that albuminuria, as a possible result of disintegration of the epithelium, has certainly some claim upon our notice. But why must this latter form be so insignificant a symptom? Is it the case that all the epithelial cells which may decay and become detached,—the epithelium of the capsules, of the glomeruli, and of all the other portions of the uriniferous tubules,—are not in sufficient quantity to yield a daily supply of half a gramme to a gramme of albumen, even if all these epithelial cells do not simultaneously perish? In my opinion a positive presumption is abundantly justified by estimating how much of the weight of the renal parenchyma is made up by the aggregate of these epithelial cells.

If, therefore, no objection can be raised against this view—and I can think of none that is really tenable—it must be admitted that the disintegration and detachment of cells may give rise to the appearance in the urine of albuminous substances, differing from the albumen of ordinary albuminuria. As a general rule the cell-protoplasm appears to contain albuminous substances (vitellin and myosin) resembling globulin, and which as such, or in some other stage of transformation, may find their way into the urine; indeed, according to Gottwalt's observations, the kidneys free from blood contain from seven to eight times as much globulin as serum-albumin (81).

We know that globulin along with serum-albumin is found in the urine in very variable quantities, so that we might perhaps suppose that, according as the albumen of the renal parenchyma is mixed with the urine, this latter secretion will contain more or less globulin in proportion to the serum-albumin. Our defective insight into the chemical processes of living and decaying cells does not allow us to do more than indulge in guesswork on this point. But it appears justifiable to mention it, for it is in these forms of albuminuria which are connected with rapid and severe epithelial decay, that the albumen exhibits several reactions differing from those ordinarily met with. This is, however, not the only possible means of explaining the occurrence of unusual forms of albumen in the urine, for there is another and a more probable explanation, viz. that the substances in question may be conveyed to the kidneys with the blood, in consequence of some change in the composition of that fluid. We shall now discuss this subject, so far as the change alluded to has any influence in the production of albuminuria.

V. The Condition of the Blood as Influencing the Production of Albuminuria.

That albuminuria is caused by an abnormal condition of the blood, is the oldest view, and the same which Cotugno, the discoverer of albumen in the urine, suggested, and which his contemporaries and immediate successors adopted and disseminated; and this theory necessarily remained unshaken so long as observers were ignorant of the connection between albuminuria and dropsy and renal disorders. But the views of physicians were dominated by this theory even for some time after the connection between these conditions was discovered by Dr. R. Bright; subsequently, however, it lost its prominence, as the progress of investigation led to the discovery of various disorders of the kidney in states of disease accompanied by albuminuria, while little or no further knowledge was gained with regard to the supposed alterations

in the blood. These changes in the blood were supposed to be due to a morbid condition of the albumen, which, in consequence, differing from the normal albumen of the blood, passed into the urine; and the cause of this morbid condition was sought for in some abnormal process of digestion or of tissue-change, or in the retention of excrementitious matters, &c. But all that could be brought forward in support of these theories resolved itself into more or less obscure suppositions or arbitrary assumptions, and consequently it was only to be expected that the theories which attributed the causation of albuminuria to the condition of the blood—the theories of a hæmatogenous albuminuria—would be set aside in favour of a nephrogenous albuminuria, so that at the present day the former views have very few adherents (82). Moreover, it has been shown by the very ingenious experiments of Stokvis, that at least in ordinary cases of albuminuria and dropsy, or of so-called "Bright's disease" and amyloid degeneration of the kidney, the albumen does not, certainly as a general rule, possess the power of inducing albuminuria in healthy kidneys. Albuminous urine, or the blood-serum of patients suffering from albuminuria, was injected into the blood or under the skin of healthy animals, and in twenty-one out of twenty-three experiments of this kind no albumen was found in the urine. In the two cases in which albuminuria occurred, it was very remarkable that the urine used for injection had been taken from a patient suffering from albuminuria without demonstrable renal disease. The very cautious conclusion drawn by Stokvis is, that in the majority of cases a modification of the albumen of the blood is not the existing cause of the albuminuria, but that it must be left for further experiments to decide whether such may be the case under exceptional circumstances, as it is not improbable from *à priori* considerations (83). My opinion is, that this conclusion must be adopted in its entirety; it corresponds even now, after nearly a decade and a half, with the actual state of our knowledge of the subject. That view especially must be rejected which attributes the occurrence of albuminuria in renal diseases to changes in the blood, particularly in the conditions, including amyloid degeneration and congestion, formerly, and to some extent also nowadays, classified

under the name "Bright's disease." There is no doubt that a cause for the symptom in question is to be found in a renal lesion. This latter certainly may (and this view is highly probable) often originate in a morbid condition of the blood, which may therefore be fairly described as the *indirect* cause of the albuminuria in these cases.

It may be difficult to explain the occurrence of albuminuria in those much rarer cases in which no lesion of the kidney exists. For it is beyond doubt that there are such cases, and that albuminuria is not always symptomatic of a nephritis or of some other demonstrable kidney-lesion. I need only refer to the detailed description already given (see page 15, *et seq.*) of albuminuria as occurring in perfectly healthy men, or to the albuminuria in patients who show no other indication of kidney-disease, and in whom when death occurs from some other cause, the most careful examination fails to detect any abnormality in the kidney, any trace of degeneration of the epithelium, any swelling and cloudiness of the parenchyma, appearances which are yet regarded by many indeed as devoid of significance with regard to albuminuria. We have become acquainted with the existence of circumstances which, in certain conditions of the circulation, may give to albuminuria in the absence of any disorder of tissue in the renal parenchyma; but not in all cases of this nature can the circulatory changes be positively demonstrated or even assumed as probable, without straining the interpretation of the symptoms. It appears to me that for such cases, probability must be admitted for the theory that the albuminuria is due to changes in the composition of the blood. I say designedly "changes in the composition of the blood," and not "changes in the albuminous substances in the blood" as is usually expressed by those who make use of the term "hæmatogenous albuminuria." Not that I consider an abnormal condition of the albuminous substances as impossible; on the contrary, I regard it as not only possible but even not improbable, so far as many cases are concerned, and I will soon give my reason for so doing; but I think it is at least as probable that other changes in the composition of the blood, having no connection with any abnormal condition of the albumen, may induce albuminuria, and I am alluding not to hypothetical

changes of composition but to such as can be demonstrated beyond possibility of doubt.

The urinary secretion in great measure depends, as has been already sufficiently explained, upon filtration from the plasma of the blood, that is, from a saline albuminous solution; and in accordance with the numerous experiments which have been performed with regard to the filtration of such fluids, it can be regarded as quite certain that the quantity of albumen contained in the filtrate is, *cæteris paribus*, dependent upon the quantity of albumen and salts contained in the original fluid. The more albumen the latter contains, the more of this constituent, *cæteris paribus*, will be found in the filtrate, though this latter quantity does not rise and fall in a degree exactly proportionate to the amount contained in the original fluid. As regards the influence of the saline constituents, the results of the investigations of Hoppe-Seyler, v. Wittich, and Nasse, notwithstanding a few differences in details, practically agree in showing that the quantity of albumen which passes through the filter increases with the increase of the saline constituents of the fluid. This is especially the case in regard to chloride of sodium, with which the majority of the investigations were made, but it is also true in the case of other salts (nitre, chloride of calcium); and it is important to observe that it holds good with respect to urea, the influence of which in facilitating the passage of albumen through a filter has been observed by Hoppe-Seyler, and recently by Newman (84).

In face of the above facts there is no reason to refuse to accept the theory of a hæmatogenous albuminuria, as is now almost universally the custom, for there cannot be a moment's doubt that, under multiform normal and pathological conditions, both the dissolved albumen and the salts of the blood may become increased, and further, that such increase really takes place. After every meal, which does not contain too great a proportion of water or an unusually small quantity of albumen and salts, the composition of the blood may become altered in the manner described, and may be a cause of albuminuria, in so far as more than the normal amount of albumen transudes through the glomerular vessels. As a matter of fact, with healthy kidneys, the occurrence of

albuminuria during digestion has been noticed by numerous observers (see page 19), and to it, therefore, a peculiar name, "digestion-albuminuria" (albuminuria alimentaria), has been attached. That likewise, in the ordinary forms of morbid albuminuria, an increase in the excretion of albumen is a frequent result of digestion, altogether independently of other conditions, is a fact already noticed by Parkes and Gubler (85), and one that has been minutely investigated by Brunton and Power (86), and confirmatory evidence of which abounds on all sides. Moreover, in addition to the increase of albumen and salts, the above-mentioned investigations of Hoppe-Seyler and Newman show that regard must be had to the increase of the quantity of urea in the blood, which occurs some time after the digestion of albuminous food, and manifests itself in the increased excretion of urea. It is well known, on the other hand, that the urine becomes increased in quantity while digestion is going on, and such increase is in a measure independent of the process of filtration, and is in excess of the activity of the latter, inasmuch as the glandular secretion proper is at the same time augmented, owing to the influence of the urea, the salts, &c. These facts, as we have already explained (see pages 28, 40), constitute a reason why in most cases the discovery of the quantity of albumen of the urine is rendered difficult. For if a fluid containing much albumen transudes through the glomerular vessels, and receives in addition, a greater quantity of non-albuminous fluid, consisting of the secretion of the glandular epithelium, the quantity of albumen in the urine as a whole may be very small, and may progressively diminish until it reaches the proportion observed when no digestion is going on, or even falls below it. The concurrence of particularly favorable circumstances is, therefore, required for this "digestion-albuminuria" to make its appearance, and it may be soonest expected after a meal containing much albumen but very little water. Food containing much saline matters will be less favorable in this respect, for the ordinary salts may act in two directions, since on the one hand they facilitate the filtration of albumen in the glomeruli, but on the other, the secretion of water by the glandular epithelium. This purely theoretical deduction

is confirmed by experience, for the diet after which "digestion-albuminuria" is noticed is usually one in which animal food largely preponderates. Christison has noticed temporary albuminuria in persons who had eaten much cheese (87). I have already (page 19) alluded to the same symptom occurring in an otherwise perfectly healthy physician, after almost every meal consisting largely of meat, and many similar cases are recorded in the literature of this subject. I am now referring not merely to the instances in which albuminuria occurred after eating eggs, for these are only partly traceable to the same cause, and in the main a different explanation is to be found for them (see page 100).

It is extremely probable that under pathological conditions similar changes take place with regard to the quantity of albumen and salts contained in the blood, and especially in the direction of an increase, whether of an absolute or of a relative character. But this assumption is based only upon theoretical considerations, for we have at present no observations which would prove its truth. Only in the case of cholera, and other conditions associated with profuse watery evacuations, we know from C. Schmidt's classic investigations that the albumen of the blood becomes relatively increased and that the saline constituents are similarly affected for a very brief interval (88). We know somewhat more with regard to the increase of urea in disease. Gscheidlen (89) has shown that the quantity of urea contained in the blood becomes increased in the febrile state, and the same thing will take place in all those conditions in which increased disintegration of albumen is accompanied by diminution of the urinary secretion, as in phosphorus-poisoning, according to Storch, Bauer, and Cazeneuve, and, according to A. Fränkel, as a result of insufficient supply of oxygen (90). In all these cases there will be an abnormally large formation of urea which will be excreted with the urine, but the amount excreted may easily be less than the excessive formation, since, for reasons which we need not here discuss, there is a considerable and simultaneous depression of the renal activity. The result, therefore, in such cases may be an accumulation of urea in the blood, whereas in other cases in which the increased formation of urea is met by a corres-

ponding increase in the secretion of urine and urea, as, for example, in diabetes, no accumulation can be supposed to occur. An accumulation of urea in the blood has been clearly demonstrated in disease of the kidney itself, simply as a result of impeded excretion, and always associated with diminution of the quantity of urine.

There is, therefore, in all these cases in which the albumen or the urea, and possibly also the salts, are increased in quantity, a certain factor present, endued with the power of causing a much increased transudation of albumen, and consequently albuminuria. But it is by no means a single factor, for a number of more or less active influences, capable of producing the same effect, come into play in the above-mentioned instances. Thus in phosphorus-poisoning, there is the degeneration of the epithelium, and the diminution in the quantity of urine consequent upon diminished arterial pressure; this latter is also present in cases of profuse watery evacuations, in cholera, violent diarrhœas, &c., while in fever, of which we shall presently speak particularly, many other factors co-operate, by which the albuminuria may be explained.

While, therefore, there is a positive basis of facts for the assumption that quantitative changes in the composition of the blood may, under certain circumstances, cause albuminuria to appear, or increase this symptom if already existing, the other assumption, hitherto maintained only by the supporters of a hæmatogenous albuminuria, to the effect that the symptom in question has its origin in qualitative changes in the albumen of the blood, can claim no equally definite and positive facts as a foundation but only sundry reasons which invest it with a certain amount of probability. This, however, holds good only with regard to the pathological albuminuria, for there is a certain kind of physiological albuminuria which is doubtless a result of qualitative changes in the albumen of the blood. This is the albuminuria which occurs after the introduction of egg-albumen into the blood—and not merely after the immediate introduction or its injection under the skin, as often done in experiments, but likewise after its introduction into the stomach. Many observers, namely Tégart, Brown-Séquard, Becquerel,

and Barreswil, Hammond, Cl. Bernard, J. Chr. Lehmann, and Stokvis, have observed albuminuria after eating many eggs, or a long-continued diet of this character, the experiments being performed on themselves, or on other persons, or on rabbits and dogs (91). As a general rule, this albuminuria is explained as due to the readiness with which egg-albumen passes through a filter; it is consequently supposed to traverse the glomerular vessels more readily than is the case with the normal albuminous substances of the blood; and I have already drawn attention to the fact (see p. 23) that this generally received explanation involves a recognition of the theory according to which the albuminous substances really do pass through those vessels, though in quantities varying according to the capacity for filtration possessed by each. The investigations of Lehmann, Stokvis, and Creite (92), show beyond doubt that egg-albumen is really excreted as such, and that in the majority of cases the albuminuria disappears with its excretion. It must, therefore, be assumed that in those cases in which a large number of eggs are introduced into the stomach, a portion at least escapes the action of the gastric juice and passes unchanged into the blood; and this theory is supported by the fact that Stokvis failed to produce albuminuria in rabbits when he introduced coagulated albumen into the stomach. The portion really digested may, however, by increasing the quantity of normal albumen in the blood, have contributed to the production of albuminuria in the way just described (see page 97, *et seq.*).

It appears, however, as if the admixture of dissolved egg-albumen might lead to the production of albuminuria in yet another way, for both Lehmann and Stokvis several times observed that a long-continued albuminuria was the result of injecting the albumen in question into the blood, and that more albumen (in one of Stokvis's experiments, four times as much) was excreted than was injected. In these cases, therefore, the presence of an extraneous albuminous substance in the blood must have set up, probably in the kidney, some kind of process which led to the production of albuminuria in the strict sense of the word—that is to say, to the excretion of coagulable albumen of the system itself. Here we are reminded of the fact that Creite often witnessed the occurrence

of albuminuria without admixture of blood, in rabbits, as a result of the injection of the blood-serum of animals belonging to different species. To explain this we must assume, either that the albuminous substances of these kinds of serum more readily filter through the kidneys of rabbits than through those of the animals to which they belonged; or that extraneous serum of this kind induces serious derangements, with albuminuria as an accompaniment. Taking into consideration the effect, presently to be discussed, of the serum upon the blood-corpuscles of other animals, it must be admitted that the latter assumption is the more probable of the two.

Nothing certain is yet known as to the effect produced by the injection into the blood of other soluble albuminous substances, peptone chiefly excepted. J. Chr. Lehmann observed no albuminuria after injections of Lieberkühn's albuminate of soda, of solutions of syntonin, myosin, and fibrin, whereas Runeberg witnessed the occurrence of albuminuria after the injection of an albuminous substance, which he obtained by dissolving in soda casein precipitated from milk by means of acetic acid, but not when he injected pure milk or milk mixed with soda (93). The former fluid possessed great capacity for filtration, and the question arose whether the albuminous substances made use of by Lehmann were less adapted for that process, for solubility alone is no measure of the capacity for filtration.

The other albuminous substances, distinguished by their capacity for diffusion and filtration, with which we are acquainted, are peptone (probably also propeptone) and hæmoglobin. No experiments in this direction have as yet been made with propeptone, but it may with certainty be assumed that its behaviour is the same with that of the other two substances, which, introduced in a state of solution into the blood, are, in accordance with theory, readily excreted in consequence of the marked capacity for filtration which they possess. With regard to peptone we know that it may find its way into the blood by injection, or by the absorption of exudations in which it is contained (as in pleurisy, pneumonia, and rheumatic arthritis), and that peptonuria then makes its appearance. The same origin

may be assumed in the case of propeptonuria. It is well known that the colouring matter of the blood is dissolved, not merely as a consequence of the injection, but likewise under various circumstances, as a result of the destruction of the red corpuscles in the blood. In this way its excretion takes place, and thus we find hæmoglobinuria (methæmoglobinuria, hæmatinuria) as a result of various poisons, and in certain states of disease (periodic hæmoglobinuria, severe infectious conditions), and, as Creite, Landois, and Ponfick have observed, after the introduction into the circulation of blood, or only blood-serum, of an animal belonging to a different species (94). The excretion of the hæmoglobin, however, appears to take place not only by filtration through the glomerular vessels, but besides this, as is the case with other colouring materials, in a specific manner through the epithelium of the uriniferous tubules (95).

This, then, is almost all that is known with certainty with regard to the dependence of albuminuria upon qualitative changes of the blood, and particularly upon alterations of the albuminous substances. All the results of experiments in reference to this question amount apparently to this, viz. that albuminous substances, normally not dissolved in the blood, appear in solution in that fluid, and that they are excreted by the kidneys in proportion to their capacity for filtration, just as was to be anticipated from the views we have developed with reference to the filtration in the glomerular vessels. In consequence of the greater capacity for filtration possessed by these substances a larger amount passes through the glomerular vessels than in the case of the normal albuminous substances of the blood; it therefore follows that the former will be more easily discoverable in the urine than the latter, and the albuminuria is mainly accounted for by this fact. It would, moreover, appear that abnormal excretion of the ordinary albumen may be induced as a result of extraneous admixtures of this description.

It might be thought that these facts amounted to sufficiently presumptive evidence in favour of the view that many forms of albuminuria are dependent upon qualitative changes of the blood, and particularly of its albuminous constituents. It is true that this view has met with but

little acceptance of late years, but for a reason which, if carefully scrutinised, will be found to be strictly of an extrinsic and formal character. The invariable custom is to include in the term albuminuria nothing but the excretion of coagulable albumen from the blood, and, as before noticed, to consider that the excretion of other albuminous substances is not comprehended under the same term. In this sense the only recognised albuminuria, which assuredly depends upon a qualitative change in the blood, is that which is caused by the introduction of egg-albumen; but this is usually considered as devoid of significance in a clinical respect. And it is quite true that up to the present time our knowledge of this albuminuria has been derived from experiment and not from clinical observation. It is indeed possible that that which is brought about in a healthy man or animal by the ingestion of an excess of egg-albumen, may become developed in a diseased organism, especially in one which possesses abnormal capacity of digestion, by a moderate or even small amount of such food, and in that case this excretion of egg-albumen would certainly possess a clinical significance as well—the same significance as may perhaps be assigned to the ingestion of amylaceous food by a patient suffering from mild diabetes, who, when he avoids such food altogether or limits its quantity, excretes no sugar, and in a general way shows no symptom of disease. It is well known that even healthy persons may be made to excrete sugar (glycosuria alimentaria) by feeding them upon large quantities of sugar and starch, and since this fact does not induce us to remove the so-called mild form of diabetes from the category of pathology, there is no greater amount of objection to regarding this excretion of egg-albumen as devoid of significance in a pathological respect. The question would be to determine the boundary between moderation and excess of supply—obviously a difficult task.

With regard to the other albuminous substances which occur in the urine, not merely in the course of experiments, but likewise under purely clinical conditions in various forms of disease, we are quite justified in separating hæmoglobinuria from albuminuria proper, inasmuch as the hæmoglobin, as well as the colouring matters derived therefrom, does not

belong to the albuminous substances properly so-called. On the other hand, no strict line of separation can now be drawn between "peptonuria," "propeptonuria," and albuminuria, for the two former occur alternately with albuminuria in the strict sense of the word, and also combined with it (see p. 9, *et seq.*). If, however, they must be considered as belonging to albuminuria in the wider sense of the term, this view involves the admission that forms of albuminuria exist which are dependent upon qualitative changes in the albumen of the blood, for I scarcely know what other form of albuminuria could be expected to arise from such qualitative changes, and otherwise we should find ourselves in an endless circle of terms. If coagulable albuminous substances are excreted, and albuminuria in the ordinary strict sense therefore exists, the substances in question must necessarily be invariably regarded merely as serum-albumin or as globulin (we can disregard egg-albumen, as that appears only after introduction from without), and it will therefore be needless to assume the existence of any qualitative change, or of any modification of the albumen of the blood. If, however, one of the substances, in other respects resembling albumen, but wanting in coagulability, is found in the urine, if, therefore, some modification be present, this is set aside as "peptonuria or propeptonuria," and is regarded as something peculiar. But in this way it will scarcely ever be possible to prove or disprove the existence of albuminuria due to changes in the condition of the blood. For the distinctions once considered important aids towards demonstrating modifications of albumen in the urine, and consequently in the blood, are of quite subordinate significance if we take into consideration the facility with which the several albuminous substances undergo conversion; and all the experiments, especially those performed some years ago, with the view of demonstrating the presence of morbid modifications of albumen by the variations in the diffusion of albuminous urines, are of little value, because such variations are caused by alterations in the quantity of the saline constituents, the urea, &c.

Much more value would be attached to a physiological experiment which would demonstrate a property of the

albumen contained in the urine or the blood in cases of albuminuria, that is, if it could be shown that this albumen injected into the blood or subcutaneously, was, like egg-albumen, capable of producing albuminuria. It is true that, connected with this subject, we have a single isolated statement by Stokvis, which has been already incidentally referred to (see p. 95). As an exception to, and differing from, the great majority of his other experiments in this direction, he found that the albuminous urine of a patient, free from any manifest disease of the kidney, produced albuminuria in one dog and one rabbit, the symptom disappearing in a few days. We cannot suppose that this extraordinary result was due to any oversight or confusion; had any such existed, Stokvis must have discovered them, for these two experiments are inconsistent with the conclusions warranted by all the others, and must therefore remain as unexplained exceptions. For the present, however, it will be well not to invest them with any great amount of significance, but to regard them only as an index for further researches in this direction. Until the time comes that they are confirmed, the question as to the existence in diseases of a modification of the albuminous substances, capable of producing an albuminuria not to be designated peptonuria or propeptonuria, must be regarded as unsettled.

In like manner the question as to the means whereby any modification of albuminous substances of the blood takes place, must be considered as still awaiting solution. There are many possibilities, but at present there is no theory which can be proved and certainly none which can be disproved. It is therefore possible that, in accordance with the theory especially favoured by Prout among English observers, abnormal processes of digestion are responsible for the passage into the blood of an albumen differing from the normal substance; or that under the influence of some kind of morbid condition in the blood and fluids of the body, such alterations take place; as the old doctrines of crasis were always ready to assume. Inasmuch, however, as we are not as yet able to recognise with certainty any single morbid modification of albumen in the blood and fluids, we ought in this matter to assume an attitude of reserve, if not of incredulity.

Among the alterations in the condition of the blood, we have finally to notice one which contrasts with the above-described deviations which concern only the chemical composition. The alteration in question is of a physical character, having perhaps the power of increasing to an abnormal extent the transudation of albumen until albuminuria is produced. I refer to an increased temperature. We have already alluded to the fact that filtration of albuminous solutions is facilitated by increase of temperature, but we do not know what proportion the albumen contained in the filtrate bears to that of the original solution (*conf.* page 51). We must, therefore, be satisfied with having drawn attention to this point which may, among other things, be of unmistakable importance in reference to the albuminuria of febrile disorders. There is also another point, possibly connected with this one just mentioned, but to which in the present state of our knowledge we can make only a passing allusion, viz. that with the elevation of temperature the molecular composition of the albuminous substances becomes so changed, that they pass more readily through a filter than is the case in their normal condition. On this subject conjectures have been hazarded from many quarters, but nothing certain is known, and, in accordance with the explanation already given, the occurrence of such a change under the influence of increased temperature, may be capable of recognition only because the coagulable albuminous substances are converted into peptone and propeptone.

As far as we have gone at present in our description of the influence of modified conditions of the blood upon the production of albuminuria, we have referred only to the processes of filtration going on in the kidneys, but we have said nothing about the process of secretion proper, because, to all appearances, this latter, in the majority of cases, is only indirectly concerned. I know of only one instance which supports the view that true glands may allow the abnormal escape of albumen, when extraneous albuminous substances are present in the blood, one of these substances being allowed to escape. This observation, likewise made by Stokvis, refers to the case of a strong dog, in which egg-albumen was subcutaneously injected, with the result that the secre-

tion from the parotids, previously filtered and found perfectly non-albuminous, now contained a marked proportion of egg-albumen. If it be desired to draw any conclusion from this observation, it may be assumed as probable, that not only the glomerular vessels, whose action in this respect has been proved, but likewise the interstitial vascular system participates with the epithelium of the uriniferous tubules in the excretion of egg-albumen in the kidney. Besides, the latter portions of the kidney may, as already stated, in consequence of morbid changes in the condition of the blood, indirectly contribute towards the production of albuminuria, inasmuch as the latter gives rise to derangement of nutrition and degeneration of the epithelium, conditions which, in the way already described, lead to the excretion of albumen.

VI. Certain Peculiar Forms of Morbid Albuminuria.

In the foregoing sections we have become acquainted with so many varying conditions which may assist in the production of albuminuria, that we cannot agree with the attempts which have been made to attribute to a single cause all those forms of albuminuria which do not originate in the coarser disorders of the kidney. How the conditions which are submitted to our consideration, viz. the state of the circulation and of the blood-pressure, the condition of the membranes concerned in the secretion, and the state of the blood, act in various diseases, is in a general way too little known for us to be able to estimate the influence each exerts in every case. But this much we know, that in the majority of diseased states the separate conditions mutually influence each other, so that it is very difficult to discover in every instance the really active condition, and altogether impossible always to point out the only one that is so. The conditions are proportionately simple in a few cases only, and these have been described in their proper places in foregoing pages; we refer especially to the albuminuria in venous congestion (page 71, *et seq.*), in convulsive conditions (page 51, *et seq.*), and at any rate in many forms of poisoning (pages 55 and 80). In all other cases the conditions are

less simple and intelligible, so that although it may be positively affirmed that the albuminuria has originated in some one or other or in several of the ways above described, its exact cause, and wherein consist the conditions for one or other mode of origin cannot be so definitely stated. The best illustration of the manner in which various conditions co-operate to produce albuminuria, and how these may alternate in one and the same morbid process, is to be found in what Gerhardt was the first to designate as "febrile albuminuria" (96). And in this instance we are in the position of being able to recognise, with distinctness, at least a few individual conditions. Even with regard to the conditions now occupying us, there is still much that is wanting for the completion of our knowledge of the processes in fever, but the more recent investigations with reference to the circulation, tissue-changes, and nutrition, enable us to indicate the conditions, some with certainty and others with more or less probability, which will give rise to albuminuria in various febrile disorders unaccompanied by any special lesion of the kidneys. In the first place, as regards the circulation, and the blood-pressure in particular, which, as previously explained in detail, has the greatest influence in the production of albuminuria, it is obvious that it does not remain the same, or invariably undergo changes of the same character, in all febrile diseases, or in any single disease throughout its whole course. That it should remain unchanged is not to be expected, inasmuch as the heart's functional capacity, upon which the blood-pressure mainly depends, varies according to the nature and duration of the disease. As a general rule, in those febrile conditions in which the cardiac activity has not been previously impaired by the severity of the infection, the action of poison and the like, the blood-pressure at the commencement must of necessity be increased in consequence of the numerical increase in the heart's pulsations which accompanies the rise of temperature, the force of the contraction in each separate systole remaining the same, or possibly augmented by the increase of temperature. This result has been obtained in the few experiments recently performed by Zadek and Basch with the object of measuring the pressure in febrile conditions (97). In this respect, therefore, the condi-

tion of a patient suffering from fever exactly resembles that of an animal artificially heated, so long as the increase in the bodily temperature does not exceed certain limits and does not exercise a paralysing effect upon the nervous apparatus (see page 47). The diminution also in the quantity of urine is as common in fevers, and for reasons that are well known, as in cases where the temperature is artificially raised, so that those instances of febrile albuminuria which present themselves, accompanied by a properly maintained action of the heart, a strong pulse, &c., may, without any hesitation, be classed together with that form which is induced by the artificial application of heat. The elevation of the bodily temperature is the essential, if not the only condition for the production of the albuminuria, the immediate origin of which has been explained in previous pages. The conditions may be entirely different in the subsequent course of a fever, or in cases of severe infection or poisoning, or even at the commencement of a fever in a person already debilitated. In these cases, no doubt, the blood-pressure will fall below the normal degree. And therein, as we have seen when discussing the influence of diminished pressure, may be discovered a condition for the production of albuminuria, but one which will probably be insufficient *per se* for such a purpose, inasmuch as the pressure is not usually reduced to a considerable extent. But there are other factors of various kinds which act in the same direction, and whose influence is greater in severe febrile conditions accompanied by decreasing cardiac activity than in others; among these a prominent place may be assigned to derangements of the nutrition of the renal epithelium and possibly also of the walls of the vessels. We know that such derangements occur, and that their intensity varies with the disease which originates them, and we have already discussed their possible influence in the causation of albuminuria. As a further step we have to consider the altered condition of the blood in fevers. We do not know much about this, but we know enough to enable us to assert that this factor also can play a certain part in the production of febrile albuminuria. I here refer to what has been stated in a previous page with regard to the influence which the increased amount of urea contained in the blood exercises

upon the filtration of albumen (see page 97, *et seq.*) The above-mentioned factors are assuredly sufficient for the explanation of every febrile albuminuria in the ordinary sense of the words, involving as they do only the excretion of coagulable albumen. But the possibility is by no means excluded that the febrile varieties of the metamorphosis of tissue, the changes in the condition of the blood, unknown it may be to us, but doubtless present, may serve as a screen for a number of other factors favorable to the development of albuminuria. This supposition must almost necessarily be accepted with regard to the instances designated by Gerhardt as "latent albuminuria," in which albuminous substances, non-coagulable by heat, were excreted; and consequently for cases of peptonuria or propeptonuria, as these occur not merely in non-febrile but also in febrile diseases, and, as it would appear, often under the influence of the fever alone, and not of the particular disease which the latter accompanies. The few statements that can be made on this subject have already found a place in the foregoing sections (see pp. 8 and 107). Unfortunately, under the circumstances, the domain of facts has its narrow limits while there is a wide field for hypothesis.

This wide field would have to be thoroughly traversed if the attempt were to be made to explain every instance of albuminuria occurring in the course of disordered states, but without the kidneys being deeply involved. I do not assert that the morbid processes are everywhere enveloped in so much obscurity, that the causes which have led to the albuminuria are always beyond our comprehension. This is by no means the case, for in this or that disorder the particular circumstance to which the albuminuria is due can often be definitely specified; we know, for example, that the albuminuria in cholera is due to the great inspissation of the blood, whereby it is made to contain more albumen and, for a short time, more saline constituents (see pp. 97—99); secondly, to the intense venous congestion of the kidneys; and lastly, to that degeneration of the epithelium and walls of the vessels which necessarily results when the tissues are traversed by abnormally constituted blood flowing in an abnormally retarded current. Each of these factors may *per se*, as

already explained, give rise to albuminuria; two or three of them combined will, as a matter of course, be the more certain to produce it. With regard to the temporary albuminuria, not seldom to be observed in diarrhœas of other kinds, and which has recently attracted attention (98), we are to some extent justified in attributing it to the diminution of the water of the blood and the diminished blood-pressure which usually co-exists; but these exhaust the category of morbid conditions, apart from kidney disorders proper, in which there are at least a few facts as a basis of support for the explanation of any existent albuminuria.

There are, as a matter of course, on the whole but few difficulties in explaining the excretion of albumen in renal disorders proper, *i. e.* nephritis in its various forms and amyloid degeneration; that is to say, in those processes in which albuminuria is so constant and so marked a phenomenon that they were in former times simply identified with it. And the acute inflammatory processes are the simplest of all as regards the facility with which the albuminuria can be explained. For the fact is that each vascular portion in a state of inflammation is abnormally permeable, and permits the escape of a fluid containing in particular a large amount of albumen and of cells in addition. Albuminuria is consequently a necessary symptom of all active inflammations in the kidneys, that is, both of those forms which are acute from their commencement and throughout their course, and of the acute inflammatory exacerbations which occur in the chronic forms. If it be not invariably observed in inflammations of a purely circumscribed character, the reason for this may be partly because the connection of the inflamed portions with the discharge-tubes is interrupted, and partly also because the amount of albumen yielded by the area in question is too small to be detected, especially if such circumscribed inflammations do not affect the glomerular vessels. For there can be no possible doubt but that the glomeruli are invariably the principal and most prolific source of the albumen, and hence it is that the urine containing the highest proportion of albumen is always yielded in cases of acute (diffuse) nephritis. In every acute inflammation of the kidney the glomeruli are especially affected, or we may rather say tha

they are the starting-point for the inflammation, as has been shown by the investigations of Klebs, Salvioli, Cohnheim, Friedländer, and Ribbert (99) on the subject of glomerulo-nephritis. In all cases the deposit of exudation in the Bowman-Müller's capsules can be easily demonstrated. The possible participation of the interstitial vascular system in the excretion of albumen must, however, be assumed, because the interstitial tissue and the epithelium of the uriniferous tubules almost always become involved, and an escape of albumen into the interior of the tubules may doubtless take place in consequence. But from the anatomical appearances it is easy to form a conception of the condition of the urine in some other respects in every case of acute nephritis, and it would appear especially that the diminution in the quantity must necessarily be referred to the co-operation of several conditions. In the first place the glomerular vessels are compressed, on the one hand, by the escape of the exudation into the capsules, and by the cell-proliferation proceeding from the walls of the latter; on the other hand, their walls become infiltrated with fluid and swollen, and their calibre greatly diminished. A second factor is the retardation in the current, which, according to Cohnheim, takes place in inflamed tissues; and, lastly, the uriniferous tubules situated in the inflamed interstitial areas must become occluded by the pressure from without, and by the infiltrated and detached epithelial cells.

It is, I think, a less easy and a less simple task, to arrive at an explanation of the processes which take place in those renal affections which are embraced under the term "chronic nephritis" or "chronic form of Bright's disease." This is not the place to enter minutely into the question, so warmly discussed of late, as to whether all those affections included under that designation, are naturally connected and invariably depend on one and the same process, or whether and to what extent they are to be distinguished from each other. It is the less necessary to discuss this question inasmuch as no great difference of opinion prevails on those points in which we are now interested, that is, with regard to the condition of the urine, and to the anatomical appearances in the kidney—the differences in question mainly relating to the

development and succession of the processes, and to the relation which the hypertrophy of the heart, and the vascular changes in general, bear to the renal affection. It is thus almost universally admitted that, from a clinical point of view, there are two well-marked types of chronic kidney disease. The first of these exhibits from the commencement the following characteristics: more or less extensive anasarca, marked anæmia, highly albuminous urine abounding in morphotic constituents (casts, red and white blood-corpuscles, epithelial cells), scanty in quantity when the disease is at its height, or scarcely reaching the normal amount, and, lastly, no manifest hypertrophy of the heart. The second variety commences without anasarca, and generally without any remarkable disturbance of the general health; the urine is limpid, clear, poor in albumen, but excessive in quantity, and these symptoms are usually associated with hypertrophy of the left side of the heart. Every physician who has had only a moderate amount of experience is acquainted with cases of disease which correspond to the one or other of these types—cases, the symptoms of which, without any other disease or any precursory stage, make their appearance exclusively in one or other of these modes, and which run their course for many years, it may be, with variations in intensity until death ensues.[1] In like manner, considered anatomically, two typical forms stand out sharply from the great variety of all

[1] In discussing the question of the development of cardiac hypertrophy in renal diseases, several authorities attach considerable importance to the influence of the prolonged duration of the latter, and the chronic character of their course, and I therefore make the express remark that I have repeatedly seen cases belonging to the former of the above-mentioned types going on for many years without cardiac hypertrophy, but with good general nutrition, if death occurred before the above-described first type became changed; that is, before it passed into that named by myself and others, *secondary* atrophy, a process the development of which may require many years. It is true that in these cases the whole series of symptoms was not uninterruptedly present; the dropsy altogether disappeared at intervals, whereas the albuminuria, and, therefore, the renal lesion as well, were continuous. At the present time there is in my division of the hospital a strong carpenter, admitted for the third time, who a year and nine months ago first fell ill with symptoms of the first type of nephritis, but apparently regained his strength and went on with his work. After being under treatment for several months the dropsical symptoms have now again abated, and the

chronic renal affections; the one described as the large white or mottled kidney (chronic parenchymatous, diffuse nephritis, Weigert's sub-chronic nephritis, &c.), and the other known under the name of the "red granular or contracted kidney" (genuine renal cirrhosis); the former is the more inflammatory form, the chief microscopical characters of which are swelling and fatty degeneration of the epithelium of the uriniferous tubules and accumulation of round cells in the interstices (and likewise in the Malpighian capsules); the latter is more of an indurated character, in consequence of the development of a firm connective tissue with but few cells and tending towards atrophy, and of wasting of the glomerular vessels. I repeat that there are these two forms, standing out clearly by reason of their marked character, but I also lay stress upon the fact that all chronically diseased kidneys by no means correspond exactly to one or the other type, but that numerous cases occupy an intermediate position between them, or approach partly to the one and partly to the other type; and that consequently these two types may be regarded as the terminal extremities of a series in which there are gradual transitions from the one extremity to the other. And this further remark must be made that the first form may even clinically pass by degrees into the second, and inasmuch as we are accustomed to regard the anatomical changes in both cases as of an inflammatory nature, those of the first type being of more recent origin, those of the second of an earlier date and as resulting from the former, we may well suppose that the second type represents, in its anatomy at least, a later stage of the first type, or, at all events, a slower form of development. But the question may well be asked, do all these considerations constitute a sufficient reason for adopting a modern view, which regards all forms as mutually connected in every respect, and promulgates the doctrine of the "unity of chronic nephritis" or of "Bright's disease of the kidney?" If this view be a reasonable one, then no distinction, clinical

patient appears to be in good health. Notwithstanding the long duration of the disease there is not a single symptom of hypertrophy of the heart, and there is no ground whatever for the suggestion of amyloid disease, in which the hypertrophy is absent. On the other hand, it is well known that in the second type the earliest symptom of renal disorder is usually accompanied by cardiac hypertrophy, which can for the most part be clinically demonstrated,

or anatomical, could be drawn between "chronic interstitial pneumonia or cirrhosis of the lung" and other forms of "chronic pneumonia," for these conditions are precisely analagous to those of the chronic affections of the kidney. Among the vast number of forms of chronic pneumonia, there is one which clinically stands out so sharply from all the others, that every experienced physician immediately recognises in it the typical cirrhosis of the lung which, since Corrigan's classical description, has been and still is, unhesitatingly regarded as a peculiar form. And need I say that there are other forms of "chronic pneumonia," whose symptoms differ from this latter type, as widely as day does from night, so that the two cannot be confounded together? or need I say that between these typical forms, placed as they are at each extremity of the large series of chronic pneumonias, there exist an innumerable number of cases representing very gradual stages of transition from the one to the other, in such a manner as to prevent any definition of an exact limit to the series? And is the case different in an anatomical point of view? No anatomist will have any hesitation in recognising a typical "cirrhosis of the lung," set up perhaps as a result of pleuritis or of some disease due to inhalation of irritating dust, and in distinguishing it from other chronic forms of inflammation of the lung, notwithstanding the numerous imperceptible transitions from the latter to the former and *vice versâ*, and in spite of the fact that a really pure interstitial pneumonia as little exists as a pure, exclusively interstitial nephritis. Neither in cirrhosis of the lungs, of the kidneys, or of any other organ is the morbid process confined exclusively to the interstitial tissue, nor in the other chronic inflammations is the so-called parenchyma alone involved and the connective-tissue framework unaffected. It would be difficult to imagine that any such exemptions exist in the living body, in which no system of the elementary tissues is separated from its neighbour by an impenetrable wall. We are scarcely able in all cases of chronic pneumonia even to determine which system forms the starting-point of the disease, and there is just the same difficulty in many cases of chronic nephritis. The important point is to determine the predominant factor in the morbid process, what

it is that impresses special features upon the disease and stamps the anatomical marks upon the affected organ. This, in typical cases of cirrhosis, is represented by a development of fibrillary connective tissue, which forces everything else into the background in consequence of its vast predominance and its results; on the other hand, in the cases which are the farthest removed from this type, the new formation of connective tissue recedes into the background when contrasted with the cellular infiltration and the parenchymatous degeneration of the epithelium. It is not the longer duration and the more tedious progress of the disease which are the primary and principal factors in the production of the cirrhotic type, for, as I have already mentioned, years may pass by without this type becoming developed, while in other cases it makes its appearance after a brief interval. A better opportunity is afforded us in the lungs than in any other organs, of watching the development within a few months of a perfect state of cirrhosis, such as in other cases of chronic pneumonia is not attained after an interval of several years. It is, therefore, not the peculiarity of every chronic inflammation to induce cirrhosis; for there are, for example, suppurations running a very chronic course, but the production of cirrhosis necessitates the antecedent occurrence of something or other that is peculiar, or its supervention in the course of the disease. The nature of this peculiarity does not fall within the scope of our present investigation.

It would, moreover, appear that the diversity in the course of the two types is not dependent upon the renal affection alone. In addition to the fact that dropsy is as constant in the one type as it is rare in the other, the way in which this symptom makes its appearance is so peculiar that the idea suggests itself that some disease of the skin is concerned in its production. It is perfectly evident that the dropsical effusion cannot be referred to suppression or diminution of the urinary secretion, an explanation advanced by some observers. Even the complete interruption of the discharge of urine, which may originate from obstruction in the urinary passages, does not, *per se*, produce dropsy as a consequence. Cohnheim (100) very particularly refers to the fact that neither hydræmia nor hydræmic plethora can be the cause of the dropsy in

those diseases of the kidney. Its proportionately rapid supervention, the great extent of the anasarca, and above all its localisation, would rather seem to indicate the existence of special local abnormalities, which Cohnheim is inclined to look for in some inflammatory change of the skin or its vessels. It is in fact difficult, without some such assumption, to explain how it comes to pass that, in the first form of chronic nephritis (and also in acute nephritis), the eyelids or the scrotum are the usual seats of the œdema, and often those which are first affected; for, as regards these parts, the influence of gravity cannot be a sufficient cause for the appearance of the symptom.[1] How different are the features presented by the genuine renal cirrhosis! Even in this affection a period of dropsy may set in, especially if the heart's action becomes defective and the typical features of the disorder become obliterated. The condition of such patients obviously then resembles that of cases of heart-disease in the stage of defective compensation, and especially with regard to the dropsy, which in these latter depends mainly upon venous congestion. Hence in these cases the localities in which œdema first appears are the lower extremities, and the dependent parts in general; and this œdema is accompanied by more or less intense cyanosis, of which no trace is exhibited by the dropsical patients included in the first category, unless, perhaps, some special causes supervene in addition.

All these diversities serve to explain the dissimilar conditions of the urine in the two typical forms. In the one, the large white (mottled) kidney, the condition which influences the result is the abnormal permeability of all the elements of the tissue, due to the swelling and fatty degeneration of the epithelium, and to the infiltration of the organ by round cells; a condition from which the glomeruli and their capsules are not exempt. Under these circumstances, the transudatory and secretory apparatus must necessarily yield a highly albuminous fluid, in the production of which

[1] In two cases of scarlatinal dropsy without albuminuria I noticed œdema of the face alone in one instance; in the other only œdema of the face and scrotum, persisting for several weeks, in the latter case without the previous appearance of any other trace of dropsy.

the swelling and degeneration of the epithelium, namely, of the capsules, glomeruli, and uriniferous tubules, though not unconcerned in the escape of the albumen (see page 78), have not even been taken into account. It is evident that there will be no lack of blood and pus-corpuscles, epithelial cells and casts, whether these last are formed out of coagulated albumen or epithelium. In like manner it is obvious that the urine must be reduced in quantity in consequence of the pressure which the infiltrated cells exercise upon the capsules and glomeruli, and likewise upon the uriniferous tubules, and in consequence of the more or less decided occlusion which will take place in the last-named structures as a result of the swelling, degeneration, and detachment of the epithelial cells. In this respect the conditions resemble those of acute nephritis, but in the latter the progress of the symptoms is of a more violent character, and corresponding with the acuteness of the inflammation, is marked by intense hyperæmia and considerable hæmorrhage. But even in the chronic form, to which we are now alluding, such acute exacerbations are by no means rare; on the other hand, an improvement may set in, the morbid process coming to an end in certain spots, though complete restoration does not take place. That such changes occur is shown by the varying condition of the urine, and especially by the diminution from time to time of the formed constituents.

We have already stated that it is more than probable that simultaneously with this nephritis some peculiar disorder of the skin sets in, causing it to become œdematous. When this is the case, it is obvious that an improvement may occur, accompanied by absorption of the watery effusion, which is more freely removed by the kidneys, if at the same time the morbid process of which they are the seat undergoes an improvement or its progress becomes arrested. But even in the absence of any such improvement in the state of the kidneys, the absorption of copious effusion would cause a strong current of fluid to set in towards these organs, and any inflammatory products that might be present would be thereby steadily removed. As a matter of fact, we often notice that as œdema disappears, the urine becomes more abundant, but without losing any of its

other characteristics; that is to say, it remains cloudy, contains more or less blood and much albumen, and deposits a copious precipitate of formed elements. Possibly this is to be explained by the fact that the inflammatory processes in the kidney still continue, whereas the condition of the skin becomes improved. The increase in the quantity of urine, which occurs at a certain stage in this form of chronic nephritis, might consequently be regarded as due to the absorption of the dropsical effusion; but the converse could not be assumed, viz. that the disappearance of the latter is due to increased diuresis. This latter process takes place when the form of disease has its starting point in ("secondary") atrophy, which is induced when the development of the fibrillary connective tissue preponderates. The conditions in that case more and more approximate to those of the primary or genuine cirrhosis of the kidney.

In this last-named condition it is not the abnormal permeability of the tissue and of the vessels in particular which is the determining factor, but the increased arterial pressure. We may not assume that the vessels embedded in the close meshes of a fibrous connective tissue, and themselves for the most part thickened and indurated, are to an abnormal extent permeable by albumen; and such a view is also decidedly contravened by the state of cicatricial connective tissue in other parts, *e.g.* the skin, when dropsical effusion exists. The small and the varying amount of albumen found in the urine in cases of renal cirrhosis must, therefore, in my opinion, be referred to the coexistence of small inflammatory areas, the predominance of which is a feature of the former type, but which are not altogether wanting in the disease in question. In cases in which these are absent, or no longer active, the albuminuria, that is, the manifest excretion of albumen, will disappear, and the more so because the quantity of urine will be simultaneously increased and its percentage of albumen consequently still further reduced. It is universally admitted that the increase in the quantity of urine is the result of the enormous increase of pressure, which must occur in the vessels which are still pervious, in consequence of the wide-spread destruction of these parts and the co-existent cardiac hypertrophy. I regard it also as

certain, that in this cirrhosis, in which the large majority of the glomeruli are not seldom destroyed, the increase in the quantity of the urine is in great measure due to the increased activity of the secretory epithelium, a large portion of which is usually in a normal condition. This view is further supported by the fact that the quantity of the specific urinary constituents (at least of the urea), secreted by such kidneys, corresponds absolutely with the conditions connected with the nutrition, though the percentage is reduced. The epithelium in this case acts similarly to all other glandular epithelium when under increased arterial pressure (*conf.*, p. 40).

Disorder of the skin with tendency to œdema, as occurs in the first type, or in acute nephritis, is absent in the typical cirrhosis, although the cutaneous arteries are not seldom thickened just as those in the kidneys (101). This vascular disorder, however, is not such as to cause œdema. It is only when the heart's action becomes feeble that œdema and other effusions set in, and are accompanied by decreased secretion of urine.

I must repeat that the above descriptions correspond only to the marked types of chronic nephritis, to those which stand at the ends of the scale, and that there are numberless cases in which the typical characters are more or less obliterated. To arrive at a proper estimate of these conditions it will always be useful to remember the analogy they present with chronic inflammations of the lung, the mutual relationships of which, however, are more distinct.

Finally, with reference to amyloid degeneration I can be very brief in my remarks, for I have little that is positive to state with regard to the influence of this condition upon the secretion of urine. I agree with others in supposing that the vessels in a state of amyloid degeneration are abnormally permeable, and therefore offer less than the normal obstacle to the escape of the albuminous substances dissolved in the blood. No strict proof can be adduced for that hypothesis, which, however, is perhaps supported by the fact that obstinate diarrhœa is such a common symptom in cases of extensive amyloid degeneration of the intestines. This may, however, be due rather to defective absorption and increased peristaltic action. It

appears to me to be probable that not only the glomerular vessels when in a state of amyloid degeneration, but that the epithelium and the interstitial vascular system as well, when similarly affected, are involved in the excretion of albumen. When examining kidneys in a state of amyloid degeneration I have repeatedly noticed a marked deposit of albumen in the tubuli uriniferi, especially of the cortex, with but very little excretion in the capsules. Posner also incidentally alludes to the same appearances (102). It is, moreover, a well-ascertained fact that pure amyloid degeneration rarely occurs alone, but is far more often associated with interstitial nephritis; the urinary secretion, in such cases, exhibiting corresponding alterations.

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EXPLANATION OF THE ENGRAVING.

Section of rabbit's kidney hardened by boiling and alcohol, coloured by a solution of picro-carmine. The red colouration is not shown in the drawing.

Fig. 1. After a ligature had been kept on the vein for ten minutes. *a*. Deposit of albumen in the uriniferous tubules of the medulla.

Fig. 2. Ligature on one ureter, retained for thirteen minutes. *a*. Extensive lymph-spaces filled with albumen, between the much-dilated tubuli uriniferi. *b*. Œdematous adventitia of an artery.

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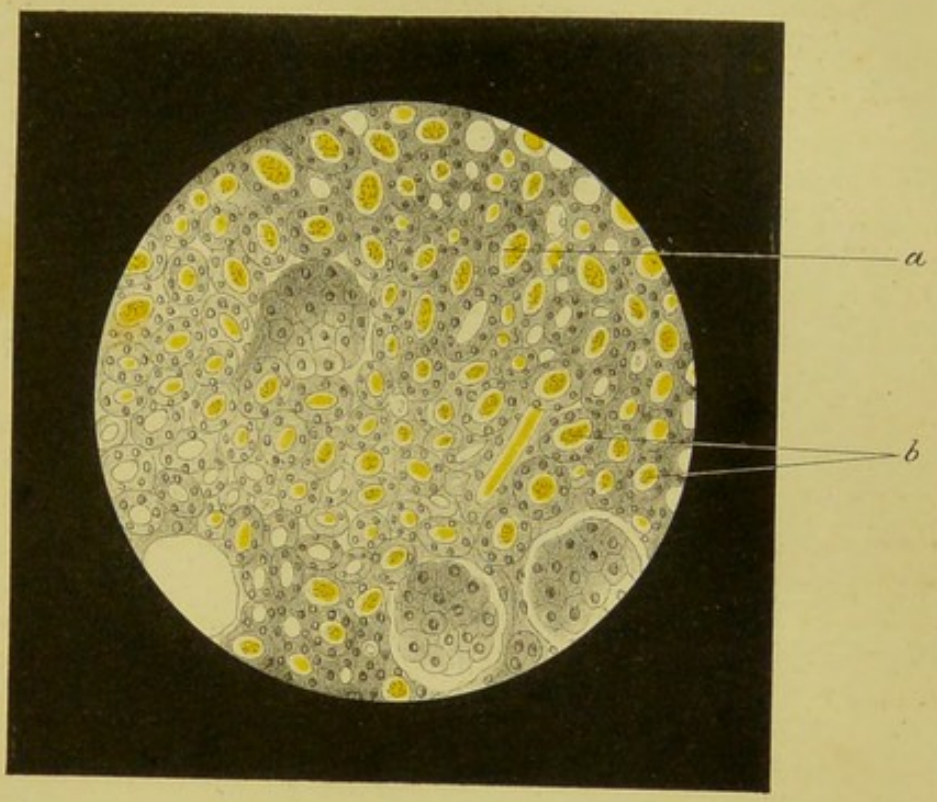

2.

Dr Bachoff del.

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A CONTRIBUTION

TO THE

THEORY OF URINARY SECRETION.[1]

It is well known that there are, at the present time, two opposite theories on the subject of the secretion of urine; one of these may be described as the filtration theory, the other the secretion theory. The former, originated, as we all know, by Ludwig, transfers the entire process of secretion to the Malpighian corpuscles, and assumes that the filtered fluid is subsequently converted into urine in accordance with purely physical laws. I may take it for granted that this view is well known.

In like manner I need not enter upon a minute discussion of the objections to this theory, and which, especially of late, have assumed some prominence. Although these do not all possess an equal amount of demonstrative force, and, from the standpoint of Ludwig's theory, one or other of them may be invalidated (as I shall by-and-bye have some opportunity of showing), it must be admitted that certain facts are irreconcilable with the theory in question. These objections have been recently summed up by Heidenhain,[2] and, so far as they refer to the secretion of the so-called specific constituents of the urine, he has, I think, given a very proper estimate of them. The result is that more weight must again be attached to Bowman's view with regard

[1] A translation of a lecture delivered by Dr. Senator before the Physiological Society of Berlin, December 9th, 1881, and appended to the foregoing essay at the author's request. 'Archiv für Anatomie und Physiologie,' 1882.

[2] Hermann's 'Handbuch der Physiologie,' v. i.

to the secretory function of the epithelium of the uriniferous tubules, and since Heidenhain and his pupils have experimentally demonstrated the correctness of this view, there can scarcely be any doubt as to this function, even apart from the consideration, adduced also by Bowman, that the nature of these cells places them in the category of the glandular epithelia. I consider it, therefore, to be an established fact that the epithelial cells of the convoluted uriniferous tubules, and possibly also to some extent those of Henle's loops, do secrete in a specific manner certain constituents of the urine.

But Heidenhain has lately gone a step beyond this, inasmuch as he regards the excretion of the water (and of the salts, its invariable accompaniment) from the vascular tufts, as an instance, not merely of filtration, but of a real secretion from the epithelium covering the glomeruli. And indeed he believes that the activity of these epithelial cells depends upon their supply of blood—its diminution when the current is retarded being an especial feature—and that their normal activity is also shown in preventing the escape of the serum-albumen. The last remnants of Ludwig's theory would thus be demolished. It seems to me, however, that, on the one hand, this portion of Heidenhain's theory is destitute of a sufficient basis, and is open to numerous objections; and, on the other, that there is no necessity for abandoning the assumption that filtration takes place from the glomerular vessels, whilst the epithelial cells of the tubuli uriniferi are the seat of secretion.

In the first place it appears to me that to regard those epithelial cells as actively secreting agents, that is, as glandular epithelium, involves a very bold assumption, since they differ in their histological characters from all the really glandular epithelium with which we are acquainted. For, after birth these cells are quite flat, and resemble the endothelium of serous membranes or possibly the connective-tissue cells; and we should scarcely be disposed to attribute to these such secretory activity as belongs only to cells very differently constituted, even if it were merely a question of the secretion of water. We are the less inclined to do so, inasmuch as we are acquainted with glands exercising such

a function, but as a matter of fact, exhibiting very differently constituted epithelial cells. The glands in question, which secrete only water and the salts which are its invariable accompaniment, are the lacrymal glands and the sweat-glands. Their glandular epithelium is, however, not to be compared with the epithelium of the vascular tufts. And also in other respects, the condition of these glands is very different from that of the glomeruli. The former are to a certain extent independent of the blood-supply; on the other hand, they are to a large extent influenced by specific irritants and the condition of their innervation, as has recently been demonstrated particularly with regard to the secretion of the sweat-glands.[1] It is a point of special importance that the secretion of sweat continues even when the flow of blood is diminished. With regard to the influence of the blood-supply upon the lacrymal secretion, there are no experimental observations possessing an equal amount of demonstrative force, but all experience supports the view that similar conditions obtain. We know at least that lacrymation is not checked in spite of the greatest reduction in the arterial pressure and notwithstanding the most profound anæmia; the flow, indeed, is rather more copious in these conditions under the influence of the ordinary stimuli. And it may here be mentioned that Cl. Bernard in his experiments upon horses, fully under the toxic influence of curara, noticed that the lacrymal secretion was increased before death—when the blood-supply was assuredly diminished.

The circumstances of the case with regard to the glomeruli are of an entirely different character. Their excretion is dependent exclusively upon the supply of blood, and no stimulus can re-establish it when it has been arrested by reducing the supply. In this respect this excretion differs essentially from that which takes places in the uriniferous tubules. For our knowledge on this point we are indebted to Heidenhain's investigations, and also to those of Nussbaum, Grützner, and others. In animals in which the spinal cord has been divided, and the blood-pressure is consequently so much reduced that no urine is secreted, the secretion

[1] S. Luchsinger in Hermann's 'Handbuch der Physiologie,' v. i.

may be re-established, and even without increasing the blood-pressure, by the employment of various forms of stimuli, but especially by injecting the so-called "specific potential constituents of urine" (*harnfähige Stoffe*). This excretion, however, is solely the result of specific glandular secretion in the uriniferous tubules, as is shown by the fact that it continues after the vascular tufts have been excluded, either by (as in frogs) applying a ligature to their vessels, or by cauterisation.[1] The epithelial cells of the tubuli uriniferi behave like other glands, the activity of which is called forth or increased by adequate stimuli; the vascular tufts, on the other hand, notwithstanding their epithelial covering, do not exhibit a similar capacity. Heidenhain is therefore compelled to assume further that the quantity of water in the blood is the factor which alone determines the activity of these epithelial cells, and is at the same time their adequate stimulus; although he does not overlook the necessity which consequently arises of assigning an exceptional position among all glandular cells to these epithelial structures.[2]

If, then, the assumption of a specific secretion of water by those epithelial cells appears to be open to serious objections, there are also many pathological facts which flatly contradict it, and are evidences against the existence of those peculiar properties which Heidenhain is compelled to ascribe to the cells in question. We know that in renal atrophy a large proportion of the vascular tufts, with their epithelial investment and their capsules, are completely destroyed, and yet the quantity of urine is not diminished, but, on the contrary, very considerably increased. If the filtration theory be accepted, the explanation of all this is very simple, viz. that the pressure is very considerably increased in the vessels which have escaped destruction and in those of the interstitial vascular system; in the first place because a large number of small vessels of the kidney have been destroyed, and in the second, because of the cardiac hypertrophy which is not simply the mechanical consequence of the destruction of the vessels. In this case the attempt might be made to explain the increased secretion, if Heidenhain's view be

[1] See Heidenhain, ibid., pp. 338—340, p. 350, *et seq.*

[2] Ibid., pp. 337-338.

adopted, by supposing that the epithelial cells of the non-affected glomeruli secrete more freely than usual, in consequence of the increased arterial pressure and the increased blood-supply associated therewith. But anyone who has ever seen a kidney of this kind, in which only a few remnants of parenchyma are discoverable, will assuredly come round to the opinion that it is scarcely possible to attribute such an increase of secretion to the few unaffected glomeruli. Nevertheless I admit that this is no cogent objection against Heidenhain's view, and that the explanation referred to, although a highly improbable one, might be admitted in its favour. There is, however, more difficulty in the case of amyloid degeneration of the kidney. In this affection the degenerative process usually involves first and foremost the Malpighian tufts; it is frequently confined to these and to their epithelium, of which nothing is eventually recognisable. At the same time the quantity of urine is not only not diminished, but as a general rule, is increased beyond the normal amount. This fact can scarcely be reconciled with Heidenhain's assumption, but it is a necessary result if we adopt the theory that the Malpighian tufts are the seat of filtration. For in amyloid degeneration, hydræmia, that is to say, an abnormally slight concentration of blood, is always present; and it is well known that *cæteris paribus*, a diluted solution passes more readily through a filter than a concentrated one. In order to explain only the increased quantity of urine excreted, it is not necessary to assume that the vessels in a state of amyloid degeneration are abnormally pervious, an assumption that may do well for the explanation of the albuminuria, and which, moreover, has no connection with Heidenhain's view. In addition to this, amyloid degeneration of the kidneys sometimes occurs without albuminuria. Lastly, we are indebted to Bartels[1] for a clinical observation which completely contradicts that view of Heidenhain's. In the case of a man suffering from thrombosis of the inferior vena cava, but with normal action of the heart, the urine passed was copious in quantity and contained much blood and albumen. In consequence of the thrombosis, there existed a marked congestion in all the

[1] See v. Ziemssen's 'Handbuch der Pathologie,' ix, 1, pp. 39, 177.

rootlets of the inferior vena cava (as evidenced by the intense œdema of the lower half of the body), and consequently a retardation of the blood-current in the kidneys, which, according to Heidenhain, would necessarily impair the function of the epithelium in question, and cause a decrease in the quantity of urine. In this manner indeed, he explains the albuminuria, associated with a diminution in the quantity of urine, which occurs in other kinds of venous congestion in the kidneys.[1] Now, in this case of Bartels', either the nutrition and function of the epithelium were impaired in consequence of the retardation of the blood-current, in which case the excretion of urine must have decreased and not increased; or else they were not injured in spite of the retardation of blood, a supposition, however, which is not compatible with Heidenhain's view. And moreover, they would, if intact, allow no albumen or blood to pass. And if it be supposed that in this case the albumen and blood could be derived not from the vascular tufts, but from the interstitial vascular system, an assumption which Heidenhain himself decidedly opposes, we do not get out of the difficulty; whereas if the filtration theory be accepted, the condition as a consequence of the highly increased pressure, admits of a very simple explanation.

There are, besides, experimental facts which controvert the assumption that the epithelium is capable of secretory activity in the manner suggested; or at least negative the idea that the cells permit albumen to transude only when the blood-supply is diminished or the blood-current is retarded, but not when the supply of blood is increased. As these matters belong to that division of albuminuria which is to some extent pathological, and have already been discussed very briefly in another place,[2] I shall not enter into them here, and will only express the opinion that they can easily be explained if we accept the theory of filtration, and not that of secretion in the tufts.

All alleged objections, some of which have a greater and others a lesser weight, appear to me sufficient as a whole to upset the assumption of a secretory action of the epithe-

[1] Ibid., p. 371.

[2] See pp. 39—77 of the foregoing translation.

lial cells which cover the tufts. Nevertheless there is some justification for the reasons which have caused Heidenhain to give up so completely the filtration theory. So far as they relate to the excretion of the so-called specific constituents of urine, and these latter are regarded as a product of secretion by the epithelium of the uriniferous tubules, and not the result of filtration, there can, as I said before, be no question as to their correctness. It is, in some measure, these same reasons which have induced Heidenhain to allow that the process which takes place in the kidneys in general is one of secretion, and not of filtration, and have, therefore, caused him to place these organs on the same footing with all other glands in which indubitably no mere process of filtration takes place. For, as he very justly says, "as regards all other glands, without exception, we know definitely that the passage of water out of the blood into the secretion-spaces does not depend upon simple filtration."[1] But, according to my judgment, this very comparison with all other glands would necessarily prevent us from expecting that the processes in the kidneys would take the same course. For the kidneys differ in their construction very essentially from all other glands; it is not only by leaving out of consideration the whole system of the Malpighian bodies, with their afferent and efferent vessels, that any general comparison can be instituted between the kidneys and other glands. But as they most assuredly possess that system, we are the more justified in expecting differences in the processes of excretion rather than a conformity with those of other glands. From this point of view, therefore, we have certainly no grounds for refusing to admit that filtration takes place in the vascular tufts. Heidenhain thinks that his view is further supported by the fact that the quantity of urine by no means invariably coincides, that is to say, rises and falls, with the increase and diminution of pressure, as would necessarily be the case if the excretion depended upon filtration. This objection can easily be refuted by the statement that, in point of fact, the kidney is no mere filtration apparatus, but is in some degree a true gland, and that therefore we must not expect that its action will be entirely

[1] Ibid., p 331.

dependent upon pressure. The reason, however, why Heidenhain maintains this plea is that he considers the water of the urine to be derived as a rule only from the Malpighian tufts, and not from the capillaries surrounding the uriniferous tubules, in accordance with the opinion hitherto held with regard to the former structures. However, this assumption also seems to be contrary to all analogy and probability, for no true gland yields a secretion free from water.[1] And that the uriniferous tubules, after removal of the glomerular system, can supply a true fluid urine has been proved by Nussbaum in his experiments on frogs. The potential constituents of urine, which are constantly present in the blood, form, as I have already mentioned, the stimulus to the secretory activity. When the force of the circulation has been diminished after experimental division of the spinal cord, a proportionately greater quantity of only one of these potential urinary constituents is required in order to excite to secretion the epithelium whose function the retarded supply has impaired. The case must obviously be different under normal conditions of circulation. In the first place, the epithelium of the uriniferous tubules, when in a state of normal nutrition, will react to slighter stimuli; for the blood which circulates around these tubules is, as Ludwig urged some years ago, concentrated by the escape of water, and therefore conveys proportionately more urinary materials to the epithelium. There is also this other fact to be considered, viz. that in the present case several urinary constituents (urea, uric-acid-salts, chloride of sodium, &c.) cooperate, and may, under normal conditions, produce an effect identical with that which under abnormal conditions (after division of the spinal cord, &c.) is evoked by a larger quantity of a *single* constituent. It must, therefore, be considered as proved that a portion of the water of the urine, and doubtless the smaller portion, is supplied by the epithelium of the uriniferous tubules, and is a product of true glandular activity. If, therefore, the watery constituent of the urine is not always excreted in such quantity as Ludwig's theory of filtration would lead us to anticipate, the

[1] In the sebaceous glands there is no real secretion, but only proliferation and fatty metamorphosis of the epithelium.

explanation is to be found in the fact that the kidney is a filtrating apparatus only to a partial extent, and that the anomalies are due not to the filtered, but to the secreted elements of the urine.

But even if we admit, just for a moment, that all the water in the urine is derived only from the tufts, yet the facts adduced by Heidenhain with reference to the secretion of water are by no means inconsistent with the filtration theory. These facts are :—1. That after constriction or closure of the renal veins the urine is immediately diminished in quantity (and at the same time becomes albuminous), whereas the pressure in the glomeruli is increased. The usual explanation of this, according to Ludwig, is that when a ligature is applied to a renal vein, the efferent uriniferous tubules are compressed by the turgid veins in the pyramidal portions, and thus the escape of urine is prevented in a purely mechanical manner. Heidenhain does not agree with this explanation, because such a dilatation of the veins requires a certain time for its development, whereas immediately after ligature of the veins the flow of urine almost entirely ceases and is very soon completely arrested, indications which point to the conclusion that the process of excretion is rapidly suspended.[1] Much importance cannot certainly be attached to this objection, for it appears to me very probable that when the escape of blood is prevented, the mechanical obstacle is *immediately* produced, and in the direction from the pyramidal portions backwards towards the glomeruli, and that a few seconds only are required for this to take place. But even if, in this case, the flow of urine ceases with such remarkable rapidity, the fact appears to me to be inconsistent rather than otherwise with the assumption that the excretion has come to a standstill. For in true glands, as, *e.g.* the liver and the salivary glands, the excretion does not cease at the very instant that the blood-current is interrupted, but only in a gradual manner[2]—a fact which is perfectly intelligible.

2. The other point relates to the condition of the urine as regards its quantity when a liquid is conveyed into the blood. On the one hand, for instance, according to Ponfick, a con-

[1] Ibid., p. 325. [2] Heidenhain, loc. cit., pp. 264, 46.

siderable quantity of serum or dog's blood can be injected without any perceptible increase in the excretion of urine; on the other hand, according to Pawlow, the absorption of large quantities of fluids from the stomach produces an augmentation of the urinary excretion without any rise in the blood-pressure.[1] I cannot conceive that these facts constitute any obstacles to the theory of filtration. For, as Heidenhain himself insists, if, in the first case, when fluid is injected into the blood, the pressure does not rise in the manner previously supposed, it follows that there is, *cæteris paribus*, no reason why increased filtration should take place: but as regards the diuretic effect of the introduction of fluids into the stomach, in this case the question of the dilution of the blood has to be taken into consideration, for, as we all know, the filtration is very considerably promoted by the dilution (see above). As a matter of course the dilution produces the same effect, even when it is not the result of absorption from the stomach. Thus, if instead of serum or blood a dilute solution of chloride of sodium be injected into the blood, a considerable increase in the quantity of the urinary excretion would be noticed just as before, and this increase would be unattended by any increase of pressure.

If all these considerations do not directly establish the truth of the filtration theory, at all events they do not contradict it.

But finally, and this is a fundamental point of great importance, Heidenhain thinks that, generally speaking, the capillaries are not endowed with any universal capacity for allowing larger quantities of fluid to pass through their coats when the arterial blood-supply is increased, and that the capillaries of the renal glomeruli, on account of their epithelial covering, must offer more than usual resistance to the filtration pressure. The latter point, *i.e.* the greater resistance of a wall covered with epithelium need not be contested, but surely the statement that transudation from the capillaries is independent of pressure and especially of arterial pressure, is contrary to the laws of filtration. In proof of his assumption, Heidenhain appeals to experiments

[1] Ibid., p. 333.

made in Ludwig's laboratory, with regard to the condition of the lymphatic current under increased arterial pressure. I do not consider this comparison between the flow from an opened lymphatic and transudation from the capillaries to be admissible, for reasons given in the monograph already alluded to,[1] and which, to avoid prolixity, I will not again discuss. It is quite sufficient for me to point out that morphologically as well as chemically, the contents of the lymphatic trunks differ from transudations; that those contents cease to flow under circumstances which undoubtedly have no similar effect upon transudations; and that all the lymph-channels in one portion of the body may be cut off without inducing œdema. The invariable and only cause of œdema is the insufficiency of the veins, or of the veins and lymphatics together, to carry away the transuded fluid. Therefore what is true of the contents of the lymphatic trunks does not apply as a matter of course to transudation from the capillaries. In order to discover how transudations are affected, they themselves must be studied, and so far as this has been done, the result has always proved that augmented pressure is invariably followed by increased transudation from the capillaries. Chabbas, acting under Grünhagen's direction, has found this to be correct with regard to the aqueous humour[2] which most certainly is to be considered as a transudation, and our manifold clinical experience confirms the statement that real transudations (not exudations) rise and fall with corresponding variations of pressure. This statement, however, as a matter of course, does not exclude the possibility that other conditions may co-operate.

And now to recapitulate my observations, I consider myself justified in concluding that there are no reasons whatever for relinquishing the theory that a transudation alone escapes from the glomerular vessels of the kidneys, in accordance with the laws of filtration. Secondly, that a secretion of the specific constituents of urine in a watery (concentrated) solution takes place in the convoluted uriniferous tubules,

[1] 'Die Albuminurie,' &c., pp. 28—30, and the foregoing translation, 29—31.

[2] Dogiel and Jesner's later investigations on this subject refer to the alterations in the quantity of albumen.

and it therefore follows that perfect urine represents a mixture of a transuded with a secreted solution.

Now if the fluid which escapes from the Malpighian tufts be a transudation, it must necessarily contain the constituents common to all transudations—that is to say, besides water there are the materials held in solution in the blood-plasma, in proportion of course to their capacity for filtration, and, therefore, not merely salts, traces of urea, &c., but also albuminous substances (serum-albumin and globulin). For there is no transudation without albumen, and if a fluid containing albuminous matters in solution filters at all through an animal membrane, the albuminous substances will likewise pass through in a quantity corresponding to their capacity for filtration, such capacity being usually slight, the quantity also being subject to variations under different conditions. These conditions are: the quality of the membrane, especially its thickness, the concentration of the solution, the salts it contains, its temperature and the pressure under which filtration goes on. As these conditions undoubtedly vary in different parts of the body, there need be no surprise at the differences as regards quantity of albumen in the transudations. Those normal transudations in particular which are separated from the capillaries by epithelium, such as the aqueous humour, the cerebro-spinal fluid and the endo-lymph, contain so little albumen that they are often set down as "almost non-albuminous." For the same reason the transudation from the glomeruli must likewise be considered as only very slightly albuminous, but certainly not as absolutely free from albumen.

It is well known that the older theories of Küss, v. Wittig and Henle assumed that the glomerular transudation contains albumen. In order to explain the non-appearance of this substance in normal urine, the idea was suggested that the epithelium of the uriniferous tubules withdraws the albumen from the passing fluid. The correctness of this last view has never been thoroughly demonstrated, but just as little has it been effectually controverted. It used to be asserted that degeneration of the epithelium is not followed by albu-

minuria, but this statement is not correct.[1] All that can be maintained against the view in question is, that it scarcely coincides with our ideas as to the function of glandular epithelium to assume that these structures absorb anything from the secretions whose escape is unimpeded, and it is still more extraordinary that albumen alone should be absorbed.

In contrast with this theory, Ludwig, as we know, imagines that the filtrate which escapes from the tufts is free from albumen, but he does not explain why this should be the case, and regards the question of the absence of albumen as unsolved, and one which requires elucidation.[2] It would appear, however, that the sole object of his assumption is to explain the absence of albumen in normal urine. In the meantime, however, the state of the question has become changed. Many recent investigations have shown that small quantities of albumen are to be found in the urine of healthy persons, and that this is not a rare occurrence, but one whose frequency is remarkable, considering the views hitherto received.[3] Physiologists have taken no cognisance of this fact, or have devoted no space to it in their discussions; but treating it with neglect they go on searching for an explanation of the absence of albumen from normal urine, and especially from the fluid which escapes from the Malpighian tufts. The majority of physiologists adduce as an explanation that the normal albuminous substances of the blood-plasma are unable to escape from the vascular tufts, being prevented by the epithelial investment, or the peculiar structure of the walls of the vessels. This assumption is destitute of all analogy; and if it be true that all other capillaries, whether with or without an epithelial covering, are permeable for albumen, it is clearly the less admissible, inasmuch as certain albuminous substances which pass readily through a filter (egg-albumen, peptone, &c.) do undoubtedly pass through the glomeruli. We may, therefore, reply that the assumption is only made *ad hoc*, that is to say, only for a desired purpose—in order to explain the absence of albumen in urine.

[1] See Senator, 'Die Albuminurie,' &c., p. 73 (p. 78 of foregoing translation).

[2] 'Lehrbuch der Physiologie,' ii Aufl., ii, 1861, pp. 428, 429.

[3] See Senator, l. c., p. 15.

But this purpose is defeated, *for as a matter of fact, albumen can be found even in normal urine.* How is this fact to be explained by the aid of that assumption ?[1]

The opinions hitherto maintained with regard to the constituent parts of the glomerular transudation are therefore unfounded. It must be admitted that this transudation does contain albumen, but, as I have already mentioned, the epithelial investment prevents the quantity of albumen from being otherwise than extremely minute. And there is another important circumstance which must cause the quantity of albumen in this transudation to be still less than, for instance, in the so-called "nearly non-albuminous" transudations (the aqueous humour, the cerebro-spinal fluid, the endolymph) ; the fact in question being that this filtrate escapes under much higher pressure. We know that the highest capillary pressure in the body (not excluding that in the intestinal capillaries) prevails in the glomerular vessels. If the filtration pressure rises, then more fluid will pass out from an albuminous solution, but this fluid will be *relatively* poorer in albumen though the absolute quantity may be increased. Consequently, the transudation from the glomeruli will be still poorer in albumen than those above alluded to as containing the smallest proportion of that constituent. But, finally, and in the third place, it must be mentioned that when this transudation reaches the uriniferous tubules, the specific secretion of the latter, which is undoubtedly free from albumen, is added to it ; so that in proportion as this addition is made, perfect urine will finally contain a less percentage of albumen than is to be found in the glomerular transudation.

So far as I can see, this explanation rests upon no arbitrary assumption, but upon recognised physical laws, and it offers us a means of explaining the occasional appearance of albumen in normal urine. For, doubtless, even in the normal condition of things, the pressure in the glomeruli, as well as the quantity of the secretion escaping from the uriniferous tubules, is liable to variations ; and in proportion as the influence of these two factors is increased or lessened, will the urine contain either more or less albumen, the pre-

[1] For additional details see Senator, "Albuminurie," pp. 22—24 (the same pages in the translation).

sence of which will be more or less easily detected. The circumstances, therefore, with regard to albumen are completely analogous to those of many other normal constituents of urine (sugar, oxalic acid, pyrocatechin, &c.), which are found, under physiological conditions, sometimes in infinitesimally small quantities, and sometimes in larger proportion and therefore more easily detected. I refrain from entering into further particulars, inasmuch as in the monograph already alluded to I have discussed at some length other circumstances which, even in the normal state, may conduce to the appearance of large and easily detectible quantities of albumen in the urine, that is to say, may set up a physiological albuminuria. I have also in that treatise explained the influence of changes in the renal circulation and of other pathological conditions.

THE

HYGIENIC TREATMENT OF ALBUMINURIA.[1]

In the treatment of various disorders of the kidneys, and especially of those usually classed together as acute or chronic forms of Bright's disease, albuminuria is the symptom which deserves the closest attention; but not, as is still generally asserted, because a long-continued excretion of albumen impairs the nutrition of the system. This, in my opinion, is a mistaken notion, founded as it is on a highly exaggerated estimate of the loss of albumen such as occurs in most cases even of very serious affections of the kidneys. In a very marked case of albuminuria, the albumen rarely amounts to more than a few tenths per cent., and we know that the percentage generally increases with the diminution in the quantity of urine and diminishes when the latter is increased. If, therefore, the proportion of albumen in the urine be very considerable, say ·5 or ·8, this being an extraordinarily high percentage, the daily amount passed will weigh only a few grammes, perhaps six or eight, or possibly ten as a maximum. A loss of albumen exceeding this amount is altogether exceptional, and, when it occurs, is of very short duration and never lasts for many days or weeks. Losses of albumen to this extent do not appreciably injure an adult, unless his system has been extremely reduced in other ways. Six to eight grammes of albumen represent the quantity contained in forty grammes of beef; and it is evident that if that is all, half-a-pound of meat would almost suffice to cover the

[1] A lecture delivered before the Berlin Medical Society, October 11th, 1882.

weekly loss of albumen, even when very considerable in amount.

The importance of albuminuria, as a symptom, is due rather to the fact that it furnishes us, so to say, with a means whereby the existence and gravity of renal disorders may be determined and estimated. If this symptom were absent, it would not be easy to diagnosticate with certainty a renal affection, and it would be still more difficult to decide that the case was one of Bright's disease (though in this latter albumen may sometimes be absent from the urine) ; moreover in estimating the gravity, the progress, or the retrogression of the disease, we depend, as a general rule, upon the quantity of albumen contained in the urine. Another reason for the importance of albuminuria as a symptom may also be adduced, viz. the possibility that in some cases the excretion of albumen may act as a stimulus to the kidney. We do not, indeed, know that this effect can be produced by those forms of albumen which are found in the urine in cases of ordinary albuminuria, but we do know that egg-albumen acts in this way. When this substance finds its way into the blood, it is excreted by the kidneys, but frequently this is not all that happens, for, as J. C. Lehmann and Stokvis have observed, *more albumen is excreted than is introduced ;* as a matter of course not more egg-albumen, but a form which possesses the properties of the ordinary albuminous substances of the serum (serum-albumin and globulin). It is not improbable that peptone likewise, and perhaps also hemi-albumose (propeptone) may act in a similar way, *i.e.* excite albuminuria. Moreover, the idea has been repeatedly entertained, both in ancient and modern times, that the albumen in ordinary albuminuria, in the various forms of Bright's disease, is not altogether the same with the normal albumen of the serum, and if this view be correct, though its truth has never been demonstrated, it may well be supposed that this albumen also has an irritative action on the kidney, like that of a foreign body or possibly of a poison.

As regards the therapeutics of albuminuria, every unprejudiced physician will confess that up to the present time we have not on the whole been very successful

with our purely medicinal treatment. This is the reason why new remedies are continually being recommended. They are, however, as little efficacious as the old ones. Neither the astringents, tannin in particular, nor fuchsin, nor pilocarpin, nor the acids, produce the effects with which they have been credited, and the same remark applies to nitric acid, lately recommended in accordance with Hansen's prescription forty years ago, but which Frerichs [1] has already found to be useless. There is, at most, one drug, viz. iodide of potassium, which must be allowed to possess a certain amount of efficacy in some forms of chronic nephritis. It must be understood that I am referring now to *the albuminuria*, and not to other symptoms such as the dropsy and the asthmatic attacks, for which we possess really efficacious remedies.

It is easy to be deceived with regard to the usefulness of a remedy employed in cases of *acute renal affection*, for these very often terminate favorably without any medicinal treatment, and the albuminuria in particular, which not unfrequently continues for some time as the last remaining symptom, eventually disappears, unless the circumstances are altogether unfavorable. And in dealing with *chronic renal affections* it is easy to over-estimate the value of a remedy if we forget that their course is usually fluctuating, and that whether medicines are given or not the quantity of albumen in the urine may go on diminishing for days and weeks. Such fluctuations are due to the fact that the inflammatory process in the kidney is not uniform but irregular in its course, and to the occurrence of complications which increase the albumen; while against these may be set the avoidance of injurious influences and the adoption of *suitable hygienic arrangements*.

Since we can do so little with medicines in the treatment of albuminuria, the greatest importance must be attached to hygienic measures, and experience teaches us that the results to be anticipated, if not remarkably brilliant, are far better than those attained by any medicinal treatment whatever in the absence of such measures. That such is the case is proved by the fact that in chronic renal affections

[1] 'Die Bright'sche Nierenkrankheit,' 1851, s. 237.

better results are often obtained in hospitals than outside these institutions. For ordinary hospital patients are under much more favorable hygienic conditions than are found outside, and I have not unfrequently seen patients improve in hospital either without medicine, or under the same treatment to which they had for a long time been subjected, and without benefit, as out-patients.

The most important question is that which refers to the *diet*. Some amount of attention has been given to this subject, but it appears to me that the indications have not been sufficiently considered, nor the full purport of the question properly appreciated. In discussing questions as to the dieting of patients suffering from nephritis, there are two points to be considered:—(1) The influence which the *process of digestion per se* has upon the albuminuria, and (2) the influence which the *kind of food* has upon the symptom in question. With regard to the first, recent observations have shown that during digestion the excretion of albumen is augmented in patients suffering from albuminuria, or that the symptom recurs when temporarily in abeyance; and a transient condition of albuminuria has indeed been noticed in healthy persons during the digestion of a full meal. Hence the rule that *in albuminuria the demands of the system for food are to be satisfied by small quantities frequently repeated, and not by abundant supplies.*

The choice of the food, however, is a still more important consideration. Physicians long ago noticed that the albumen was increased after the ingestion of certain articles of food and drink; but it is only recently that these observations have been tolerably well understood and added to by others. The fact has been demonstrated by the experiments, already alluded to, of J. C. Lehmann and Stokvis, and it must be further observed that the injurious effect of egg-albumen was witnessed not only when this was injected into the blood, but when introduced in large quantities into the stomach—a fact noticed by other observers besides those above mentioned.

It would therefore appear that egg-albumen acts injuriously not only when it is introduced as such into the blood, but also after it has undergone alterations in the stomach and

bowels. At any rate *it is advisable strictly to forbid the use of eggs whenever albuminuria exists.*

With regard also to other albuminous articles of food, and especially meat, if we wish to get rid of the albuminuria, we must limit the supply within much narrower bounds than physicians now prescribe, guided as they are by what I have already alluded to as an exaggerated estimate of the loss of albumen. I have repeatedly noticed the occurrence of albuminuria in apparently healthy men after eating heartily of meat, and still more frequently observed increased amount of albumen in cases of albuminuria. Similar observations with regard to cheese have been recorded by Christison.

It is unnecessary now for me to enter into an explanation with regard to the injurious effects of highly albuminous food, for a year ago I had the honour to address you on the subject of the pathogenesis of albuminuria, and I then discussed this question.[1] I will just add that Lichtheim[2] has recently pointed out another danger attendant upon a full albuminous diet, viz. the possibility of an accumulation of urea in the blood and its accompanying results (uræmia), as a consequence of the abundant formation of urea with co-existent disease of the kidneys ; and this warning holds good not only of urea but of other final products of the disintegration of albumen, especially of phosphoric acid, the excretion of which, according to Fleischer's investigations, is likewise impeded in renal affections.

We must therefore adopt the rule of limiting the amount of meat, and if it cannot be altogether withheld, we must recommend those kinds which contain less albumen, such as veal and young fowls, *i.e.* the so-called "white meat." For the same reason fish, which also contains less albumen than beef, for example, may be more safely recommended for such patients.

After what has been already said, I need not further explain why the use of *vegetable food* may be freely permitted. Green vegetables, salads, cabbages, and the like,

1 *Vide* 'Verhandlungen der Berliner med. Ges.,' xiii, 1881-82, and Senator's foregoing treatise 'Albuminuria in Health and Disease,' Berlin, 1882, pp. 19, 54, 94, *et seq.*

2 'Corresp.-Bl. der Schweitzer Aerzte,' 1882, No. 7.

which are poor in albumen, are preferable to those which contain more of this constituent, and especially to the leguminous kinds.

As a matter of course, the choice of the several articles of diet will depend upon the state of the digestion and other individual conditions. And especially with reference to the question as to whether *fat* in large quantities is to be allowed or not—a very important point as regards nutrition when the supply of albumen is limited—the answer to this will depend simply upon the condition of the organs of digestion.

With regard to *liquids*, alcoholic drinks have been hitherto forbidden for purely empirical reasons, based, however, upon clinical experience to the effect that renal diseases are caused by the misuse of alcohol, and especially by the immoderate consumption of brandy. Quite recently it has been proved by Pentzoldt's [1] experiments that inflammatory renal affections can be produced in dogs by administering ethyl- and amyl-alcohol. The patients must therefore be confined to water, effervescing drinks, and the various alkaline acidulous waters; but it will be difficult altogether to forbid alcohol, especially to patients accustomed to its use. Spirits, Cognac, &c., must be forbidden under all conditions unless their employment be necessitated by special indications, *e.g.* collapse, &c. The prohibition need not be so strict with regard to wine. I have hitherto adopted the rule of allowing claret, more from habit than for any special reasons. Some importance has been attached to the amount of tannin contained in claret, but the quantity is too small to be of any consequence, and, as before observed, I am unable to attribute any great efficacy to tannin as a remedy. Beer is generally considered to be more injurious than wine, although the strongest kinds of beer contain less alcohol than the majority of wines. But beer appears to me to act more injuriously than wine in cases of albuminuria. Only in one case of so-called albuminuria with healthy kidneys, occurring in a colleague, the patient stated that wine invariably caused a reappearance or increase of albumen, whereas no such effect was observed after drinking beer.

[1] 'Sitzgsb. d. phys. med. Soc. zu Erlangen,' 1882, Juni 19.

In the last place experience teaches that hot spices and highly cured meats are hurtful; *errors of diet* of every kind always do mischief. I have had repeated opportunities of observing cases in which the albuminuria was undoubtedly increased after grave errors in diet.

It is a fact of the greatest interest that the dietetic treatment of albuminuria, the so-called "milk-cure" which was long ago adopted and still maintains its deserved reputation, is in perfect harmony with the principles laid down in the foregoing remarks. It is true that a *diet consisting exclusively of milk*, though very often talked about, can but rarely be persevered with for a lengthened period, and the expression, therefore, is not to be taken quite literally. If, for example, a patient takes two litres of milk *per diem*, a very considerable quantity, he does not obtain even the minimum amount of albumen which an adult requires to support life. Voit[1] states that an adult prisoner, doing nothing and receiving only the so-called "sustentation-diet," requires a daily supply of 85 grammes of albumen, 30 grammes of fat and 300 grammes of hydrocarbons, and that even for old women, the objects of charity and living under very miserable conditions, the amount of albumen must not be reduced below 76—80 grammes. If, however, we take the average percentage in milk as 3·41, two litres will contain only 68 to 70 grammes, or considerably less than the quantity required to support life. The fat contained will amount to 74 grammes or more than twice the necessary quantity; the hydrocarbons again, not quite 100 grammes, will be much below the standard. This latter deficiency will, however, be partly made good by the excess of fat which can supply the place of the hydrocarbons. A further supply of the latter is required to cover the deficiency. I have already said that according to my experience, milk alone is scarcely ever taken or tolerated, at least for more than a few days, but patients can and will take it if hydrocarbons, especially in the form of a few hundred grammes of white bread, be added, and if, instead of pure milk, we give them milk-gruel made with flour, oatmeal, &c. A "milk diet" of this kind can easily be continued for many weeks with very good results, as

[1] "Physiol. des Stoffwechsels," &c., in Hermann's 'Hdb.,' vi, 1, 1881.

it appears to me, corresponding as it does to the recommendations I have made with regard to the diet for cases of albuminuria.

I append a few remarks with regard to the employment of *mineral waters*, though these belong as much to the medicinal as to hygienic treatment. Some good results are often obtained from the external and internal use of these waters, but we are not always able to explain the manner in which the effects are produced. Experience teaches us that for drinking purposes, *the saline*, or the *alkaline-saline waters* produce the most favorable results, and for certain special peculiarities connected with each case, which I cannot now discuss, either a cold or a warm spring may be selected. In accordance with ordinary notions, we should expect that these waters would act injuriously rather than otherwise, inasmuch as saline substances are considered to be renal irritants, and the general rule is sedulously to avoid all irritants when dealing with inflamed organs. It therefore appears to me that the effect must be explained in another manner. It may be that these waters have a beneficial effect upon the digestion, and subsequently upon the blood, either by producing quantitative changes in the composition of the latter, or by altering its quality, especially in regard to its albuminous constituents. On these latter points we have, however, no certain knowledge. In England the opinion has for a long time prevailed that a faulty assimilation and condition of the albumen are the cause of albuminuria, and that the latter is to be treated by improving the digestion and assimilation. But as this idea is based rather upon speculation than upon positive proofs, it has, generally speaking, gained no acceptance among us. We must not, however, in my opinion thus absolutely refuse to entertain the idea of a faulty condition of the blood as a cause of albuminuria, and from this point of view the internal use of certain waters is the more intelligible. With regard to treatment by a *course of baths*, the effect of these in promoting tissue-changes is unquestionable, and they may possibly so far have a good effect upon the albuminuria, but their purely local action upon *the skin* is a matter of still greater importance.

And this brings me to the second portion of the hygienic treatment, viz. to *the attention to the functions of the skin.* In the treatment of renal affections this point has always been regarded as more important even than the diet, doubtless from a consideration of the fact that these disorders may result from taking cold and from other injurious influences acting upon the skin, *e.g.* burns and certain irritating applications which produce inflammation. Moreover, the fact that, in some respects, the skin and kidneys act vicariously for each other, has led to the practice of exciting the activity of the cutaneous functions in treating renal disorders, with the object, as we say, of relieving the kidneys. We can only suppose that with increased activity of the skin, the kidneys have to excrete less water and a smaller quantity of solid constituents, since their blood-supply is reduced. I will not attempt to decide whether these causes alone are sufficient to produce such results. The fact is that, when a considerable quantity of water passes off through the skin, and the urine in consequence is very much diminished in quantity, the albumen is *absolutely* reduced, but perhaps *relatively* increased. It may be that a portion of the albumen passes off through the skin, for this substance is found in the cutaneous perspiration (Leube) though in very minute quantity ; or perhaps some proportion of the albumen, which otherwise would have escaped with the urine, remains in the system.

It is, therefore, advisable that the skin should be constantly kept in a more or less active state of perspiration, and thus we find a reason for the good effects of a course of bathing, not that any good results while the bath is being taken, for much less water is then being excreted by the skin, but because repeated warm baths, even when the water is pure, increase the turgescence of the cutaneous vessels. But a prolonged use of saline baths causes this turgescence to be still more protracted, because the tissue of the skin becomes impregnated with the saline constituents, and these, which are only gradually removed and remain for some time after the course of bathing is ended, have an irritating action upon the skin and keep up a certain amount of congestion.

It follows from what has been said, that the patients ought to take the greatest care to avoid chills, and that they should

wear woollen under-clothing. Our object is best attained by keeping the patients in bed for as long a period as is possible, for weeks, and if necessary for months; the adoption of this plan keeps the skin under the most favourable conditions for promoting its function. It involves also another point, which is an important one as regards treatment, viz. *it prevents as far as possible muscular movement.*

Observations have repeatedly proved, as I stated in my essay of last year, that active muscular exertion increases the albuminuria. The obvious cause of this increase I then explained, and I need not now recur to it. The patients must, therefore, be prevented from taking excessive exercise, and even when we wish to give them the benefit of fresh air, which is highly desirable owing to the anæmia characteristic of renal affections, all active exercise must as far as possible be avoided, and the patients must not walk or run or ascend heights, &c., but confine themselves to driving about, under proper precautions against chills. But otherwise it will be best to confine the patients to their beds, where so many favorable conditions exist in combination.

In addition to the three above-mentioned points with regard to the diet, the cutaneous function and muscular repose, there are a few other conditions which influence the course of albuminuria, and which are, therefore, deserving of consideration; among these are *psychical influences*. It is probable that some of you have noticed that sudden affections of the mind, such as anger, fear, &c., increase the albumen in the urine. Several very striking instances of this kind have come under my own observation. The rule must, therefore, be adopted of guarding the patients as much as possible from such influences, and the best way to do this is to enforce a prolonged withdrawal from business and other avocations.

In the last place I have observed that the albumen is regularly increased in female patients during *menstruation*. I need scarcely say that this observation refers to perfectly pure urine, and not to such as contains any admixture of menstrual blood. It may be that the pelvic congestion which occurs in menstruation, causes an increase in any inflammatory processes which exist in the kidneys, and thus augments the albumen; or perhaps the various systemic processes,

the changes in the metamorphoses of the tissues, which certainly take place in menstruation, affect the symptom in question. It is a well-established rule that the patients, at these periods, must be even more carefully and strictly watched than at other times, and kept absolutely to their beds, in cases in which some relaxation of this rule has been allowed for a greater or less portion of the day.

In taking as far as possible a full and complete survey of all the points which seem to be of importance in the treatment of albuminuria, you will easily have gathered from what I have said that a *suitable climate* is preeminently indicated. For this includes a combination of all possible and desirable conditions for producing a favorable effect. The improvement or cure which may result from baths and changes of climate is due, not to any single factor, not merely to the water and the air, but to a variety of other conditions which have been already indicated.

Southern and dry localities, such as Gries and Meran, are to be recommended for our purpose, and better than these are various spots on the Riviera, especially Bordighera, Cannes, &c., but the best of all is Cairo and Egypt generally, which, however, owing to political circumstances, is not very inviting just at present. A lengthened sojourn in such places is decidedly advantageous in consequence of the effects produced upon the cutaneous functions and the removal of the patient from all unfavorable mental impressions. Active exercise also becomes unnecessary, and, lastly, the diet, which in southern latitudes contains more vegetables and less animal and albuminous substances, fulfils the indications above detailed.

In the case of poor patients, *admission into a hospital*, though not to be compared with a suitable climate, has in some respects a similar effect, as I mentioned in my introduction, and it is for these patients a kind of substitute for change of climate. In the wards of a hospital they are at any rate quieter and more sheltered than they could otherwise be, and the greater part of the day or even the whole day can be spent in bed.

I cannot conclude this address without uttering an impressive warning against too sanguine expectations with regard

to the results of the above methods in dealing with *chronic* albuminuria. Astonishing and brilliant cures are just as little to be obtained by these as by any other plans. With a certain amount of perseverance we can sometimes succeed in considerably reducing the amount of albumen, and even in causing the symptom to disappear for a longer or shorter interval—a change which some may proclaim as a cure. We must for the present be content with these modest results in a domain which presents so many difficulties to therapeutics.

NOTE.—The proof-sheets of this translation of Dr. Senator's essays have been revised by the author, whose kindness and courtesy the translator desires gratefully to acknowledge.

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SOME CONSIDERATIONS ON THE NATURE AND PATHOLOGY

OF

TYPHUS AND TYPHOID FEVER,

APPLIED TO

THE SOLUTION OF THE QUESTION OF THE IDENTITY OR NON-IDENTITY OF THE TWO DISEASES.

BY

THE LATE

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(*Read before the Parisian Medical Society on the* 16*th and* 23*rd April,* 1840.)

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ON THE NATURE AND PATHOLOGY

OF

TYPHUS AND TYPHOID FEVER.[1]

It is my object, in the following pages, to attempt, by a reference to facts, the elucidation of a question, which is confessedly surrounded with difficulties, and has called into the field of controversy some of the most gifted men of the present day,—some of them espousing one opinion, some another, while others still suspend their judgment, till the production of new facts and reasonings shall have dissipated, in part at least, the darkness in which the subject is involved. It is, indeed, not a little remarkable that, in a country like Great Britain, where typhus sacrifices yearly its thousands of victims,—where it is all but universally admitted that the local lesion of the intestinal follicles is not a characteristic sign of the disease,—and where, on that very ground, the doctrines of Broussais have never been able to establish themselves, there should still be wanting any standard work upon the nature, symptoms, and necroscopic appearances of typhus, such as those of Chomel, Louis, and others, who, on this side the Channel, have achieved the triumph of pathological science in a class of diseases of which the anatomical lesions were so long unknown. Hence the uncertainty that prevails in France in regard to the question I

[1] This paper is reprinted from a copy in the library of the Royal Medical and Chirurgical Society, which contains many marginal corrections in the handwriting of the author.

have chosen for consideration—a question which, beyond a doubt, has been for a dozen years, and is still, exciting an interest here, to which British medical men are comparatively strangers. Hence the tours of inspection undertaken from time to time by distinguished continental physicians, with a view either to weaken the conclusions that may be drawn from facts already published, or to dispel a scepticism which these facts, so limited in number, have not been able to overcome. Hence also the objections naturally enough raised to the information collected by them, on the ground of defective opportunities, hurried observation, and imperfect knowledge. Hence, in short, such reflections on the doubtful character of the information received from English authors, as are made by Chomel in his 'Lectures on Typhoid Fever,' those of MM. Gauthier de Claubry and Montault, in their prize essays on the 'Analogies and Differences between Typhus and Typhoid Fevers,' and the following words of Valleix,[1] in apology for citing American instead of English authors, in reference to fevers prevalent in Great Britain. "We admit that it would have been better to have presented a series of observations on typhus made in England itself; but where can we find them?"

These observations will derive great additional weight from a brief consideration of the work of Dr. Tweedie, entitled 'Clinical Illustrations of Fever,' which is referred to by most of the French writers on the subject, and whence a great part of their ideas on English typhus is drawn. I confess I have been puzzled, in perusing the above-named treatise, to find out what definite meaning the distinguished author attaches to the word "fever;" whether he understands by it a symptom produced by a local affection, or a distinct disease arising from a specific cause. His valuable remarks upon contagion, and his strictures upon the doctrines of Broussais, would lead to the latter, while the cases detailed give countenance to the former supposition. I cannot better or more shortly give an idea of the cause of my difficulty, than by giving an analysis of the cases referred to.

Of 73 patients who died, 54 were inspected. Of the latter

1 'Arch. de Médecine,' 3rd series, vol. iv, p. 216.

number, 10 seem referable to typhus, and 17 to typhoid fever. Of the remaining 27, I find that 4 died of well-marked and extensive meningitis, 1 of phrenitis, 2 of erysipelas of the head and neck, 5 of phthisis, 4 of pleuro-pneumonia, 3 of pneumonia, 2 of pleuro-pericarditis, 1 of pleuritis, 1 of pulmonary apoplexy, 1 of abscess of lung, 4 of peritonitis. Two of the above cases were sequelæ of scarlatina and smallpox. Of those cases which were not inspected, there are, as far as can be gathered from the brief account of symptoms, 5 of typhus, 3 of typhoid fever, 3 of meningitis, 2 of pneumonia, and 1 of phthisis. Of several, it is stated that they were evidently cases of purely head, chest, or abdominal affections, and in one, which was proved by dissection to depend on intussusception and gangrene of a considerable portion of gut, it is remarked, that "the symptoms were referable to some obstruction in the intestines, rather than to any form of fever." In the above summary, I have been careful to exclude those that were evidently, or appeared to be *sequelæ* of continued fevers, and to include those only in which the feverish symptoms clearly depended, throughout their course, on local affections. Deducting, however, those I have alluded to, and which form rather more than the half of the cases detailed, there still remain 35, more or less clearly marked out as belonging to two kinds of specific fevers, the one differing from the other in the extraordinary gravity of its symptoms, the rapidity of its course, and the absence of local lesions.

This leads me to remark, in concluding these preliminary observations, that when I began, in 1836, the practical study of fever, I was much struck with the simultaneous occurrence, in the wards of the Glasgow Fever Hospital, of two sets of cases, in which the symptoms (however little most of them might seem to differ, when viewed individually) presented, when taken collectively, characters so marked as to defy misconception, and to enable the observer to form, with the utmost precision, the diagnosis of the nature of the disease, and the lesions to be revealed by dissection. More particularly, it was remarkable to observe that while, in the one disease, the affection, in those who presented no erup-

tion, was so slight and of so short duration, as to make it very questionable whether it deserved the name of typhus, and while the fatal cases presented an abundant and generally a profuse eruption; those labouring under the other, which equally, and even in a much higher proportion, went on to a fatal termination, rarely presented any, and then only a very scanty eruption. It was further remarkable, that while in the one several successive patients had either been restored to health, or fallen victims to the severity of the affection, the disease, under which those laboured, who lay side by side with them, though characterised by much less urgent symptoms, pursued its gradual course through weeks and months consecutively, and in the majority of cases to a fatal issue. And finally, it was more remarkable still, that, to complete the contrast already so striking, dissection proved the existence, in the one disease, of most extensive local lesions, in the other, the absence of all prominent local lesion whatsoever.

Another circumstance, I must not forget to mention, viz. that though, during the summer and autumn of 1836, the cases of typhoid fever were numerous; from the month of November in that year (at which time both the type and the amount of typhus became much more formidable), till June, 1838, the period that my connection with the hospital ceased, not more than a dozen cases—if there were even so many, and these at long intervals, were admitted for treatment. In speaking of the two varieties of fever as totally different diseases, before going into the proof of the assertion which is the point in dispute, I have merely employed a phraseology in common use, of which even those who maintain the complete identity of the affections have not been able to rid themselves, and whereon, if I were disposed to insist upon words, I might found a strong argument in support of my position; and have expressed a conviction forced upon me by the consideration of facts and reasonings, which I now proceed to unfold.

It is far from my intention, in this paper, to take up in all its bearings the vast subject of the "analogies and differences between typhus and typhoid fever." That task I hope to be able, along with my friend and fellow-worker,

Dr. Anderson, to undertake hereafter. My present object is to develop, as shortly and as clearly as I can, some of their leading features of difference, in support of the position I have taken up. I propose, then, to consider:—1. The probable origin of the two affections. 2. Their proximate causes. 3. Their course. 4. Some of their symptoms. 5. Some of their anatomical lesions. 6. Their treatment.

I. Without entering upon the abstruse question of infectious germs, or discussing theoretically the probability of the opinion that ascribes the origin of continued fever to animal, as intermittents are known to proceed from vegetable miasmata, I proceed at once to examine the question in a practical point of view. Dr. Copland, in his able article upon infection, places exanthematic typhus under the third class, viz. that of "specific infections," characterised as "immediately or mediately disseminating and perpetuating their kinds," &c., and directly refers that form of typhus to "emanations from the secretions, excretions, and surfaces of persons already affected." Under the second class, viz. of "conditionally perpetuating or contaminating infections," he places "animal effluvia," or more particularly, "emanations from living bodies in close and unventilated situations." Among the "diseases resulting therefrom," stand "adynamic, putro-adynamic, and malignant fevers." Now, in this classification, it is plain that while we are informed of the origin of the latter, we are only told the mode of propagation of the former. Exanthematic typhus is said to be the result of "emanations from persons already affected;" but whence the original disease? In reference to this question, I observe, that Dr. Roupell[1] had given the weight of his name to the opinion that exanthematic typhus does not originate from vitiated air, extreme filth, closeness, &c. I cannot but express my surprise at this opinion, as the amount of evidence against it seems quite overwhelming.

On no two points (and so far I must anticipate my remarks on the eruption in typhus) are the distinguished authors who describe the frightful epidemics of army typhus during the last six centuries more completely agreed, than

[1] 'Roupell's Treatise, &c.,' p. 64, quoted at p. 107, vol. xxxi of 'Med.-Chirurg. Review.'

on the influence of impure air in the production of the disease, and the very frequent presence of the rubeoloid exanthema in those affected with it. Dr. Tweedie, in his interesting observations on the 'Influence of Impure Air on the Generation of Typhus,' refers to the opinion of Sir John Pringle, who states, "that he has observed the hospitals of an army, not only when crowded with sick, but at any time when the air is confined, and especially in hot weather, produce fever of a peculiar kind, which is often mortal; and he remarked, that the same thing arose in full and crowded barracks, and in transport ships, when filled beyond a due number, and detained long by contrary winds, or when the men had been long kept at sea under close hatches in stormy weather." He also refers to the experience of Mr. Pearson, who, he says, "told me, that when he was surgeon of the Lock Hospital, he uniformly observed, when more than a certain number of patients were placed in any of the wards, fever became prevalent in the establishment; and that, from repeated observation of this fact, he was induced to restrict the number of beds in each ward, and never afterwards witnessed the recurrence of fever in the house." It appears to me that the instance cited by Dr. Roupell (at p. 54 of his work) of fever coming on, in the Refuge, open in 1830-31, on the north side of the Thames, at a time when the wards were crowded to excess, might alone suffice to cast strong doubts on the correctness of his opinion.

If we consult the details given from p. 7 to p. 42 of Gauthier de Claubry's elaborate treatise, of various epidemics of typhus, described by such men as Pringle, Roux, Hufeland, Ducastaing, Reveillé Parise, Tresat, Tort, Gilles de la Tourette, Laurent, Ardy, Magnin, &c., and his summary from p. 100 to p. 114, we find the unanimous voice referring the generation of the disease to vitiated air, arising from the crowded state of the hospitals, the carnage among troops and inhabitants, and the want of burial; the scarcity of provisions, depressing passions, exposure to cold and fatigue are likewise more or less frequently mentioned; but we are entitled to regard these as merely accessary; for we have already seen the development of typhus where these conditions were wanting. Montault, from an extensive induction of facts,

likewise comes to the same conclusions; Copland,[1] expressly points out the influence of the circumstance in question, in producing "highly infectious and typhoid forms of fever."

How, indeed, is it possible, except on this supposition, to explain the appearance of typhus in a country, and in the midst of armies, perfectly healthy, till the development of the conditions in question, but which, under their influence, were desolated by a pestilence, whose ravages, more frightful than those of war, consigned many, whom the sword had spared, to "an ever-yawning and never-satisfied grave?" We cannot, in order to account for this, admit the idea, that, by some strange and inexplicable principle of election, infectious miasmata direct their course from distant shores to the seat of war, especially when we see in their invariable antecedence and sequence, the relation of cause and effect so clearly made out between the conditions referred to, and the appearance of the disease. What reasonable cause can be assigned for the murderous epidemic that arose, in the spring of 1810, on board the Plymouth prison-ships, and cannot be referred to contagion or infection, as the disease first declared itself among the prisoners confined in what have been well termed "these floating tombs," if not the very natural one of the long-continued confinement of hundreds of persons, in a situation uniting all the conditions most adverse to health? These considerations are borne out by the undisputed fact, that the type of fever becomes much more malignant in hospitals crowded to excess, and by Gauthier de Claubry's curious statements at p. 148 of his work, where, speaking of the treatment of typhus, he mentions that, during "the wars of the empire," patients previously despaired of, notwithstanding the assiduous use of therapeutic agents, were quickly restored to health, on the evacuation of the hospitals, with fresh air, barley-water, and lemonade. How completely does this agree with the experience of Pringle, as quoted above. Let me further refer to a paper by Dr. Peebles, in the forty-fourth volume (No. 125) of the 'Edin. Med. and Surg. Journal,' as detailing the results of his own experience in Italy, and giving a

[1] Art. "Infection," p. 352.

remarkably clear, succinct, and comprehensive digest of the mass of evidence bearing on this interesting subject. Dr. Alison's statements in his recent pamphlet on the Scottish Poor Laws fully confirm the general opinion. I have only to add, in again partly anticipating details, to which I shall presently advert, that 22 of 30 patients who positively declared to me that they had not in any way been exposed to infection, replied as follows to my inquiries respecting the number who lived together. In three cases there were 4; in seven 5; in two 6; in three 7; in two 8; in one 9; in one 10; in one 12; in one 13. Many of those, who traced their affection clearly to contagion, stated the number of those who lived together as 12 and 13, and in one or two instances, as "16 at least." This, be it recollected, in one, or at most two small rooms, in the most miserable, darkest, and worst aired purlieus of a large manufacturing city. After the facts I have adduced, I think we may safely conclude, that "animal effluvia," to use the term of Dr. Copland, or "emanations from living bodies in close and unventilated situations," possess the property of generating the specific poison, whatever be its nature, that gives rise to exanthematic typhus.

With regard to the producing cause of typhoid fever, all is vague and uncertain. Chomel, after his analysis of the causes assigned by patients, remarks (p. 306), "Thus, scarcely, out of a number of patients so considerable (115), is there one in four who attributes his state to some slightly energetic cause." But what chiefly concerns us to know is, whether the conditions we have seen to be so powerful in the production of typhus are the same, or anything like the same, in regard to typhoid fever. Exactly on this point, Louis remarks,[1] "No more can the dwelling in places, low, and inhabited during night by too great a number of individuals, figure among the causes in question, one eighteenth only of the patients being in that condition;" and he concludes, from a comparison of all the commonly assigned causes with the facts ascertained by himself, in these remarkable words: "The deepest darkness then prevails regarding the causes of the affection under consideration." Chomel

[1] Vol. ii, p. 457.

likewise (p. 300) uses almost the same words in beginning his third article, "The causes of typhoid fever are wrapt in the greatest obscurity." It were difficult to find a more striking instance of the little effect of the circumstances we have mentioned, in producing typhoid fever, than the epidemic at Bischofsheim, in the department of the Lower Rhine, in August, September, and October, 1832, described by Ruef in the 'Gazette Medicale' for 1834, and referred to by Gauthier de Claubry, who maintains the identity of that disease and typhus. (P. 133, *et seq.* of his essay.) The disease first showed itself, without suspicion of being transmitted, in the upper, best aired, and, as is expressly said, "the most healthy part of the village," which is situated partly on and partly at the foot of a rising ground, and spread successively to the middle and lower quarters. Again, the result of Lombard's researches, conducted at Geneva throughout several successive years, is, that the cases of typhoid fever are numerous during the autumn, and comparatively few during the winter months; whereas the ravages of typhus, as everyone knows, are most formidable during winter.

II. I come now to consider the proximate causes of the two diseases. To avoid all ambiguity in the use of the terms, infection and contagion, which have caused, perhaps, a greater war of words than any two terms in any science, I shall merely state that, with Dr. Copland, I take infection in its classical sense, and understand by it the imparting of any taint, pollution, or contamination whatsoever, to a person previously in health. By contagion, I understand a mode of infection, viz. infection by contact. As applied to the subject of fevers, it will at once be seen that infection has not merely a reference to disease, but to miasmata, or specific poisons, independent of disease, to the contaminating agent, as well as the individual contaminated, and communicating disease. In this point of view, the question is set at rest by the remarks offered under the first head. Hence, we consider typhus as a truly and eminently infectious disease. The same, we have seen, cannot be said of typhoid fever. The question, then, we have now to consider, narrows itself into one of transmission or propagation from a diseased to a

healthy person. The difference, in this point of view, between infection and contagion is one of degree, and not of kind; and there are cases of morbid impression, which it would be very difficult, if not impossible, to refer either to the one or to the other, so imperceptibly do they glide into each other. It is natural, however, to conclude that, as the genus includes the species, so a disease that is first produced by the action of a specific infectious agent on the animal economy, must be directly transmissible from a person diseased to a person in health. To argue at any length in favour of the transmission of typhus, whether through the medium of the atmosphere, or by the direct inhalation of the morbific matter from a persom labouring under its effects, would be a waste of time. It is admitted all but universally. The fearful rapidity with which it extends its ravages; the great number of cases in which patients refer their complaint to living with, or attending others affected with it; the all but universal occurrence of the disease in nurses and resident medical attendants, put beyond a doubt the communication of the disease. Lassis, shortly after publishing his ingenious work, 'Recherches sur les Veritables Causes de Typhus, ou de la Non-contagion des Maladies Typhoides,' fell a victim to the contagion, the existence of which he denied. Need I further refer for the proof of this position to the names of Sydenham, Morton, Pringle, Huxham, Hildenbrand, Chomel, Tweedie, Roupell, Gauthier de Claubry, Montault, Lombard, indeed to almost all the writers on the subject? The facts stated by Tweedie, from p. 85 to p. 99 of his treatise, by Gauthier de Claubry (p. 100 to 114), and by Dr. West in his able paper on "Exanthematic Typhus," in the fiftieth volume of the 'Edinburgh Medical and Surgical Journal,' are conclusive as to the communication of typhus to medical attendants, nurses, and patients labouring under other complaints, and they are completely borne out by my own experience. It is well known, that, for many years past, every resident clerk in the Glasgow Infirmary, with very rare exceptions, many students who frequented the fever wards, several of the acting physicians, and almost all the nurses, have, at one time or other, been attacked with typhus, and that not a few have fallen

victims; and Dr. Cowan, under whom I had for a long period the honour of serving, in his pamphlet, entitled 'Statistics of Fever and Smallpox in Glasgow,' says (p. 10) of the district surgeons, "Few of those gentlemen escape an attack of fever." Dr. West's statements as to the spread of the disease among patients admitted for other complaints to St. Bartholomew's, but placed in the same wards with others affected with typhus, and the necessity of at length closing the ward, show the groundlessness of the objection that contagion arises merely from concentration. During the winter of 1837-38, an isolated case of typhus in one of the medical wards of Glasgow Infirmary, communicated the disease to most of those in the same ward, and several died. The following are the results I obtained from the statements of 139 patients, whose cases I investigated with particular care in the years 1837-38. Much more extensive researches subsequently made by Dr. Anderson and myself, the details of which, unfortunately, I have not with me, will, I doubt not, bear out this statement.

	Doubtful or cause unknown.		From infection.		Typhus in family.		In same house.		Contagion.
Males	21	.	39	of	27	.	8	.	4
Females	18	.	61	whom	42	.	12	.	7
	39[1]		100		69		20		11

I have already spoken of the conditions in which 22 of the patients were placed who denied the influence of infection in the production of their complaint. Gauthier de Claubry remarks (p. 113) that it requires "some degree of medical courage" to uphold the propagation and perpetuation of typhus by specific infectious germs, according to Hildenbrand. On the supposition of its identity with typhoid fever, this may be so; the difficulty vanishes on supposing them different.

What is the state of the case in typhoid fever? It is well known, and stated both by Chomel and Gauthier de Claubry, that the all but universal opinion in France is against its being infectious. Louis does not even mention it; Andral

[1] Of these 39, there were 30 who positively denied contagion, 6 are not marked, and 3 were doubtful.

(p. 728, English edition) gives his verdict against it; Montault (pp. 12 and 186) recapitulates the arguments against it. On the other hand, MM. Bretonneau, Gendron, Chomel, Lombard, and Gauthier de Claubry maintain, more or less decidedly, the contagious nature of the disease. It is rather amusing that the last-named author, while arguing for the contagion of typhoid fever, in order to prove its identity with typhus, adduces the contagion of the latter in the English hospitals, in support of his position, while Montault, holding the difference of the two affections, feels constrained, in order to get over an imaginary difficulty, to express a doubt (p. 12) as to the cleanness and salubrity of the British, as compared with the French hospitals, and to assert that "the English physicians are not perfectly acquainted with the anatomical alteration of typhoid fever;" while Gauthier de Claubry, in order to get over the real difficulty arising from the absence of anatomical lesions in the Glasgow and Edinburgh typhus, speaks of the "serious circumstances of misery and exhaustion of strength, which have so deplorably modified the constitution of the inhabitants of these wretched countries, Scotland and Ireland." Those who know the admirable cleanness and ventilation of our fever hospitals, and the acquaintance of our medical men with the lesions referred to, will readily dispense with any serious refutation of such gratuitous suppositions; and the too sweeping assertion of Gauthier de Claubry, as we shall afterwards see (though partially borne out by the recent statements of Dr. Alison, regarding the condition of the lower classes in the large cities of Scotland), even were it admitted in its fullest extent, proves nothing in his favour. Chomel's third conclusion (p. 339) is, that, if the identity of the anatomical lesions in the two diseases were proved, the question of contagion would be set at rest. We shall see hereafter what is the testimony of facts in regard to the lesions in typhus. Meantime, let us consider the facts in reference to contagion.

Louis, I have mentioned, does not allude to the subject. From this we might conclude, that he had found no facts in support of the theory of contagion, for we can scarcely suppose that an observer so scrupulously accurate would neglect

so important a branch of his subject; but we prefer an appeal to positive facts. Chomel, we have already seen, states how very few patients ascribed their illness to any energetic cause. Five only out of 115 were placed "in circumstances favorable to contagion," 79 could "assign no appreciable cause" (p. 306). In accordance with this I may state that, in no case, though questioned with the greatest care, either in Scotland or in the hospitals of Paris, have I ever found the disease referred to contagion. On the other hand, it appears from the statements regarding the epidemics of Nancy by Leuret, of Chateau de Loire by Gendron, of Andlau and Stolzheim by Mistler, of Bischofsheim by Reuf, and those contained in Bretonneau's memoirs (commented on by Gauthier de Claubry, from p. 118 to 135), and from the clear statements, borne out by dissections, of Lombard (in his valuable paper upon the Geneva epidemic of 1835, inserted in the 'Gazette Médicale'), seem to prove the possible transmission of typhoid fever, when epidemic. Without hazarding any decided conclusion on a point still so keenly debated, I would simply direct attention to the differences as regards the probable origin and propagation of the two diseases, which the foregoing facts appear to establish; and also to the interesting question, whether, if typhoid fever really depends on, and is propagated by a specific poison, that poison is, or is not generated in the same circumstances as the infection of typhus?

III. There are circumstances connected with the course of the two diseases well worthy of attention. The fact, as undoubted as it is remarkable, that the mean duration of typhus is about one half that of typhoid fever, is one that perplexes considerably the advocates of their identity. Chomel argues (p. 337) that it only shows a difference of intensity. Gauthier de Claubry, who very judiciously does not venture to face the statistical details on the subject, and treats of it only under the head of the relative intensity of the two diseases, meets the objections on the same ground as Chomel. I have already remarked the extraordinary difference of the duration in the Glasgow epidemic of 1836. Hildenbrand states the mean duration of typhus at about twenty-two days, and that of the Glasgow typhus, from the

results of many thousand cases, during successive years, approaches very nearly to this result, being about twenty-one days. An analysis of my own cases gives me a mean duration of 20·82 days, or, deducting three cases, one of which terminated fatally from phthisis on the 42nd, another not convalescent from consecutive paralysis on the 60th, and a third restored to health about the 100th day, after severe attacks of glossitis, pleuro-pneumonia, and a variety of other complaints, 19·6 days. Valleix, in his third memoir on 'Typhus and Typhoid Fever,'[1] states the mean duration of six cases of the former, and seven of the latter (observed in London by Dr. Shattuck, of Boston), as respectively 24½ and 22⅕ days. To account for this discrepancy with the unanimous voice of other authors, it is enough to refer to the small number of cases, and to state that the cases of typhus were of great severity (five out of six having died), while those of typhoid fever were very mild, and only one in seven terminated fatally. It appears, on the other hand, from Chomel (p. 44), that three fourths of the cases cured began to show signs of amendment, only between the 15th and 30th, and eighteen of the whole number only between the 31st and 40th days. Louis again (vol. i, p. 9, 13) mentions the duration in cases of death to be from eight to beyond forty days; in serious cases which were cured, from fifteen to beyond fifty; or on an average thirty-two days; while that of the light cases was twenty-eight or twenty-nine days. Montault (p. 22) states, that of sixty-three cases observed by him, the mean duration was thirty-six days. The mean duration of nine cases of all different shades of intensity, very carefully watched by myself, chiefly in the wards of M. Chomel in the Hôtel Dieu, is 38·7 days. One of these, admitted on the 13th of last December, has not yet terminated. I take the duration of the fever from his first convalescence (on the 75th day), after which he had a relapse without any marked local affection, and his recovery is very doubtful. M. Chomel in his summary on the 22nd of last March, of all the fever cases (twenty-five in number) that had occurred during the previous six months, stated

[1] 'Arch. de Médecine,' 3me série, t. vi, p. 135-138.

The mean duration of the very serious cases as - 31 days.
those of moderate intensity, as 18
the lightest cases, as - - 10

giving an average for the whole number of 19·6 days. He, however, called particular attention to the fact, that the cases were much less numerous, and the affection greatly milder, and much less fatal than usual, the mortality, commonly 1 in 3 or 4, being only 1 in 12·6.

Being firmly persuaded that the remarkable length of typhoid fever often depends upon conditions not commonly adverted to, I beg the reader's indulgence while I go into some details on the subject. What I wish to establish is, that typhus, when uncomplicated with any secondary affection, terminates in convalescence or death in the course of a single attack; in other words, that a second attack of typhus does not occur in the course of one and the same illness. On the other hand, it shall be my object to prove that such is not the case with typhoid fever.

With respect to typhus, I allege generally, notwithstanding the high authority of Dr. Copland, who maintains directly the contrary,[1] that (apart from secondary complications, which often completely change the course and termination of the disease) however long may be the period of excitement, —however long the adynamic stage,—however tedious the period of convalescence, I have never, among thousands of cases, seen a single case of relapse, in the proper sense of the term, after the symptoms had begun to decline. Is this objected to as vague? I appeal to the following facts. Of 139 cases, examined during the course of the disease with great minuteness, I find that 119 presented no secondary complications. Of these 20 died at the height of the fever, the symptoms having presented a constantly increasing intensity till the fatal termination. The remaining 99 offered, without a single exception, a steady aggravation of all the symptoms till a certain period (more or less distant, according to circumstances, from the beginning of the disease), after which they exhibited as steady a decline. The remaining 19 were complicated in varying proportions

[1] 'Dictionary,' p. 1012, art. "Typhus Fever."

with phthisis, pneumonia, chronic bronchitis, hemiplegia, erysipelas, bedsores, diarrhœa, &c., to one or other of which diseases four fell victims, the secondary affection generally declaring itself after a temporary amendment.

Let us now inquire into the state of the case in typhoid fever. I find Montault (p. 18), when treating of convalescence, remarking, that "relapses are easy" in that period. Louis,[1] speaking of the same period, uses these words: "Yet several of them (the healthy functions) were re-established very slowly in several individuals, and convalescence was thereby retarded. The heat was more or less great; the pulse continued more or less quick; the diarrhœa persisted, though it could not always be referred to imprudences in diet." This is all I have been able to find in the various authors I have consulted. I shall now cite several cases in point.

On the 26th of February, 1838, a young man, seventeen years of age, was admitted to the wards of the Glasgow Fever Hospital. He had been ten days ill with most of the symptoms of follicular enteritis. On admission he had a few rosy spots on the belly, very red and papillated tongue and fauces, circumscribed flushing of cheeks, disturbed sleep, tickling cough, extensive bronchitis, dull pain, tumefaction, and gurgling in the belly, with moderate diarrhœa. He had expistaxis on the 12th, 18th, 19th, and 23rd days of the disease, and on one occasion to the extent of one or two pounds. The eruption became pretty copious, then declined, and on the 30th day disappeared. At that time the pulse, which had ranged between 104 and 90, fell to 80 and 76; the bronchitis all but disappeared; the tongue, which had been excessively red, chapped, and aphthous, became smooth and less florid; the gurgling less; and the appetite good. On the 20th March (the 32nd day), he had rigors, continued on the 21st with rise of pulse to 104, and followed on the 23rd by pretty copious eruption on breast and back, by return of thirst, clamminess of tongue, loss of appetite, sibilant wheeze in chest, renewed tenderness of belly, frequent bilious vomiting, continued for five days, and epistaxis for

[1] Vol. ii, p. 11.

six days running. The symptoms followed their regular course, and the eruption had nearly disappeared, when, on the 4th of April (47th day), a fresh, elevated, lenticular eruption appeared on the chest and epigastrium. The only altered indication after the appearance of this eruption was a temporary rise of pulse from 92 to 104. On the 11th of April (54th day) he was dismissed by his own desire; and I heard some time afterwards that he had rapidly convalesced after his removal.

The phenomena observed in this case scarcely require any comment; and I therefore proceed to detail others that have fallen under my notice.

On the 9th of last December, a young woman, æt. 22, entered M. Chomel's female ward, the Salle St. Augustin, with symptoms of typhoid fever of ten days' standing. The affection was mild, the eruption abundant, the intelligence good. Amendment soon took place. The countenance, previously very languid, became more lively, the pulse sunk to 92, and she seemed rapidly convalescing, when, on the 24th of December (25th day)—as I learn from the reports furnished me by my friend M. Gueneau de Mussy, to whose experience and kindness I am much indebted—the pulse rose to 108. There were marked and sudden prostration, pain, and crepitant râle in the right subscapular region; deafness, which had been considerable, became complete; stupor and involuntary discharges came on after some days, and death followed on the 28th of December. Inspection disclosed the followed appearances. Incomplete splenisation of lower part of right lung; diseased aggregate glands at the lower part of the ileum, some ulcerated, some going on towards cicatrization, others not ulcerated, and in the state in which they are described about the sixth day of the disease. Who can ascribe the supervention of such serious symptoms as those I have sketched to the inflammation of a small portion of the lower lobe of the right lung? Is it not much more rational to refer them, and to ascribe the morbid appearances, as M. Chomel did in his lecture on this case, to a fresh intestinal eruption of a few days' standing?

Again, on the 27th of last February, a young man, æt. 21, entered the Salle St. Louis in the Hôtel Dieu, on the

8th day of typhoid fever. The eruption appeared next day, became pretty copious, and on the 13th of March (the 23rd day of the fever) it is reported as all but gone. All the symptoms had diminished greatly in intensity. The pulse had fallen to 76. Convalescence took place. He was eating and relished the half-diet of the hospital, when, on the afternoon of the 31st, the pulse, in the morning 90, rose to 136. Violent diarrhœa came on. The pain of belly, formerly slight, became considerable, and a pretty copious eruption of well-marked "typhoid spots" appeared. The chest, minutely examined, presented nothing unusual. On low diet the symptoms declined; and I find him marked in my notes convalescent on the 4th of April. He is about to leave the Hôtel Dieu.

I refer further to a case detailed at p. 30 of the first volume of Louis's work. The patient had been three weeks ill. During the first fortnight the ordinary symptoms, it seems, were observed; more particularly headache, relieved by expistaxis, during the first week; then gradually increasing diarrhœa and pain of belly. In the third week renewal of rigors and heat of skin. Appearance of the eruption and recurrence of epistaxis on the 23rd day. These phenomena were followed by violent delirium, terminating in partial stupor, and death on the 26th day. On dissection solitary glands, not ulcerated, in the upper; both solitary and aggregate glands ulcerated in the lower part of the small intestine; "a great number of small tumours flattened, not ulcerated," in the large intestines. Do not the symptoms fully bear out the opinion, that the formation of the tumours described by Louis was coincident with the aggravation of symptoms in the third week, and that the extensive ulceration at the foot of the ileum was the result of disease of much longer standing? The invaluable researches made of late years into the pathological anatomy of this affection by Bretonneau, Petit, and Serres, Andral, Louis, and Chomel, would necessarily lead to this conclusion. And, in point of fact, Louis says (p. 37) that they were "most probably developed at the same time as those in the first half of the small intestines, being, like them, not ulcerated." Now, according to the generally received theory, the

glands in the upper half of the small intestine are developed at an advanced period of the disease.

I conclude my remarks on this head with a reference to Chomel's work. At p. 361 is the case of a patient, admitted on the eighth day of the disease. Prostration, nausea, some epigastric pain, slight diarrhœa, "a few rosy spots," were the leading symptoms. They quickly yielded; the diarrhœa had almost ceased, and a great amelioration had taken place, when, on the 23rd day, all the symptoms returned, in a much graver form, accompanied by stupor, violent diarrhœa, "inclination to vomit (it is expressly said), *like that* at the beginning of the disease;"—"slightly marked" improvement on the 27th and 28th days; recurrence of same symptoms, with delirium and erysipelas, on the 30th day; extension of erysipelas to whole right side of face, and neck, and also to scalp. Almost every trace of it had disappeared on the 34th day, when epistaxis and stupor, approaching to coma, were observed. Death ensued on the 35th day. Dissection disclosed *a lobule* of grey hepatisation in one of the lungs. In the upper part of the ileum, "patches partially ulcerated; in the last fifteen inches, numerous ulcerations of very small extent, and which appear to depend on the alteration of the isolated follicles; besides, that a great number of the last-named glands present an abnormal volume, without ulceration" (p. 364).

The symptoms mentioned as recurring on the 23rd day, after the almost complete cessation of the diarrhœa and other unfavorable signs, cannot certainly be accounted for by the "lobule of grey hepatization," found after death, nor by the erysipelas, which did not come on for seven days afterwards, and was then ushered in by a new exacerbation, after a second decline of all the symptoms. In fact, we have there all the phenomena of a fresh attack on the 23rd day, followed on the 30th by erysipelas as a *sequela*. Am I not warranted, then, in concluding, that the exacerbation observed at the former date was accompanied by a fresh eruption of isolated follicles? This is strongly supported by another post-mortem appearance observed, viz. the uniform rosy tint of the whole of that portion of the small gut that lay in the lower pelvis.

After the facts that have been adduced, I feel almost entitled to expect assent to the likelihood of the opinion, which, I am convinced, future observation will confirm, that in *typhus*, when uncomplicated with any secondary affection, a second attack does not take place, while in *typhoid* fever the contrary is the case.

I have a few more remarks to offer upon the duration of typhus. There are cases which are said to run their course in a few days. I do not speak of the *typhus siderans* of Saragossa, Torgau, and Mayence—a disease that was often mortal in a few hours. It is not to these cases of frightful intensity that I allude, though I may remark with Montault, that the earliest death recorded in typhoid fever is, I believe, that of a patient of M. Bretonneau, on the 5th day. The cases I refer to are those in which a cure is said to take place on the 7th or 8th day, or even earlier. Many authors of distinguished eminence detail cases wherein certain modes of treatment, early applied, have cut short, as the phrase is, attacks of continued fever. In short, it has been often laid down, that an early bleeding, an emetic, a strong purgative, can crush the hydra in the birth, and restore the patient to health. Dr. Henderson in his part of the Report on the Epidemic Fever of Edinburgh,[1] states expressly, "If the presence of maculæ, red spots, or typhoid eruption, be considered as conclusive of the nature of the disease, though not necessarily a symptom in all cases of continued fever, then I can have no hesitation in deciding that this fever may run its course in a few days; for instances of convalescence have occurred to me on the 7th and 8th days, in which the eruption had existed, without which the diagnosis would have been perplexed, as in other cases."

Without denying the possibility of the above, I remark, that it would have been desirable to know, whether, in the cases spoken of, very minute inquiries were instituted respecting the exact days on which the patients were first attacked; for every one knows that the premonitory period of languor and uneasiness, extending often over a week, or even more, is seldom taken into account by patients in describing their complaints. But I object to the general

1 'Edinburgh Medical and Surgical Journal,' No. 141.

position on other grounds. It is matter of notoriety that very many cases of catarrh at all seasons, of influenza when it is epidemic, and of bilious affections during the heat of summer, are sent to the fever hospitals, because they simulate, in a most remarkable degree, the symptoms of commencing typhus. So it was in Glasgow Fever Hospital. These cases yielded in a few days to the exhibition of one or other of the above-mentioned remedies, while the same means were used, apparently without any effect, either as regarded their duration or intensity, in very many cases of genuine typhus. So firm is the belief in Glasgow that those which terminate in a few days are not cases of continued fever, that they are not permitted to go into the convalescent wards, where sad experience has shown the much greater contagiousness of the disease. In fact (and this militates strongly against Hildenbrand's opinion, that typhus is most contagious during the "nervous stage," beginning to be so when the eruption appears), scarcely one of the hundreds dismissed from the *acute* wards ever returned labouring under typhus, though they had remained for a week or ten days in wards sometimes crowded to excess; while, of the few who, by mistake, went into the *convalescent* wards, scarcely one escaped the disease, and several died. No patient who presented the eruption was ever known to contract the disease anew, even among the convalescent patients.

I cannot omit to notice another circumstance connected with the course of the two diseases, which appears to me one of considerable interest; I mean the occurrence of *crises*. I do not refer, by this expression, to the dark and debated question of *critical evacuations*, nor will I institute any inquiry as to whether certain discharges, on certain days, are or are not attended with an improvement in the general symptoms. All that I insist upon is the frequent, I may say, the common occurrence of a perceptible crisis, or what is vulgarly termed a turn in typhus. I think I may appeal to the experience of every physician, and more especially of every resident clerk in a fever hospital (for they have more constant opportunities of observation), whether they have not often been struck at seeing, during their morning visit, the glassy eye, the haggard features, the low

muttering delirium, the stupor approaching to coma, the tremor, the subsultus, the carphology, the rapid, thready, tremulous, and intermittent pulse, of the previous evening, the formidable array of symptoms, in short, which seemed to indicate a speedy and fatal termination, exchanged for the clear eye, the intelligent countenance, the steady hand, the comparatively slow and firm pulse, and the returning appetite of approaching convalescence. To such cases as these we might almost apply the Scripture phrase, "At such an *hour*, the fever left him;" and if the crisis is not *very* frequently so marked, we can, in the great majority of cases, point with precision at least, to the *day* on which amendment began to take place. In reply to the question whether this is the case in typhoid fever, I can only adduce my own experience, when I state, that neither in the numerous cases I saw in Scotland, nor in those I have watched in Paris (about a dozen of them very carefully), have I ever seen anything approaching, in the remotest degree, to what I have noticed so frequently in typhus.

IV. I proceed to a fourth and very important branch of my subject, viz. the consideration of symptoms. I shall not be tempted, by the highly ingenious and visionary conclusions drawn by Valleix, from his six cases of typhus,[1] to make any lengthened digression. That author actually considers the absence of headache and affections of the organs of sense as important diagnostic marks of typhus. Instead of wearying the reader with the laboured refutation I had prepared of this strange fancy, I shall merely state, regarding my own cases, that, excluding those in whom the headache ceased before the 5th day, it was present after that period in 98. In between one sixth and one seventh of this number it ceased before the 10th day; in the remaining five-sixths it continued throughout the advanced stages of the disease, being present in many till the 20th and 25th days; and in 11 throughout the whole course of the affection. Hearing, again, was either more acute, or perverted, or impaired in 69 patients. And finally, while I refer to the almost unanimous testimony of the multitude of distinguished authors cited by Gauthier de Claubry in pp. 5 and 39 of his

1 'Arch. de Méd.,' 3me série, t. vi.

work, I find recorded in my notes, as among the most constant symptoms, excessive intolerance of light (of which I can add my own experience); suffusion of the eyes to an extent rarely if ever witnessed in typhoid fever; and minute injection of the conjunctival vessels. In not a few cases chemosis, corneitis producing ulceration and onyx, iritis, and in two or three cases Egyptian ophthalmia, occurred in the course of the disease. What, again, are more common in typhus than the glassy eye (considered by Rochoux peculiar to it, from its very rare occurrence in typhoid fever), the immensely dilated and extremely contracted pupil, and even total blindness? If reminded that, in adducing those facts, I am proving the identity of the two diseases, I reply, that if to prove the existence of analogies is to prove identity, there is an end to all conclusive reasoning and accurate knowledge. In order to establish from analogy the identity of any two diseases, the resemblances must be so numerous, so striking, and, above all, so important, and the differences so few, so trifling, and so easily accounted for, as to warrant the overlooking of the latter conditions, which have certainly not as yet been answered in the case before us.

1. I have now to consider symptoms that have a material bearing on the decision of this important question. The state of the abdomen and bowels during life is naturally regarded as worthy of particular attention. The common use of purgatives in Great Britain throughout the course of typhus, and their well-known beneficial effects, while they render the declaration of Montault (p. 93), that Hamilton's method "still counts some partisans in England," not a little amusing, and "passing strange," the sweeping condemnation pronounced against their repeated administration by Gauthier de Claubry (p. 156–172), prove at the same time the existence of certain symptoms to be combated. Though not called upon here to undertake a defence of the English mode of practice as applicable in all cases of typhus, I refer to it as furnishing some very remarkable facts bearing on the question at issue. Dr. West, in his able paper on Exanthematic Typhus (in the fiftieth volume of the 'Edin. Med. and Surg. Journal'), makes the following observations:— "The action of the bowels was not disturbed in the great

majority of cases; in fact, the administration of mild laxatives was necessary, in most instances, in order to obtain an evacuation, once in forty-eight hours; and in some of the most severe cases the bowels were very constipated. Diarrhœa occurred only in 10 of these 60 cases; in three of which the patients died, and it was only four times that it lasted for longer than forty-eight or sixty hours. . . . In 13 cases the bowels were constipated." The following is an abstract of the statements of Dr. Henderson in regard to the state of the bowels in 154 cases of typhus.

	Males.	Females.	Total.
Bowels easy in . .	26	73	99
loose in . .	—	5	5
costive in .	19	31	50
	45	109	154

In reference to the following results obtained by myself, I have only to remark, that in many of those cases in which diarrhœa is described as spontaneous, the requisite information was unfortunately not got as to the previous administration of purgatives.

		Males.	Females.	Total.
Diarrhœa	Spontaneous in . .	13	10	23
	From medicine in . .	26	27	53
	Doubtful in . . .	1	—	1
		40	37	77
Costiveness notwithstanding the exhibition of purgatives . . .		20	42	62
		60	79	139

Thus, according to Dr. West's observations, the bowels were constipated in nearly one fourth; in Dr. Henderson's in about one third; in my own in between one half and one third of the cases. Again, spontaneous diarrhœa occurred in one sixth of Dr. West's; in only one thirtieth of Dr. Henderson's, or, adding three, in which it came on very late in the disease, and might be referred to secondary affections, in one nineteenth; and in nearly one sixth of my own cases.

How different is the state of matters in regard to typhoid fever? Chomel (pp. 6, 10) mentions diarrhœa as a symptom

scarcely ever wanting, and states it as occurring (p. 230) in 40 out of the 42 fatal cases recorded by him. In the still more precise observations of Louis, we find it wanting in only 3 of 128 cases, and in 58 of these it was contemporaneous with the commencement of the affection. Spontaneous diarrhœa was present in every one of the 9 cases observed by myself; in 3, it was one of the first symptoms, having declared itself on the first day, along with the general premonitory symptoms. In one, it began on the 4th; in another, on the 6th; in two more, on the 10th; in an eighth, on the 11th; in the ninth, it was observed after a few days' illness, the precise day not being specified. This symptom was present, in one case, from the 1st till the 22nd day, recurred on the 36th, and continued, during a second attack, till the 46th day. Its shortest and longest durations were respectively ten and forty-three days; and its mean duration above twenty-two days.

As to its characters, in two it was very violent, and opium had little effect in checking it. In two, it occurred in severe attacks, which assumed a periodic character; and in the rest it was moderate but always or nearly always present. Without insisting further on the comparative duration of the diarrhœa in the two diseases, though it would, if minutely investigated, bring out still more clearly the extraordinary contrast already presented,—I proceed to the consideration of the state of the abdomen in the two complaints. This I propose to do under two heads, relating, *first*, to the feelings of the patient, and *second*, to the sensations of the medical attendant.

2. In 30 out of the 60 cases observed by Dr. West there was pain in the abdomen, but in only 9 was it severe. Dr. Henderson remarked it in 46 out of 198, and never in the right iliac fossa. The following are the results of my own observation:

Abdomen painful in 109 out of 139 patients.

	Males.	Females.	Total.
Pain intense, or at least severe, in	17	27	44
slight	24	41	65
	41	68	109

The duration of the abdominal pain was as follows:

	Males.	Females.	Total.
Throughout the disease, in . . .	18	27	45
a considerable part of it, in .	6	9	15
From two to six days, in . . .	12	24	36
One day only	5	8	13
	41	68	109

Deducting the 13, in whom this symptom was present only one day, and in whom it may fairly be attributed to some accidental cause, we have 96 cases out of 139, in whom abdominal pain was somewhat permanent, and in 60 only did it continue throughout the greater part of the illness. In the great majority, the pain was general,—in 32, it was chiefly or entirely confined to the region of the liver, a circumstance noted by Dr. West;—3 of those in whom this was observed had long laboured under chronic hepatitis. In 17 of the 32 alluded to (between one half and one third of those in whom the pain was severe) the liver was exceedingly tender to pressure; and it was the seat of pain, more or less acute, in 20 out of the 60 patients in whom the abdomen was tender during the greater part of the disease. Dr. West mentions the usual duration of the abdominal pain as "about four days, though once it continued for fifteen days, and in three other instances from six to eight days." In 11 only of my own cases was pain observed in the right iliac region, 8 of these presenting it during one day, 2 during two days, and only one for three days consecutively. Valleix notices the presence of diffuse pain on pressure, for three or four days, in two of six typhus patients; and of pain on pressure and constant uneasiness in four, and of colic in a fifth of the six labouring under typhoid fever.

According to Louis, abdominal pain was present in 112 cases only of 128, was rarely general, occupied in the vast majority either the iliac fossæ or the hypogastric region, continued generally throughout the greater part of the disease, its duration being commonly from ten to twenty-five days. In six cases it lasted a whole month. The results of my own observation are, that abdominal pain was present in every case examined by me; and that in six, in whom pain

was observed during their residence in the hospital, it was either confined to the ileo-cæcal fossa, or more severe there than elsewhere, having been diffused over the belly in three of them during a part of their disease. It began in four on the 1st, in one on the 6th, in one on the 10th, in one on the 14th day of the fever. In the remaining two, it was present, in a decided degree, on their admission to the hospital, on the 11th and 13th days, and had most likely been present for some time before, though not felt by the patient.

This leads me to observe that the leading diagnostic symptoms in the Glasgow epidemic of 1836 were the red, chapped, and papillar tongue, the dull, circumscribed abdominal pain existing for weeks together, along with constant diarrhœa, harsh skin, and obstinate bronchitis. But what chiefly merits attention is, that while in typhoid fever the pain accompanies diarrhœa, in typhus the pain is often most severe when the bowels are costive, and is relieved on the exhibition of a purgative, proving its frequent connection with and dependence on a loaded state of the intestinal canal. In typhoid fever, on the other hand, it exists independently of any such condition; a circumstance which, along with its fixed seat, demonstrates its dependence upon local lesion. This is particularly pointed out both by Dr. West and Dr. Henderson, and it is fully confirmed by my own experience.

To sum up what I have stated on this head; abdominal pain occurred in 186 of 403 cases of typhus, or, deducting those in whom it was altogether transitory, in 173, *i.e.* between one half and one third of the whole; while in the 128 cases of Louis, it occurred 112 times (about five sixths), or, adding those detailed by Valleix and myself, in 126 out of 143 cases, *i. e.* in about ten elevenths of the whole.

3. The next subject that demands attention is the feel of the abdomen. Dr. West mentions 11 cases out of 60, as presenting "some fulness and tympanitic distension;" Dr. Henderson, only 8 out of 198. In those observed by myself, I find that the abdomen was

	Males.	Females.	Total.
Highly tumid and tympanitic in . . .	12	3	15
Slightly so	12	27	39
	24	30	54
Of natural feel	35	50	85
	59	80	139
This symptom lasted throughout the disease in	9	3	12
During a considerable part of it, in . .	3	2	5
From two to six days	7	16	23
One day	5	9	14
	24	30	54

Deducting, again, as I am fairly entitled to do, those cases in which the symptom was very transitory, there remain only 40 in whom it was at all permanent, and in 17 only out of 139 did it continue during the greater part of the affection.

In typhoid fever, on the other hand, Chomel (p. 12) remarks its almost constant occurrence, and Louis discovered it in 89 of 134 cases, in the great majority of which it lasted during the greater part of the disease. In one only did it disappear in twenty-four hours. Valleix[1] remarks its transient existence in one out of six or seven cases of typhus observed in London by Dr. Shattuck, whereas it was present at an earlier stage, in a much higher degree, and for a much longer period (its mean duration being about nine days) in five out of six patients labouring under typhoid fever. It was remarked in eight of the nine cases examined by myself, and was present from four to thirty-four days, or on an average a little more than nineteen days. In the remaining case it was so very slight as to render it doubtful.

Adding together all the cases of typhus above referred to, we find the symptom in question occurring in 74 out of 403 cases, or between one fifth and one sixth of the whole. Deducting those in which it was transitory, it occurred in 59 only, or in less than one seventh of the whole number; whereas it was observed in 102 out of 149, or in rather more than two thirds of all the cases of typhoid fever—no small difference assuredly.

[1] 'Arch. de Médecine,' 3me série, t. vi, p. 142.

4. Yet the statements I have made are very far from exhausting the evidence on the subject. I, therefore, proceed to inquire into the relation subsisting between the symptoms spoken of; and *first*, in regard to the coexistence of pain of belly and looseness of bowels in typhus. Of 50 patients in whom pain and diarrhœa existed at one period or other of the fever:

	Males.	Females.	Total.
Diarrhœa was { spontaneous in . . .	8	9	17
Diarrhœa was { consecutive in	15	18	33
	23	27	50
Pain and constipation were observed in . .	16	41	57

The numbers in which diarrhœa and constipation were observed were respectively 77 and 62. Therefore, had abdominal pain occurred in the same proportion in those who had diarrhœa, as it did in those who had constipation, instead of having 50 only, we should have met with it in 70·8. In other terms, the occurrence in the same cases of pain and diarrhœa is as 1 : 1·54, while that of pain and constipation is as 1 : 1·08.

Remarkable as this difference is, it is in reality much greater. I find from a further and very careful analysis of the cases before me that abdominal pain and diarrhœa were

	Males.	Females.	Total.
Coincident in	14	16	30
Not coincident in	3	4	7
Pain was relieved by supervention of diarrhœa in	4	8	12
	21	28	49

Again, abdominal pain and constipation were

	Males.	Females.	Total.
Coincident in . .	16	35	51
Not coincident in .	0	6	6
	16	41	57

Thus, in only 30 out of 77 cases in which diarrhœa, either spontaneous or consecutive, was noticed, did abdominal pain and diarrhœa exist simultaneously; whereas, out of 62 cases in which the bowels were confined, pain in the belly and

constipation coexisted in no less than 51. Had the latter proportion been kept up, we should have had 63·3 instead of 30, so that, according to our present data, the coincidence of abdominal pain with constipation in typhus is to its occurrence along with diarrhœa as three is to one. I might still further heighten the contrast, by deducting one who died of phthisis, in whom the abdominal pain did not supervene till after the fever had run its course, and in whom it was found on dissection to depend on very extensive tubercular suppuration and ulceration of the intestines; another in whom it supervened about the 20th day, along with a profuse discharge from a very large bedsore; and a third, in whom the evacuations, though frequent, were mixed throughout with scybala; but the difference is striking enough without any such refinement.

I have marked a considerable number of the above as relieved by diarrhœa, a circumstance which naturally provokes inquiry into the previous state of the bowels. I find, then, that of the 30 in whom pain and diarrhœa were contemporaneous, 10 may be called cases of spontaneous diarrhœa, *i. e.* the purging was not, so far as I know, brought on by medicine; while in the remainder it may be ascribed to that cause. In 28 of those in whom diarrhœa, whether spontaneous or consecutive, was observed, the bowels were more or less obstinately constipated during the first stage of the fever; and in that number are included almost all those in whom diarrhœa gave relief to pain. And finally, of the 51 in whom constipation lasting throughout the disease was accompanied by pain (in many cases exceedingly acute) the operation of purgative medicine,—of castor-oil, turpentine, jalap, calomel, or the neutral salts, given according to the strength of the patients, and the indications presented in the course of the disease, was followed by decided and immediate relief in 20 cases. Dr. West mentions, that of 30 who had pain, 8 only had diarrhœa, while 15 were constipated; a result that fully bears out my statements.

Abdominal pain and spontaneous diarrhœa were coincident in all of the nine patients labouring under typhoid fever to whom I have alluded. The shortest period of coexistence was four, the longest thirty-two, and the average is a little

more than fifteen days. So constant did Louis find the coexistence of these two symptoms, that (vol. ii, p. 34, 38) he considers abdominal pain, both in typhoid fever and other diseases, as pathognomonic of an inflamed state of the mucous membrane. In opposition to this I may quote Dr. Henderson, who remarks in his report, "In eight, a degree of general tenderness of the abdomen coexisted with tumidity and tension, in only one of which was there diarrhœa."

These statements form the best possible apology for the judicious administration of purgatives in typhus, while the spontaneous supervention of diarrhœa in many whose bowels were confined demonstrates its propriety beyond the shadow of a doubt, and fully warrants, as it appears to me, a most important conclusion, in proof of which I have adduced the above rather dry and tiresome details, namely, that whereas in typhoid fever there is an early and almost uniform tendency towards looseness of the bowels, there is as constant a tendency in typhus to constipation; that while in the former diarrhœa is a symptom, and a natural, I might almost say a necessary, result of a constant local lesion, it arises in the latter from the presence of an irritating substance, which it is the object of the *vis medicatrix* to eliminate from the system.

5. I shall offer only a few remarks on the connection between the state of the bowels and the feel of the belly. Of 54 patients in whom, as has been seen, more or less swelling and tympanites (*meteorismus*) were observed, 25 had diarrhœa, and 29 were constipated. The following tables will show much more concisely and clearly than I could express it in words the duration and extent of the tympanites in these two groups of cases:

	Bowels loose.			Bowels bound.		
	Males.	Females.	Total.	Males.	Females.	Total.
Tympanites continued throughout the disease in . . .	6	1	7	3	2	5
Throughout the greater part of it in	1	0	1	1	2	3
From two to six days in . .	5	4	9	2	12	14
During one day in . . .	4	4	8	2	5	7
	16	9	25	8	21	29

On this table I have only to remark, that 7 of the 25 had spontaneous diarrhœa, but of these 7 only 2 had tension of the belly for more than one day. But, by a careful analysis of the cases, we arrive at still more remarkable conclusions. The following are the results of my investigations:

	Males.	Females.	Total.
Tympanites and diarrhœa coexisted in . .	12	5	17
Did not coexist in	3	2	5
Tympanites was relieved by diarrhœa in . .	1	2	3
	16	9	25
Tympanites and constipation coexisted in . .	8	18	26
Did not coexist in	0	3	3
	8	21	29

Of the 29, the distension was relieved in 3 by the exhibition of purgatives. The 5 in whom diarrhœa and tympanites did not coexist were constipated at the time the tumefaction was noticed, so that in point of fact distension of the belly was coexistent with diarrhœa in 17, whereas it existed along with constipation in 31. Finally, of the 7 mentioned above as having meteorismus and spontaneous diarrhœa, the two symptoms existed simultaneously only in 4. Dr. West, I observe, states,[1] that of 11 cases in which "there was some fulness and tympanitic distension of the abdomen," diarrhœa was coexistent with it in 7, "while in 3 the bowels were constipated." As to the extent of the tympanites, I find that

	Bowels loose.			Bowels bound.		
	Males.	Females.	Total.	Males.	Females.	Total.
The abdomen was highly tympanitic in	8	0	8	4	3	7
Moderately or slightly so in . .	8	9	17	4	18	22
	16	9	25	8	21	29

6. In 46 (17 males and 29 females) out of 54, in whom meteorismus was observed, abdominal pain was also present at one period or other of the complaint, but the two symptoms were coexistent only in 37 patients, in several of whom the pain was so very transient and so slight, in comparison to the duration and extent of the distension, as scarcely to

[1] 'Edinburgh Medical and Surgical Journal,' vol. i, p. 134.

merit notice. To show still further that there is no proportion between the extent of abdominal tension and the pain experienced, I would merely state that, of 15 patients in whom the belly was highly tumid, 6 only felt severe pain; in 5 there was slight tenderness; in 4 none whatever. Of 44, on the other hand, in whom the belly was acutely painful, we have seen that only six presented any remarkable tension; in 18 it was slight; in 20 there was none at all. Dr. West's statements are widely different. He states (loc. cit.) that pain was present in all, except one, of those in whom the belly was tympanitic.

I shall conclude my remarks on this head with a few statements regarding the simultaneous occurrence of the above symptoms in my own cases of typhoid fever. Tumefaction of the belly and diarrhœa coexisted in eight patients, from 4 to 30, giving an average of fifteen days and three-quarters. Tumefaction was unaccompanied by pain in one patient, and lasted from the 12th to the 33rd day. In another, abdominal pain was present on admission, but disappeared next day, though the distension persisted throughout. In a third, the abdomen was swollen, but not painful, from the 9th to the 24th, both swollen and painful from the 25th to the 31st day. In the remaining 5, the symptoms coexisted from four to thirty-four days, or on an average about twenty days and a half.

I go on to speak of the eruptions observed in the two diseases. This subject has lately begun to excite much interest, more, perhaps, in Great Britain than in France. The proposal which has been made to overturn long-received opinions, and to blot out a long and elaborate section from the nosological dictionary, by placing typhus among the exanthemata, could not fail to awaken a most interesting controversy, and to bring under review the whole history, and much of the symptomatology of typhus. Hildenbrand was the first to propose this innovation. But it was started in England and Scotland by two physicians, who had neither had any communication with that distinguished author nor with each other, viz. by Dr. Roupell, who, it appears from his work, brought the subject under the notice of the London College of Physicians in 1831; and by my friend Dr. Peebles,

who, on his return to Scotland in 1832, after a long residence in Italy (where he had been struck with the constant occurrence of an eruption in the epidemics of contagious fever), drew the attention of the profession in Edinburgh to its presence, previously unnoticed, in the typhus of that capital; and after two years of attentive inquiry, published the result of his researches, accompanied with a comprehensive sketch of the history of eruptions in contagious fever, in the forty-fourth volume of the 'Edinburgh Medical and Surgical Journal'—being the first, as I believe, to submit his sentiments to the medical world. I observe that Dr. West, in an able and learned paper in the last number of the same periodical, maintains the opposite opinion. For several reasons I shall not enter upon this controversy. Besides that the proof of the positions I mean to take up does not require it, it appears to me that we are not yet in possession of data sufficient to demonstrate the truth of the doctrine. That must be the work of many years of patient and very extensive inquiry. I may remark, however, that there is everywhere a growing conviction in its favour, and that, if the claim of typhus to rank among the exanthemata has not yet been fairly made out, its remarkable analogies with that class of diseases are attested, alike by the uniform experience of the past, and the observation of present times. Nor can I consent, without reserve, to conclusions drawn from the alleged absence of eruption; for the fact I have already referred to (viz. that the eruption in the Edinburgh typhus was unheeded before 1832), shows how appearances may escape the eye of the most distinguished and practised physicians when their attention is not particularly drawn to them. It is also well known to many that, previous to a visit which Dr. Peebles made to the Glasgow Fever Hospital in the spring of 1835, the exanthema of typhus, then found to be of general occurrence, had neither been looked for nor registered in that institution, and was actually hailed as a new discovery. And finally, I must express my conviction, founded on observation, that a very large proportion of those cases mentioned in fever reports, as having no eruption, consists of patients (such as I have already referred to) in whom the disease is only of a few days' duration, and does

not, for the reasons stated above, seem to merit the name of typhus fever. These considerations, it appears to me, have an important bearing on the decision of this debated question.

While, however, I decline making any further remarks on this branch of inquiry, I must refer shortly, for the practical elucidation of my subject, to the history of the typhous eruption. Much confusion prevails on this point. The meaning of the term *petechiæ* has, in the course of time, undergone a great change. That it was used by the old authors (loosely enough, doubtless) to denote what would have been much better called an eruption, is evident from their writings, and has been very clearly shown by Dr. Peebles, five years ago, and by Dr. West in the April number of the 'Edinburgh Journal.' Dr. Peebles accordingly proposed to retain the old term in its altered but now generally received acceptation, and to consider the rosy eruption as an exanthema. Yet many, from the use of the term petechial by the older writers, persist in denying their acquaintance with the eruption; some, on the other hand (Rasori, Pinel, Tweedie), having used the same word in reference to the eruption, are blamed for want of "precision and exactness in the use of terms;" Chomel (p. 336) considers "the true petechiæ or purple spots" to be very much more frequent in typhus than in typhoid fever; Valleix[1] maintains the eruption to be always dark and persistent, and takes it as synonymous with vibices; while Copland[2] and Montault (p. 154) describe "ecchymoses and petechiæ as common to typhus and other diseases," and the measly eruption as peculiar to it. Again, Roupell quotes Louis and Chomel in support of his views as to the true exanthematous character of typhus. One of his reviewers,[3] reasoning on the generally admitted difference of the French and British continued fever, retorts Dr. Roupell's arguments as proof positive against the truth of his theory, while, with praiseworthy consistency (p. 103), he cites Chomel's statements regarding the eruption in support of his own opinions.

[1] 'Arch. de Méd.,' 3me série, t. vi, pp. 133-4-6, 148-9.
[2] 'Dictionary,' p. 1010, art. "Typhus."
[3] 'Med.-Chirurg. Review,' vol. xxxi, p. 99.

I shall now endeavour, in proof of the two following propositions, to present, in small compass, the very numerous statements of authors, to reconcile their apparent contradictions, and to give the results of my own observation regarding the exanthema of typhus. The points I wish to establish are:

First, That the rash in typhus is permanent; that in all cases it presents the two periods, longer or shorter, according to circumstances, of increase and decline; that, in the more severe cases, it may exhibit, during the period of increase, four different states, being florid, dark, livid, and petechial; and finally, that it is quite different from vibices and purpura, which are of rare occurrence.

Second, which partly flows from the foregoing, that the abundance, and particularly the darkness of the eruption, may be said to be proportional to the severity of the disease.

1. When I say that the rash is permanent, I mean that it does not consist of successive eruptions of spots, each of which disappears in the course of three or four days, but that the same eruption continues throughout the disease. As the accuracy of this position will be clearly brought out in treating of the changes the eruption undergoes, I shall only state that it was ascertained by surrounding a certain number of spots with ink, and observing them carefully several times daily.

Dr. Copland and Dr. Peebles, to whom he refers, lay down the proposition that the "eruption usually appears from the 3rd to the 7th day of the fever, but may be delayed till the 12th or 14th day." Dr. West, in his last paper, mentions that in the Vienna fever of 1757, when the disease ran its regular course, it appeared "on the 4th, or, at latest, on the 7th day."[1] Montault (p. 154) refers to Drogart, Biett, Rochoux, and Pringle, as having observed it generally on the 4th, 5th, or 7th, and sometimes (according to Pringle) on the 14th day. Gauthier de Claubry (p. 75) remarks, "That of seventeen observers who make express mention of it, four noticed it on the 4th, two on the 5th, eight in the course of the 6th and 7th, and generally, all point out its manifestation between the 7th and 10th days." Dr. Roupell

1 'Edinburgh Journal,' No. 143, p. 295.

maintains its almost uniform appearance on the 3rd or 4th day. Dr. Cowan, in his 'Statistics of Fever and Smallpox in Glasgow,' p. 24, says that it "generally makes its appearance from the 4th to the 9th day of the disease, occasionally at a later period." Dr. Henderson states that in 8 out of 12, in whom the day on which it appeared could be ascertained, it appeared from the 3rd to the 6th, in "the remaining 4 severally on the 7th, 9th, and 11th day." I have been able to ascertain the exact date of the appearance of the eruption in a much larger number than Dr. Henderson, to whose statements, in addition to the following tables, I refer for the proof of what I have advanced respecting its increase and decline. The first table refers to the time of its appearance, the second to the period at which it began to decline, the third to the date of its final disappearance.

	Males.	Females.	Total.
It appeared on the 2nd day in .	1	1	2
3rd „ .	2	1	3
4th „ .	3	2	5
5th „ .	4	12	16
6th „ .	4	9	13
7th „ .	1	4	5
8th „ .	2	1	3
9th „ .	1	3	4
13th „ .	0	1	1
	18	34	52

From this statement it results that in more than half of the entire number it appeared on the 5th and 6th days, and that, in exactly three fourths, it appeared from the 4th to the 7th day. Taking an average of the whole, it appears most commonly on or about the 6th day. I must, however, direct attention to the fact that in not a few cases it was so very copious, and in several so dark, on the 6th day, as to warrant the belief that it had appeared some days before.

Dr. Henderson states that the eruption began to fade "in 11 before the 10th day; in 20 between the 10th and 12th, inclusive; in one, as late as the 17th."[1] The following table gives the results of my own observation. It refers only

[1] 'Report,' &c., p. 13.

to 48 of the 52 cases above referred to, 4 having died before the eruption began to recede.

	Males.	Females.	Total.
It began to decline on the 8th day in .	1	0	1
9th „ .	3	3	6
10th „ .	0	5	5
11th „ .	2	3	5
12th „ .	1	4	5
13th „ .	5	3	8
14th „ .	3	3	6
15th „ .	1	3	4
16th „ .	0	2	2
17th „ .	0	2	2
18th „ .	1	2	3
19th „ .	0	1	1
	17	31	48

It is evident from the above table that we cannot fix any particular day, from the 9th to the 15th, inclusive, on which, much more frequently than any other, the eruption begins to fade. It did so, however, on one or other of these days, in 39, or five sixths of the entire number, the average of which, (12·8), assigns the 13th as the day that the change most commonly takes place.

The length of this paper, already too great, renders it impossible, as its object happily renders it unnecessary, to analyse the circumstances whereon the variations I have noted depend, such as the age, the constitution, the strength, the habits of the patient, and the greater or less intensity of the disease, all of which have a material influence in modifying its duration.

I shall now, in a third table, show the various dates of the disappearance of the eruption, in 45 cases, 3 more having died, very shortly after it began to recede.

	Males.	Females.	Total.
It disappeared on the 13th day in .	0	3	3
14th .	0	4	4
15th .	2	1	3
16th .	2	2	4
17th .	0	2	2
18th .	2	5	7
19th .	0	5	5

	Males.	Females.	Total.
It disappeared on the 20th day in . .	1	2	3
21st . .	1	1	2
22nd . .	3	2	5
11th, 12th, 23rd, 24th, 25th, 26th, and 31st days in . .	4	3	7
	15	30	45

The differences in this are still more striking than those in the preceding table. It would be easy, were such a part of my present purpose, to demonstrate that the long continuance of the eruption depends, in almost every case, on its having been dark, livid, or petechial during the disease; but, as the proof of it would not aid me in my present inquiry I go on at once to deduce some more practical inferences from the data I have furnished. Where so many disturbing forces are at work as those I have alluded to, it would be vain to look for anything like an exact proportion between the periods of increase and decline in the typhous exanthema. Accordingly, I find, from a laborious analysis of the above cases, that though in 8 the two periods agree exactly, and in 8 others, very nearly, in length, they vary, in the remainder in all different proportions. As it would be quite useless here to enter into details, I shall simply subjoin the results of my calculations:

	Days.
Shortest period of increase . . .	3
Longest „ „ . . .	13
Average „ „ . . .	7·08
Shortest „ decrease . . .	2
Longest „ „ . . .	13
Average „ „ . . .	4·45
Shortest duration of eruption . .	6
Longest „ . . .	24
Average „ . . .	11·59

These conclusions form a considerable contrast to Dr. Copland's statement, "The duration of this eruption is from three to five days." Dr. Henderson infers "that the whole duration of the eruption occupied commonly nine or ten days,"—a result which approaches much more nearly to that I have arrived at. Though I could have adduced a much

greater number of facts regarding the decline and disappearance of the eruption, I have preferred confining my attention to those patients alone in whom the date of its commencement also was ascertained, for, having been selected for examination solely because they presented themselves in the early stage of the disease, they form a perfect group, whence general conclusions may with safety be drawn.

Before entering on the consideration of the various states of the eruption, let me refer to some of the opinions put forth respecting it. Valleix, I have already stated, holds the eruption to be always dark, and persistent under pressure, both during life and after death. "It is," says Copland, "of a florid, reddish, or reddish-pink colour; disappearing on pressure, but soon returning when pressure is removed. This circumstance is sufficient to distinguish it from petechiæ." Again, "If petechiæ occur in this fever, they seldom are observed before the 8th or 10th day, and then this eruption has usually disappeared. When the petechiæ are earlier, or the eruption continues longer, so that both exist together, they are quite distinct and different in their appearances; for the latter is never so dark or livid as the former generally is." Further on, he remarks, "In the more malignant cases, or when petechiæ appear early in the disease, the colour of the eruption may, however, become deeper." Still further, "The petechial affection . . . may occur in the advanced stage of any fever . . . and is not in any sense of the word an eruption, as it has been very improperly denominated by some writers. . . . The petechiæ or cutaneous ecchymoses vary in dimensions from minute stigmata to large patches and vibices, and in the deepness or shade of colour. . . . But the exanthematous eruption attending true typhus . . . has been confounded with petechiæ, with which it is often associated in the advanced stages of the fever." These opinions agree entirely with those expressed in his paper by Dr. Peebles. Dr. Henderson (p. 16) has the following remarks: "Purple petechiæ, the result of ecchymosis, were frequently present in the second week of the disease. In some cases they were very abundant, but in very few exceeded the number of typhoid maculæ, which commonly existed along with them.

One vibex only was remarked in the whole number of cases The petechiæ continued often distinct, though the eruption was declining." To disprove, as far as my observation enables me, what appears to be incorrect in these opinions, even though upheld by such high authority, is the task I now propose to myself.

The statements of authors on this subject are very numerous. Dr. West, in tracing the history of the eruption downwards from the fifteenth century, cites Fracastorius and Ambrose Paré, who both speak of the eruption assuming a dark colour. Conradinus is more explicit. In the article referred to,[1] I find these words: "In addition to these symptoms, however, spots like flea-bites appeared on the skin of all the patients. In some cases, these spots were of larger size than others; occasionally they occupied the whole body, but usually were confined to the chest, the spinal and interscapular regions, and the arms; and they were observed to assume a livid or blackish colour in dying persons." Willis (same paper, p. 289) mentions as one of the symptoms of the epidemic among the troops near Oxford in 1643, an eruption of spots, some of them small, and of a bright-red colour, while others were larger and of a more livid hue. The reviewer of Dr. Roupell, formerly alluded to, says,[2] "The rash is not accurately described by any of the authors cited, so that it is impossible to say whether it was the same in all. One author (Pringle) does not even mention its existence." Now, as it so happens that Pringle *has* given, both in his work on 'Diseases of Armies,' and in his letter to Mead, a description of the eruption so clear as to make it be referred to by most writers on eruptions in continued fevers, it may be as well to quote his own words, which have an especial bearing on this and other portions of my subject. "There are," says he, "certain spots, which are the frequent, but not inseparable, attendant of the fever in its worst state. These are less usual on the first breaking out in the hospitals; but when the air becomes more corrupted the spots are common. They are of the petechial kind, of an obscure red colour, paler than the measles, not raised above the skin,

[1] 'Edin. Journ.,' No. 143, p. 282.

[2] 'Med.-Chirurg. Review,' vol. xxxi, p. 103.

of no regular shape, but confluent. The nearer these spots approach to a purple colour the more ominous they are, though not absolutely mortal." The most of the authors quoted by Gauthier de Claubry mention the existence both of the "rosy and petechial eruptions," the latter coming on at a later period of the affection, and in adynamic patients.

Dr. Roupell, in speaking of the opinion of some authors, that the "conversion of petechiæ into the rash is a common occurrence," has evidently, as his reviewer remarks, said the reverse of what he meant to convey to his readers. My friend, Dr. Staberoh, of Berlin, from observations of which I can attest the accuracy, made in Glasgow Fever Hospital, where I then resided, drew attention to the frequent conversion of the rosy eruption into petechiæ, in a paper published in the 'Medical Gazette,' for March, 1838, and in reference to it Dr. West has the following remarks: "My attention having been called to the point by a paper of Dr. Staberoh, I have endeavoured to ascertain, whether, as he asserts, the spots often change into ordinary petechiæ, but have not yet been able to satisfy myself upon the point, though, from one or two cases which I have seen, in which the spots greatly resembled petechiæ, I should think that his statement is very probably correct." Dr. Staberoh, in his thesis on the Exanthematic Typhus at Halle, in 1834, maintains the same opinion. Having thus, by quotations, shown that my opinion, so far from being original, has been partly recognised for centuries, and has been fully developed, of late years, by other writers, it only remains for me to offer the fruits of my own observation.

Having observed with care, several times daily, the cases referred to by Dr. Staberoh, in which a considerable number of spots were circumscribed with ink, I have seen the changes referred to going on in the most gradual manner; indeed, the fact was so clear that I find, in my daily reports of numerous cases, taken months before his arrival, and when my attention was not particularly directed to the point, the phrases, "pale, florid, darkening, dark, livid, petechial," applied successively to the eruption at various stages of the disease. In the details I am about to give the general principle is all I wish or need to establish; the periods at

which the changes take place I do not now seek to determine. Having specified the four states of florid, dark, livid, and petechial I would have it borne in mind that, when florid, the eruption disappears readily under pressure; when dark, it still disappears, but more slowly; when livid, semi-petechial, or pseudo-petechial (as it has been called), it is only partially effaced; when petechial it is not in the least affected by pressure. In many cases, it remains florid throughout; in others, it presents one or more, and in not a few, all these alterations; and after it has reached its height the process is inverted, and it passes through the various phases of lividity, darkness, redness, and paleness, before its evanescence. When death took place at the time the eruption was petechial, the ecchymoses (for they are true ecchymoses) remained after death—a circumstance noted by Dr. West, and particularly insisted on by Valleix in the paper already referred to. Dr. Henderson says (p. 12), "In a few, the tint was somewhat purple; and this colour could be assumed by an eruption that had formerly been scarlet." By including in the following tables the deaths in the various groups of cases, they will serve for reference, when I come to speak, as I shall do presently, of the relation that subsists between the states of the eruption and the gravity of the cases.

The colour of the eruption was:

	Cases.	Deaths.	Rate of mortality.
Pale throughout in .	34	3	1 in $11\frac{1}{3}$
Florid . .	25	2	1 in $12\frac{1}{2}$
Darkish in . .	16	2	1 in 8
Dark . .	32	8	1 in 4
Livid or semipetechial in .	15	7	1 in $2\frac{1}{7}$
Petechial . .	17	4	1 in $4\frac{1}{7}$
	139	26	1 in $5\frac{1}{3}$

Hence it appears that the eruption was pale in about one fourth; florid in between one sixth and one seventh; darkish in between one eighth and one ninth; livid in rather less than one ninth; petechial in about one eighth of the whole cases. Dividing them, then, into two groups, we have the following result:

	Cases	Deaths.	Rate of mortality.
Eruption light coloured in .	59	5	1 in $11\frac{4}{5}$
dark coloured .	80	21	1 in $3\frac{4}{5}$
	139	26	1 in $5\frac{1}{3}$

I have been thus minute in my subdivisions, in order to show more clearly the almost insensible gradations by which the rash is transformed from its florid or exanthematous into its petechial state; and in order to demonstrate at once its difference from purpura and vibices, and the truth of my concluding position. I met with purpura spots in three, and vibices in only two cases. In one of the former, a plentiful crop of purple spots appeared suddenly on the 14th day, on the chest and belly, amid a "pale, scanty, and very small" eruption. The pulse, which had before gradually increased in frequency, was 128 on the 12th day; on the 13th, it fell to 116. It continued to fall steadily, by about six beats each day, becoming gradually fuller and firmer; the intensity of all the other symptoms diminished as steadily, and convalescence was completely established on the 21st day. In another case, a large crop of very minute spots, differing completely in colour from the eruption, which had been petechial for days before, appeared on the 12th day, on the right arm, and faded away long before the eruption. The pulse, 120 on the 10th day, fell on the 11th to 104, and on the 13th to 96, and during the four following days to 54; on the same day epistaxis occurred, and the violence of all the symptoms diminished, but the severity of the attack left the patient exceedingly weak, and convalescence was not fairly established till the 31st day. In the third case, very numerous spots, similar to those already described, appeared around the left elbow on the 10th day, amidst a "darkish" eruption. On the same day the patient died. The countenance, which the day before was wild, flushed, and much oppressed, is reported "languid, much better;" and the pulse had fallen from 136 to 128, and was of better strength, but an intense bronchitis and the supervention of pneumonia, with great pectoral oppression, proved fatal. The difference in the appearance of the spots is well described by Dr. Staberoh. The spots of petechial eruption are ill-defined and shaded off at their margins, and have a dusky, reddish-

brown colour, while the spots of purpura, besides having abrupt and very clearly defined margins, present a decided shade of blue, which, as Dr. Staberoh remarks, renders the name applied to them very appropriate. *Vibices* appeared upon the chest in one case on the 10th day, amid a pale eruption, that became afterwards more marked, while the ecchymoses grew paler. The pulse, on the 9th day 124, had fallen to 112; the state of the patient improved rapidly, and convalescence took place on the 18th day. In another, the vibices were pretty numerous, large, and very irregular in form, and appeared on the 12th day, amid livid and petechial eruption; the pulse fell from 132, the number of pulsations on the day before, to 122, and convalescence was complete on the 20th day. From these details it appears clearly that purpura and vibices are of rare occurrence in typhus, are quite distinct from the typhous eruption, and that, instead of being attended with peculiar danger, they would actually appear to be critical. We may also conclude that if, as Dr. Copland insists, "the petechial affection is not in any sense of the word an eruption"—"the exanthematous eruption is" not only "often associated with" petechiæ, but is often itself petechial.

I now proceed to my second proposition. Dr. West (loc. cit., p. 141) says, that in the cases he observed "the abundance or scarcity of the eruption was not at all in proportion to the severity or mildness of the different cases." He considers petechiæ not to be particularly ill omened" (an opinion somewhat borne out by the facts stated above), "and certainly by no means so much to be dreaded as the dark and livid hue of the eruption, and of the skin generally, which is sometimes observed." To the first assertion, so vague and so general, I oppose his own words (at p. 290 of the 'Edinburgh Journal' for this year), where, speaking of Willis's account of the epidemic that broke out in the spring of 1643 among the troops near Oxford, and was for some time unaccompanied by eruption, he says, "About midsummer the disease increased in fatality," and then goes on to give the description of the eruption already referred to. I oppose to it also the general fact that the eruption is seen in its greatest perfection in extensive epidemics, during which the

disease is much more violent and fatal; and the fact, that in Glasgow, when the cases were few, the disease very mild, and the deaths 1 in 10, or 1 in 12, the eruption, if present, was never observed; whereas, when the cases became more numerous, the disease more malignant, and the deaths amounted to 1 in 8 and 1 in 6½, the eruption became steadily more and more abundant. Dr. Peebles accounts for the scantiness of the eruption in Edinburgh, at the time he made his observations, by typhus not being epidemic. Dr. Henderson states the deaths among those with abundant eruption as 1 in 5; among those with scanty eruption as 1 in 8·3. Two of the three fatal cases in the latter class presented "extensive visceral disease of long standing;" while not one of the thirteen in the former class had any such disease. The duration of the cases was, on an average, between two and three days longer in the one than the other. My own observations, likewise, show the relation between the abundance of the eruption and the severity of the disease. The eruption was:

		Cases.	Deaths.	Rate of mortality.
Copious	Universally in	96	19	1 in 5
	Partially in	32	5	1 in 6·4
Scanty		11	1[1]	1 in 11

The dark tint of the eruption is noted as ominous by all authors. The quotations I have given above prove the general opinion; and I may likewise refer to most of the authors cited by Dr. West, and Gauthier de Claubry, to Dr. Tweedie (p. 73), and Dr. Henderson (p. 12). The existence of a mortality in those with dark, three times greater than in those with light-coloured eruption, in the tables I have presented above, shows in the clearest manner the truth of these opinions, and renders needless any further comment. This fact ascertained, we cease to wonder at Valleix's sweeping conclusions, drawn from six cases, five of which were fatal.

Typhoid fever has also its eruption,—a circumstance much insisted on by Gauthier de Claubry in proof of its identity with typhus. It is generally agreed (Chomel, Louis, Gauthier

[1] In this one case the eruption was petechial, and, on inspection, I found disease of old standing, and aneurismal dilatation of the aorta.

de Claubry, Montault), and my own experience leads me to assert it, that it appears later than the exanthema of typhus, but that is a point of very minor importance; the main question is, are its characters the same? According to all the best authors on the subject (Chomel, Rayer, Biett, Rochoux, &c.) it is distinguished from the morbilliform eruption of typhus by being distinct, rounded, slightly elevated above the skin, and of nearly uniform size. In only one case (which I saw in the Hôtel Dieu last winter) have I noticed anything like an approach to the irregular, and very often confluent, rash of typhus, which is generally level with the skin, or if raised, is very slightly so, and that only during the stage of excitement. In all the other cases of typhoid fever in which I have seen the eruption I should say that it appeared in small spots, rounded, and almost papular in form, being considerably elevated in the centre. Dr. Perry, of Glasgow, was the first whom I heard maintain the complete difference of the two eruptions, and I am now fully satisfied of the accuracy of that opinion, for the following reasons:

1. The typhoid eruption is not permanent. That the typhous exanthema is so, has, I think, been shown by abundant evidence; but it is generally agreed that the rash in typhoid fever is "composed of several successive eruptions,"[1] that its duration varies from three to seventeen days,[2] the mean duration being seven and a half, and that "each rosy spot is not commonly visible for more than three or four days, and sometimes less." I have often been able to verify this remark.

2. I have never seen a single case in which the typhoid eruption became petechial or even dark, and no author I have consulted pretends that it does. On the contrary, it always retains the same characters, the last crops (and I have seen them appear the day before death, in cases where there was the most complete prostration) being as florid as the first. Reflections on the necessary relation between the state of the blood and the appearance of the eruption, which I cannot now develop, but which are strongly confirmed by the frequent occurrence of a buffy coat, and a pretty firm coagulum, in

[1] Chomel, 'Leçons,' &c., p. 19; also Louis, vol. ii, p. 232, 241.

[2] Chomel and Louis.

typhoid fever, will naturally suggest themselves to every mind.

3. It is beyond a doubt that there is no relation between the eruption and the severity of the cases. I confess that I long felt inclined to believe that the more plentiful the eruption the less serious the case. I was particularly led to think so by the total absence or extreme scantiness of the eruption (though carefully sought for) in the Glasgow epidemic of 1836, which was very deadly, and by afterwards finding it more or less copious in sporadic cases presenting a much less intense form of the affection. After further inquiry, I find, that though in its fullest sense the opinion is incorrect, it is to a certain extent true. From a careful perusal of the valuable work of Chomel, I find that a large proportion of the worst cases, and of those who died, had either a scanty eruption or none at all; and it appears from the lucid statements of Louis,[1] that it was wanting in 9 out of 35 fatal cases, and that it was scanty (*en petit nombre*) in 18, or three fourths of the remaining 26, some of them presenting only five or six spots; a number reckoned by Chomel (p. 18) of no value, fifteen or twenty being necessary, according to him, to "characterise the typhoid affection." Fifty-four of fifty-seven patients who had severe attacks, but recovered, had the eruption, and of the remaining three, two came to the hospital on the 14th and 40th days of the disease. "In some cases," he adds, "there was only a small number of spots." In twelve "the eruption was very abundant." It was present "in all the cases where the affection was slight," whence he infers, that "its cause is special," or specific, and that it is not like other secondary phenomena, proportioned to the gravity of the disease and the febrile movement." Hence we may at least conclude, that in severe attacks of typhoid fever, the eruption is less frequent, and generally less plentiful than in those that are less serious. Does any one object that these reasonings prove not the difference of the eruptions, but merely a different determination of one and the same eruption, in the one case to the skin, in the other to the mucous surface, which has been called "the inverted skin?"—I reply that, according to this

[1] Louis, vol. ii, 231, et seq.

hypothesis (for it is a pure hypothesis), there should be in typhus with scanty eruption a decided abdominal determination; but the fact, as undeniable as it is conclusive, is exactly the reverse; for numberless cases of typhus with scanty eruption are remarkable only for their mildness and the absence of any visceral complication. This is particularly the case in children, in whom the eruption is seldom seen.

We may also conclude generally that the facts detailed go against the opinion of Chomel (p. 336), that "the cutaneous exanthema offers the same characters in the two affections; the only differences being in the number of the spots, and the time of their appearance," and that, on the contrary, the typhous differs from the typhoid eruption in its form, its duration, the changes it undergoes, and the relation it bears, as to colour and quantity, to the severity of the disease.

V. In the plan I laid down, I proposed to consider some of the anatomical lesions. To enter on a comparison of the whole pathological anatomy of the two diseases were a task as tiresome as it would be barren of any definite results. Instead, therefore, of inquiring into the state of all the organs in all the different cavities, I shall at once meet the question, Is the lesion of Peyer's glands so constantly met with in typhoid fever, frequently, or is it ever, found in typhus?

Chomel (p. 339) appears to me to put the question in its true light. His third general conclusion is as follows: "If further observations demonstrate in typhus anatomical lesions similar to those met with in the typhoid affection, the identity of the two affections would be put beyond a doubt." To the philosophic caution of the inference of the French professor the following remark offers a strong contrast. A reviewer of various works on typhus and typhoid fever, in the 'British and Foreign Medical Review' for last year, after asserting (p. 432), that the symptoms during life are the same, and quoting a passage from Gauthier de Claubry (referring to the experience of four out of twenty-two observers who described the epidemics of army typhus during the wars of the Empire as decisive of the question), thus proceeds, "We do not know that any additional facts from the morbid

anatomy of the intestinal canal are required. In our own country we do not require any argument to persuade us of the identity of these supposed distinct diseases; the evidence is constantly before our eyes!" Montault, notwithstanding the assertion of his reviewer that his remarks are valueless, points out clearly the absence of the intestinal affection in typhus, and cites numerous authors to that effect. Roupell considers the absence of this lesion, which he fully admits, as accidental. Tweedie combats strenuously the opinions of Broussais (p. 57), and adduces the frequent absence of intestinal lesion as proof of their incorrectness. I remarked at the outset that, in the cases detailed by Dr. Tweedie, there were evidently two kinds of fever, the one distinguished from the other during life, by difference of intensity and duration, and after death by the lesions observed. In point of fact, I find thirteen cases in which ulceration of Peyer's glands had taken place, and nine in which there was no intestinal lesion whatever, but general congestion. Dalmas, in his account of what he heard and learned (not saw) at Dublin, cannot deny that the lesion is frequently absent; but only says, "it is perfectly well known" there. So it was in Glasgow, but even to me, a perfectly inexperienced observer, the distinctive symptoms were well known also. Gauthier de Claubry, evidently feeling the force of Chomel's remark, has laboured hard, throughout a very large portion of his work, to get rid of this very strong objection to his opinion. Reveillé Parise, on whose testimony he lays much weight, observed in those who died at Saragossa, "some gangrenous points in the interior of the intestines, and on the exterior, violet patches" (p. 18). Ducastaing (p. 16) found in many of those who fell victims at Gaëta in 1802, "the mucous membrane ulcerated in several points." Tresat, at Walcheren, in 1809, found "gangrene of some parts of the intestinal canal" (p. 19). Tort, at Dantzick, in 1813, found "the small intestine sprinkled with gangrenous eschars" (p. 23). Gilles de la Tourette, at Torgau, in 1813, found "serous and bloody effusions, inflammations, ecchymoses, gangrenous spots of the mucous and serous membranes" (p. 24). MM. Laurent and Ardy, Magnin, and Fauverge, mention inflammation, ulceration, gangrene in some points, violet

patches, scirrhous indurations of the mucous membrane of the small gut, as occurring in the Mayence epidemic of 1813, (p. 26–7). Thouvenel, in the department de la Meurthe, the same year, found "generally some gangrenous spots in several parts of the membranes of the intestines," (p. 35). In the epidemic of the Sâlpetrière in 1814, Pellerin, "in adynamic fever with diarrhœa, which," says Gauthier de Claubry, "he apparently believes a distinct disease in this circumstance," describes ulcerations that seem to correspond very closely with those found in typhoid fever, as "often," not always, met with.

After the foregoing investigations, are we not warranted in considering it, with Pellerin who witnessed it, "a distinct disease?" Chomel, also, with his customary caution, when speaking (p. 337–8) of the same epidemic, remarks that he "cannot place entire confidence in his recollections." If such disease as is daily found in the intestines of persons dying of typhoid fever, had been present, it could not, I should think, have been easily forgotten. Is it, then, really on such statements as these, on the existence of "violet stains" and "gangrenous spots" (which might, for aught we know to the contrary, have been the effect of decomposition, in a disease that often terminated with frightful rapidity), that Gauthier de Claubry and his reviewer would rest the decision of the question at issue? It was but yesterday that I saw, at M. Barth's rooms, large elliptical patches of ulceration in the jejunum of a female who had died of puerperal fever, with rupture of the uterus and peritonitis,—ulcers which, had they been found in a patient who had presented the ordinary symptoms of typhoid fever, would certainly have been looked upon as characteristic of the disease. Is, then, puerperal fever identical with the typhoid affection? I also saw a few days since, at M. Barth's, in a portion of gut taken from a patient who had, I believe, died of phthisis, large patches, of no determinate shape, extending around the whole calibre of the tube, the mucous membrane being in these parts immensely swollen, raised a couple of lines above the surrounding surface, traversed by deep rugæ, and having a greenish-black colour, with the characteristic gangrenous fœtor. Is it beyond the range of possibility that such may

have been the state of the intestines in the army epidemics referred to? Is it not rather highly probable that, where everything indicated a deeply altered and corrupted state of the blood, and local gangrene was of frequent occurrence, the gut as alleged by all the observers cited, may have been actually sphacelated? All that I have in view in these remarks is to demonstrate that, even were the existence of such lesions proved beyond a doubt, the alteration of the glands of Peyer, and the isolated follicles, now called characteristic, is very far from being proved thereby.

But are there no facts that bear against the reasonings of the French essayist? On the contrary, there are many. Lombard has graphically described his surprise on finding no lesions whatever in several cases he saw inspected while in Scotland and Ireland. We have seen the supposition Gauthier de Claubry has recourse to in order to explain away his testimony; and, even granting that "the circumstances of misery and exhaustion," in which, according to him, the people of Scotland are placed, do, as he supposes, produce a strong resemblance in the prevailing fever to army typhus (p. 143), does he advance one step towards a removal of the difficulty by gaining such an admission? Nay, rather, if M. Gauthier de Claubry has made out his position, that the intestinal lesion is present in typhus as well as in typhoid fever, whence this allusion to army typhus to explain the want of intestinal lesion—an allusion by which he demolishes the structure his work was intended to raise, and unwittingly discloses his own distrust in his own opinions? Again, M. Delbosc (cited at p. 38), in his account of the epidemic at Alby in 1823, mentions only "a rosy colour of the peritoneal tunic," and M. Keraudren and the Toulon physicians, "though their attention was specially directed to that point by the questions addressed to them by the Academy of Medicine,[1] never observed any intestinal lesion in the typhous epidemic at Toulon in 1829–30. I have already referred to Dr. Tweedie's data, and have cited M. Dalmas's silence as impartial evidence, though in the United Kingdom very few require evidence to convince them, that in Dublin, as elsewhere, a form of fever exists, of highly malignant character,

[1] Chomel, p. 338.

and most extensively fatal, in which no intestinal lesion is found even on the most careful inspection. Dr. Alison, of Edinburgh (from whom I cannot quote accurately, not having access to his work), corroborates the experience of other observers.[1]

I now proceed to give some more circumstantial details upon this interesting subject. Dr. West[2] gives the results of ten inspections. In five (I merely speak of the state of the bowel) "there was no morbid appearance whatever;" in the other five "there was increased vascularity of the intestinal canal, extreme in one instance." "Once the glands of Peyer appeared enlarged, and twice there was very considerable enlargement of the solitary glands; but I never," says he, "found them ulcerated. Once the mucous membrane of the cæcum was very much softened and congested, and there was slight abrasion of the surface of some of the congested patches." Dr. Reid[3] states, that of 101 cases examined in the Edinburgh Infirmary, by his predecessor, the late Dr. Home, "the elliptical patches were well defined in 29; they were more or less ulcerated in 7 of that number and in 2 out of the 7, perforation had taken place." He next gives[4] a summary of the morbid appearances in 41 cases inspected by himself. In 24, "Peyer's glands were apparent and distinctly defined;" in 6, "scarcely visible;" in 11, invisible to "the naked eye." In 4 only were they "distinctly elevated;" and in 2 of the 4, "this elevation was to no great extent, and limited to a few patches." In 2 only was there "any appearance of ulceration." In 4, the solitary glands were "distinctly visible." Regarding the connection of the symptoms during life with the post-mortem lesions, he says,[5] "In 9 cases only out of the 24, in which the elliptical patches were distinctly visible, were there any abdominal symptoms during life, and in some of those

[1] Dr. Alison has published no separate work, but Observations, &c., in the twenty-eighth volume of the 'Edinburgh Medical and Surgical Journal,' p. 233.—Ed.

[2] 'Edin. Journal,' vol. l, p. 132–3.

[3] Report, &c., p. 33.

[4] Ibid., pp. 30, 31.

[5] Ibid., p. 34.

cases these certainly could not be referred to any affection of the elliptical patches of Peyer." "In one case in which they were not visible, the bowels are stated to have been rather loose, the stools watery and dark coloured. In other two cases there was considerable abdominal tenderness, without diarrhœa."

I shall preface with a few remarks the two following and concluding tables, which give the results of my own observation. They refer to twenty-two inspections,—but a small number, it is true, yet made with as great care and attention to accuracy as I could bestow on them. The solution of the interesting question, Is there any connection between the abdominal symptoms during life, and the state of the intestinal follicles in typhus? may be attempted in different ways. We may compare the symptoms, whether positive or negative, with the mucous follicles, as regards (1) their number, and (2) their degree of development. And after having, as far as our data enable us, determined these points, we must not be unmindful of the objections that may be urged, as, for instance, that our terms are vague; that our cases are too few to draw conclusions from; that absorption of morbid matter may have taken place before death, or, it may be, that death has arrived before its deposition was possible. I shall meet some of these objections beforehand. The cases, I acknowledge, are few, but I maintain that they are a fair sample of hundreds that occur yearly, and at the same time direct attention to the numerous facts I have already cited. To take away all vagueness from the terms I employ, I make the following statements respecting the state of the intestinal follicles. It was very rare, in the cases of typhus which I either inspected or saw inspected, during a two years' residence in Glasgow Infirmary, to see these glands elevated a quarter of a line above the surrounding mucous membrane, and in the vast majority it was very difficult to determine whether they were really elevated or not, the slight roughening or irregularity of their surface often causing a visual deception. When, therefore, we speak of the elevation of the aggregate glands in typhus we are no longer occupied, as in typhoid fever, in the measurement of palpable magnitudes, but for the most part with elevations, which French pathologists,

as I have frequently witnessed, scarcely reckon any deviation from the healthy state. To avoid overlooking, not one, but many, of these (enlarged?) glands often requires very minute attention. The other objections I shall leave to be answered by the facts about to be adduced, only remarking that the removal by absorption of the morbid matter from the follicles is a very rare termination, even in typhoid fever. Chomel mentions two cases in which that process had probably taken place,[1] and I cannot see why it should be more common and more speedy in typhus. The first of the following tables refers to the number of follicles met with in the small gut; the second to their state of development.

No. of enlarged follicles.	Cases.	Diarrhœa.		Constipation.	Tympanites.			Abdominal pain.			State of follicles.			
		Spontaneous.	Consecutive.		Slight.	Considerable.	None.	Slight.	Acute.	None.	Elevated.	Very slightly so.	Not elevated.	Scarcelyseen.
None .	1	1	—	—	1	—	—	1	—	—	—	—	—	—
1 to 5	4	2	1	1	1	1	2	1	2	1	—	—	—	4
5 to 10	4	—	3		1	1	2	4	—	—	—	2	—	2
10 to 15	4	1	1	2	1	2	1	—	3	1	—	2	2	—
15 to 20	3	—	2	1	—	2	1	—	3	—	—	3	—	—
20 to 30	4	—	1	3	—	—	4	2	—	2	1	1	2	—
30 to 40	1	—	1	—	1	—	—	1	—	—	1	—	—	—
40 to 50	1	—	1	—	1	—	—	—	1	—	—	—	1	—
	22	4	10	8	6	6	10	9	9	4	2	8	5	6

Intestinal follicles.	Cases.	Diarrhœa.		Constipation.	Tympanites.			Abdominal pain.		
		Spontaneous.	Consecutive.		Slight.	Considerable.	None.	Slight.	Acute.	None.
Distinctly elevated in .	2	—	1	1	1	—	1	2	—	—
Very slightly elevated .	8	1	4	3	1	3	4	3	4	1
Not elevated . .	5	—	2	3	2	1	2	—	3	2
Scarcely seen . .	6	2	3	1	1	1	4	4	1	1
	21	3	10	8	5	5	11	9	8	4

[1] Observ. 14 and 15, pp. 169, 173.

Thus we see that 4 only of the 23 in whom diarrhœa was spontaneous rank in the above list. In one of the 4, the diarrhœa depended on an attack of simple enteritis, which came on after partial convalescence, and carried off the patient on the 34th day. No trace of follicular disease was found on inspection, but "universal softening of the mucous membrane from the duodenum to the rectum." Two of the remaining 3 may almost be said to have presented no trace of follicular disease, for the three or four follicles observed in each were barely perceptible to the naked eye. In the fourth, fourteen Peyer's glands, scarcely at all elevated, were detected. But what chiefly concerns us to notice is the striking fact, that, as the number of enlarged follicles increases, the cases of spontaneous diarrhœa diminish, and those in whom consecutive diarrhœa and costiveness were observed become more numerous. In fact, not one of those in whom the greatest number of enlarged follicles was observed, but was either constipated, or had diarrhœa brought on by medicine, during life. This confirms our former deductions, agrees entirely with those of Valleix, and shows that the appearances observed in typhus depend upon local irritation and not on specific disease.

It will also be observed that in 10 of the 22 (including 5 of those in whom from 15 to 30 enlarged glands were observed) the feel of the abdomen was natural throughout the disease. Of the 6 in whom the belly was slightly tympanitic, that symptom was observed only one day in four patients (the one in whom the greatest number of enlarged follicles was found being among them); in one with forty enlarged glands, it was present during two days; the sixth was the patient who died of enteritis. Of the six, on the other hand, who presented a high degree of tympanites, in not one were there so many as twenty, and in one whose abdomen was excessively distended scarcely a trace of follicular disease was detected.

There seems, at first sight, to be more connection between the presence of abdominal pain and the number of altered glands, but this is only apparent, for a reference to the figures given above shows that, in 8 of the 9 in whom the pain was acute, not more than twenty were found in the

whole course of the gut; that, of the 9 who complained of slight pain they were either not at all, or very slightly affected in 6; and that, if 2 of those in whom they were noticed in large numbers (twenty to thirty), had slight pain, 2 more of the same group had no pain at all, at any period of the disease. The concluding part of the table proves that quite as little analogy exists between the number of follicles and their degree of development. As a fitting commentary on what I have now advanced, I find that in the only case in which considerable elevation of Peyer's glands (about half a line), and a few ulcers (not follicular) in the cæcum, were observed, the bowels were obstinately constipated, the feel of the belly was natural throughout, and the abdominal pain was either exceedingly slight or absent, till within a few hours of death, having come on after the administration of a clyster, composed of equal parts of turpentine and castor-oil.

These remarks are still further confirmed by a reference to the second table, in which the symptoms are considered with respect to the degree of development of the follicles. The same dependence of the intestinal lesion on local irritation, the same entire disproportion between the extent of the lesion and the intensity of the symptoms, are brought out so clearly by it as to render any further reflections needless. The utter futility of the objection founded on the possible absorption or non-deposition of morbid matter, will appear from the following facts. Comparing the number of follicles with the days of death, I find that, of those belonging to the first group, one died on the 12th, another on the 13th, a third on the 14th, and a fourth on the 16th day. Of the second group, two died on the 12th, another on the 17th, and a fourth on the 36th day. Of the third, one died on the 10th, one on the 14th, the other two on the 21st and 24th days. The three belonging to the fourth group died severally on the 13th, 15th, and 19th; those in the fifth, on the 10th, 11th, and 13th, the two in the 6th and 7th, respectively on the 11th and 22nd days of the fever. Thus, then, of two who die on the same day of the affection, one presents scarcely a trace of disease, another as many as thirty enlarged glands. In one who dies on the 11th day, no less than forty Peyer's

glands are observed, while in others who die on the 12th, 13th, 14th, and 16th, scarcely any morbid appearance is detected; in another, who dies on the 22nd, no less than forty-five, while in a seventh, who dies on the 24th, only ten or eleven Peyer's glands are obscurely visible.

Again, if we consider the development of the glands, in reference to the day of death, we find that, of the first group, one died on the 10th, another on the 11th day; of the second, one on the 10th, another on the 11th, a third on the 13th, a fourth on the 15th, and the remaining four, respectively, on the 17th, 19th, 24th, and 36th days; of the third, one on the 11th, another on the 13th, a third on the 14th the remaining two on the 21st and 22nd days; of the fourth, three died on the 12th, and the remaining three severally on the 13th, 14th, and 16th days. Thus two die on the 10th and 11th days with Peyer's glands distinctly elevated (in one of them to the extent of about half a line); they are scarcely discernible in four who die on the 12th and 13th days; while they are in a state of slight elevation in eight, who die on all different days between the 10th and the 36th. Does this, I ask, look like the steady course of a constant lesion, depending upon a constant and specific cause?

The solitary glands were visible, in small number, in three cases; in one, in the small, in two, in the large intestines. I have, however, seen them very numerous, both in the one and the other, though they were never larger, and seldom so large as a pin's head. They were white, sometimes surrounded with a vivid red areola, and each had a small black point in its centre. The aggregate glands, situated on the free border of the gut, were of all sizes from two or three lines to two or three inches in length, and varied from a couple of lines to nearly an inch in breadth. They were sometimes white, sometimes greyish, sometimes slate-coloured, frequently dotted over with small black points, often reticulated, while sometimes neither the one nor the other appearance was noticed. It is a remarkable fact that, in several cases, the glands were much more numerous in the jejunum than the ileum, and that in two there were none near the ileo-cæcal valve. Not unfrequently, the mucous membrane around them was pale, at other times they were situated amid

patches of very dark congestion, in which case they partook more or less of the dark colour of the surrounding membrane. Sometimes, but very seldom, the mucous membrane itself was reddened, and injected with minute vessels; the congestion of the veins of the submucous cellular tissue was, on the contrary, very common.

No one denies the very frequent affection of the mesenteric glands in typhoid fever. The experience of every one who has seen the inspections of patients cut off by that disease must confirm the facts stated by Chomel, Louis, and Petit and Serres. Though very rare in typhus, it is nevertheless sometimes found. I find it noted by Dr. Tweedie in two cases (1 and 44). In the former, "the lungs and abdominal viscera were healthy, excepting the mesenteric glands, which were enlarged and partially affected with suppurations of a scrofulous character" (p. 108). In the latter, "there was no ulceration of the intestines, but the glands of the mesentery were enlarged." Of my own cases, the mesenteric glands were enlarged in four; in one very slightly, in two in a greater degree, in a fourth to a large extent, but not suppurated. In the first, death took place on the 11th day, and twenty or thirty Peyer's glands, not at all elevated, were found in the small gut; one of the next two presented eight very slightly developed, the other none at all; and in the fourth, there were nineteen very slightly elevated. In all of those, then, in whom the enlarged follicles were very numerous, including the only patient in whom they were considerably elevated, the mesenteric glands were unaffected.

I have been enabled, by the kindness of Dr. Reid, to present the reader with a much larger mass of information. From the publication of the Report till the end of June, thirty-three cases of fever were inspected in the Edinburgh Infirmary. Of that number only two presented the characteristic lesion of the intestinal follicles met with in typhoid fever. In both of them the lardaceous deposit (*plaque gaufrée*) was most distinctly seen, and both the aggregate and solitary glands were affected in great numbers, the latter forming the whitish "flattened tumours" so well described by Louis. Neither of the patients belonged to Edinburgh; both were workers on the Edinburgh and Glasgow Railway,

and were sent from Linlithgow. If, then, dothinenteritis is the same disease, and depends on the same causes, as typhus, how does it happen that the former is produced at Linlithgow and at Anstruther, and not in Edinburgh, where the latter is constantly occurring, and where so many circumstances favorable, not only to its production, but (by the hypothesis) likewise to that of dothinenteritis, are always at work. The duration of these cases is also remarkable. The one, it appears, had been four, the other five, weeks ill. One of them had convalesced, but had a relapse, of which he died; and, on dissection, the intestinal follicles and mesenteric glands were the only parts diseased, so that the relapse cannot be referred to any lesion of any other organ. It is further remarkable that the aggregate glands were in a state of advanced ulceration, while most of the solitary glands around them were quite entire. In the remaining 31 cases—

Peyer's glands were distinctly elevated, but not ulcerated, in	4
" " visible, but not elevated, in	9
" " scarcely visible in	7
" " not visible in	11
	31

The solitary glands were slightly developed in two of the above cases. The mesenteric glands were healthy in 25, slightly enlarged in 4, considerably enlarged and softened in 1, and in a 6th increased in size, and partially converted into chalky matter. Dr. Reid also informed me that, in a man who died lately of the effects of compound fracture, Peyer's glands were more distinctly diseased than in any case of typhus he had met with.

Having thus entered pretty minutely into the pathological anatomy of typhus, I put it to every one who has ever seen the two diseases, or read the descriptions given by the best authors, of the lesions so constantly observed in typhoid fever, whether those found in the former disease can with any truth be called "perfectly identical" with those of the latter. That the existence of trifling intestinal disease in typhus attests its general family resemblance to typhoid fever, as the presence of organs in a rudimentary state shows the analogy subsisting between individuals of different species

in the animal kingdom, might be more readily admitted; but that they are one and the same species, numerous well-established facts seem most clearly to disprove. If asked to describe shortly the pathology of typhus I might sum it up in these words—general congestion, no prominent local disease—a congestion so general and so excessive as is rarely, if ever, met with in typhoid fever or any other disease—a congestion singled out by most authors as one of its leading characteristics—a congestion that is evident, during life, by the livid skin and petechial eruption, and is found, after death, to have affected more or less every organ in the animal economy—a congestion so constant as to be often passed over as almost valueless, but which future researches may prove to be the grand peculiarity of typhus; and which, in common with many other considerations, directs attention to the blood as the essential seat of the disease. If required, on the other hand, to give a brief account of the pathology of typhoid fever, I should be inclined to sum it up in these words—prominent local lesion, comparatively little general congestion.

VI. From the treatment of the two diseases we can infer but little. The interesting discussions held at various times on the subject in the Académie de Médecine,[1] strikingly show the discordance of sentiment that prevails in France on the treatment of dothinenteritis. One upholds the purgative system as the best and the only proper one, another condemns it as fraught with the greatest danger; one insists that the rational method, which consists in combating symptoms as they arise, is the only rational one, but another condemns it as most irrational, because it kills one in three; one strongly recommends the frequent use of the lancet, according to his formula, even in advanced stages of the disease; while another, condemning not only the lancet, but all treatment whatever, advocates a method in the purest sense expectant, and unlimited faith in the workings of nature, any attempt to interfere with them being attended with evil results. And every one has facts on his side.

[1] See more particularly those on Delarroque's Memoirs in the 'Bulletins de l'Académie' for March 14th, 21st, and 28th, and April 4th and 11th, 1837. Also Bouillaud's statements at vol. i, p. 250.

The most remarkable statements, perhaps, of any, are those of Professor Bouillaud, who, employing his "bleeding formula" (viz. small bleedings repeated two or three times daily, sometimes even during the adynamic stage, when the patients presented "fuliginous teeth and tongue, and the last degree of prostration"), and including only those cases that were somewhat serious, found a mortality among his patients of one in 6·5.[1] Again, at one of the meetings in March, 1837,[2] he declared, that, "counting all the cases, he had lost only one in 24, and excluding all the slight cases, one in 16 or 17." Who that is acquainted with typhus, and has seen the sudden and alarming asthenia that often follows the abstraction of a few ounces of blood during a state of great excitement and oppression, but must feel convinced that, treated after M. Bouillaud's method, 99 out of every 100 would die? Gauthier de Claubry, while he concludes, from his own experience of typhus, and the statements of numerous distinguished authors whom he cites that "bloodletting (p. 153) may be useful, if not absolutely indispensable in certain cases, indifferent in a great number, hurtful in many others" (an opinion he shares with Pringle, Hildenbrand, and the immense majority of British physicians), confesses (p. 167) that the general opinion is in favour of its use in typhoid fever; and besides referring to the constant employment of it in the beginning of typhoid fever by Louis and Chomel, states, that "twenty years of an extensive practice, and of observations made in the hospitals of a large city, as also a comparison of the results of the private practice of a great number of physicians," have led him to adopt a similar treatment. As to the use of purgatives, I may state that while in Glasgow they were generally used in typhus, the marked difference in the state of the bowels in typhoid fever led to the general use of opium, either alone, or combined with mercury and chalk. All, in short, that we can infer from the practice at present in use is, that the treatment generally proscribed in the one is generally adopted in the other disease.

On a review, then, of all that has been advanced, it would

[1] 'Séance de l'Académie,' October 16th, 1835.

[2] See 'Bulletins de l'Académie,' vol. i, p. 520.

appear that typhus and typhoid fever present important differences, as regards their probable origin, their proximate causes, their course, many of their symptoms, their diseased appearances, and the treatment applied in each. Are they then identical, or are they not? I feel that it would be presumptuous in me to hazard a direct reply; nor do I demand an answer in the affirmative merely on the faith of what I have stated. All I can ask or wish for is careful, extensive, and minute inquiry, without prepossession or love of system, and a satisfactory solution must soon be arrived at.

"Notwithstanding all the works," says Chomel (p. 338), "which the last few years have produced on the typhoid affection, its identity with typhus, though probable, is not yet certain; and yet there are few questions of which the solution would be so highly important." I have accordingly attempted in the preceding pages to concentrate attention, as far as lay in my power, on the leading peculiarities of the two affections; I have endeavoured, by frequent reference to the works of approved authors, by collecting together valuable facts hitherto widely scattered, and by adding the results of my own observation, to bring as large a mass of information as possible to bear on the question in debate; I have attempted—with what success others must judge—to establish certain important distinctions between the two diseases; I have particularly sought to found my conclusions on practical knowledge rather than theoretical reasonings; and if I have contributed towards the elucidation of this obscure subject, or shall be successful in drawing the attention of abler and more experienced observers to its investigation, my trouble is more than repaid.

MEMOIR OF DR. A. P. STEWART.

Dr. Alexander Patrick Stewart was born at Bolton, East Lothian, on August 28th, 1813. He was the son of the Rev. Andrew Stewart, M.D., the minister of the parish, who before entering the ministry had acquired considerable reputation as a physician, especially by his treatment of consumption. His mother, Margaret, was the daughter of the tenth Lord Blantyre. He received his general education in the Faculty of Arts of the University of Glasgow, where he studied in the class of Sir Daniel Sandford, and was distinguished as a good Greek scholar. From 1828 to 1830 he travelled with his family on the Continent, and acquired a thorough knowledge of the French language. On his return he entered as a student of the Medical Faculty of the University of Glasgow, and graduated as Doctor of Medicine in 1838. He held the appointment of House-Surgeon at the Glasgow Infirmary, where Dr. Perry was one of the physicians. And it was here that he made those observations which convinced him of the specific distinction of typhus and typhoid fever. After leaving Glasgow he pursued his studies for about eighteen months at Paris and Berlin. He then, in 1839, settled in London where he continued to practise till his death. In April, 1840, he communicated the results of his researches on fever to the Medical Society of Paris, and his paper, the present memoir, was published the same year in the fifty-fourth volume of the 'Edinburgh Medical and Surgical Journal.'

In 1850 Dr. Stewart was appointed Assistant Physician, and in 1855 Physician, to the Middlesex Hospital, where he also held in succession the Lectureships of Materia Medica and Medicine. In 1866 he retired from the Middlesex Hospital and was subsequently elected Consulting Physician. At the Middlesex Hospital Dr. Stewart was remarkable for the minuteness and accuracy of his observations and his great kindness to the patients.

He was elected a Fellow of the Royal College of Physicians of London in 1855 and an Honorary Fellow of the King's and Queen's College of Physicians of Ireland in 1867.

In 1850 he became a member of the British Medical Association, and continued during the rest of his life to take an influential part in its proceedings. He was Secretary of the Metropolitan Counties Branch from 1858 to 1874, and was for twenty-nine years a member of its Council. He was also a Vice-President and member of the General Council. On his retirement from the office of Secretary the members presented him with a testimonial, which with characteristic generosity he applied to the formation of a fund for the recognition and encouragement of researches into the origin and spread of epidemic disease.

In connexion with the Association Dr. Stewart took an active part in promoting medical reform, and gave much time and attention to sanitary questions. He published several papers on medical and sanitary subjects, among which may be mentioned one on Cholera in the Middlesex Hospital, which appeared in the 'Medical Times and Gazette,' in 1854. And one on the Workings of Some Provisions of the Laws relating to Public Health, which was read before the Metropolitan Counties Branch of the Medical Association in 1867, and afterwards published in a separate form. But by far his most important work, and the one on which his reputation mainly depends, is the present treatise.

Dr. Stewart was a man of much, though intermittent,

energy, of acute observation, of clear ideas with great facility in their expression. His high integrity, his warm heart, and his genial disposition, caused him to be universally beloved and respected. His religious convictions were strong, and he was an earnest member of the Presbyterian Church in which he held the office of an Elder, and in connexion with which he devoted much time to religious and charitable works. His life may be said to have been thoroughly consistent with the principles he professed.

Though in consequence, perhaps, of a certain innate deficiency in habits of business-like punctuality, he never attained to that leading position in practice which his high qualities merited, he has left behind him an unblemished reputation, and his name will always be associated with one of the great discoveries of medicine.

Dr. Stewart, who was never married, died at his house in Grosvenor Street July 17th, 1883, having nearly completed his seventieth year.

Much controversy has taken place as to whom the discovery of the distinction between typhus and typhoid fever is to be attributed, and as to the share Dr. Stewart had in determining this question; but, as so often happens in disputes as to priority, it will be found on impartial investigation that the discovery was arrived at gradually, successive observers adding fresh links to the chain of argument till at last the conclusion was securely established.

Although it cannot be claimed for Dr. Stewart that he was the first to make out the distinctions between typhus and typhoid, nevertheless at a time when the great majority of observers regarded them either as the same disease or at most only well-marked varieties, his own observations, made at Glasgow from 1836—38, and at Paris in 1839, convinced him of their essential distinction. And in the present memoir he demonstrated this more fully and conclusively than had been done before, at least in this country; for in

America Messrs. Gerhard and Pennock, in a paper published by Dr. Gerhard in the 'American Journal of Medical Science' in 1837, had fully described all the more important points of difference, and perhaps they have the strongest claims to be considered the first to have clearly proved the complete distinction between the two diseases. Nevertheless, all the leading facts and conclusions had been established before by gradual steps.

The distinction between the slow nervous fever and the putrid jail fever had been observed in England since early in the eighteenth century. In the middle of the century De Haen described accurately the rose rash of typhoid. And Pringle, in his controversy with him, pointed out its difference from the petechial rash of the jail fevers, and also the different class of persons liable to be affected by the two forms. In 1810 Hildenbrand, who regarded the two diseases as entirely distinct, showed how they differed in contagious properties. The essential lesions of typhoid fever were gradually established by French pathologists, especially Bretonneau, who invented the term dothienenteritis, though he regarded the disease as a specific fever, and not as Broussais had done, a mere inflammatory affection. In 1829 Louis gave the name of typhoid to the dothienenteritis of Bretonneau, but in France it was generally regarded as identical with the typhus of England, and in England dothienenteritis as an occasional complication of typhus.

In 1835 Dr. Peebles, who was familiar with the contagious typhus of Italy, pointed out its characteristic rash to Dr. Perry, of Glasgow, Dr. Stewart being present, and from this time Dr. Perry taught the difference between contagious typhus and dothienenteritis, and he appears to have been the first in this country to have done so; though in his paper, published in 1836, he does not seem to have regarded dothienenteritis as a specific fever accompanied by a rash of its own, but as an affection which might complicate other diseases as

typhus and smallpox, or occur independently. Subsequently he appears to have recognised and taught the distinction between the two rashes.

In 1836 Dr. Lombard, of Geneva, who had studied fever both on the Continent and in this country, published a paper in the 'Dublin Medical Journal,' in which was maintained for the first time that two separate and distinct fevers occurred in Great Britain, one contagious typhus and one identical with the dothienenteritis or typhoid fever of the French. But he did not discriminate between the eruptions and symptoms of the two.

The progress of these doctrines, notwithstanding the conclusive arguments of Lombard, Gerhard, Shattuck, H. C. Barlow, Stewart and others, was very slow, and in this country it was not till the publication of Sir William Jenner's papers in 1849, 1850, and 1853, that they obtained anything like general acceptance.

On the whole, from this very brief *résumé* of the question, I think it will be evident that to no one observer can any exclusive merit be ascribed. To Dr. Stewart belongs the credit of being one of the first to see clearly the truth and to have supported it by arguments which appear now to be absolutely convincing.

MOVEABLE KIDNEY IN WOMEN.

BY

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WITH NINE WOODCUTS.

TRANSLATED AND EDITED WITH NOTES BY

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TRANSLATOR'S PREFACE.

The scope and interest of the ensuing work are by no means expressed by the limited description on the first page. Dealing professedly with a single abnormal condition, it includes a great portion of the anatomy and physiology of the abdominal cavity, and some of the pathology of the pelvis. Interesting as such a work must be to every medical man, it is doubly so to the physician, the focus of whose work lies in the pelvis and lower abdomen, and it is a welcome addition to his knowledge at a time when the subject of uterine displacement seems to be approaching a more rational estimation.

The scientific value of the treatise is great; its literary value would have been greater had some of the references and all the quotations been revised. The latter having been found almost invariably inaccurate, are given in this translation direct from the original sources.

The Translator, who has found the usual difficulties in the way of producing a version at once idiomatic and precise, begs to assure his readers that any faults which may appear are due to anything rather than want of care.

London, 1884.

PREFACE.

THE significance of moveable kidneys in their pathological and therapeutical relations has not yet been rendered sufficiently clear in spite of many valuable works.

It has been in part wrongly interpreted, in part overlooked. By most persons it is regarded without serious clinical interest as an anatomical curiosity, the diagnosis of which leads to nothing further than the relief of the patient from the fear of a presumably serious malady. A view directly opposed to this has been lately promulgated, namely, that moveable kidney is a disease dangerous to life, which must be removed as quickly as possible by extirpation.

In the presence of this difference of opinion it is worth every doctor's while to obtain clear views on this disease, so often seen and so often mistaken. For the gynæcologist especially, the knowledge of this malady is all the more important since it attacks women by preference and stands in a far more intimate relation to affections of the female sexual organs than is commonly supposed.

The observation of numerous cases of moveable kidney has led me to conclusions differing in many respects from the views hitherto entertained, and I have thought it advisable to embody them in a systematic treatise on this disease.

LANDAU.

BERLIN;
July 16, 1881.

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MOVEABLE KIDNEY IN WOMEN.

I. History and Literature.

Although the *congenital* alterations in the position and form of the kidney had at an early date excited the attention of medical men, especially the anatomists, *acquired* moveable kidney remained long unknown. *François Pedemontanus* (1) is the first who speaks of a dislocation of the kidney arising from internal and external causes (percussio), without, however, attaching special importance to this anomaly.

Riolan (2) was the first to set forth the *clinical* importance of the disease to which he gave the name of dislocation of the kidney. His description of this disease is far too characteristic for me to neglect to repeat it in this place: "Quamvis renes adipis glutine videantur tenaciter affixi lumbis, interdum tamen luxantur et antrorsum procumbunt; interdum in hypogastrium delabuntur, *non sine vitæ detrimento;* hoc ita verum est, ut nullo modo sit dubitandum. Id potissimum accidit, non tantum liquata pinguetudine, qua sunt obvoluti, sed etiam ex pondere, ubi tam grandes sunt, ex tumore vel calculo in cavitate concluso, ut suis retinaculis in sua sede contineri nequeant, tumque ibi aliquamdiu subsistunt, sed tandem *putrescunt et abscessum patiuntur*. . . . (Renes) comprimunt psoam et nervos ad crura descendentes. . . . [1]Si vena reseretur, aut rumpatur, urinæ cruentæ funduntur: et quoniam renes communicant per nervos stomachicos cum ventriculo, *eorum affectibus condolescit* aut compatitur nauseabundus aut vomituriens."

[1] This sentence is incorrectly quoted by Landau.

Notwithstanding this suggestive description, certainly founded on excellent observation, the disease, "moveable kidney," remained entirely unknown till the beginning of the present century or was regarded only as an anatomical curiosity, even by Haller (3), for instance, who, in the case of a woman who had died from dropsy and had suffered during life from a tumour above the navel, found the kidney filled with water in the lower part of the abdominal cavity.

Baillie (4) mentions a tumour which he had observed four or five times, loose, in the region of the kidney of one or other side, which could be moved upwards and downwards by slight pressure with the hand, and which was pretty firm, and generally had the shape and size of a kidney. The patients concerned were very little inconvenienced and their general health little if at all disturbed. In women, as Baillie mentions, the tumour was often mistaken for an enlarged ovary; but it had not its shape and could not, moreover, be felt in the situation in which such a body can be usually felt. The author in question had no opportunity of making an autopsy, and was doubtful as to the nature of the tumour, but was inclined to regard it as a moveable kidney.

The contributions on this affection by Otto (5), Meckel (6), and Portal (7), which were certainly very imperfect, remained entirely unnoticed.

The first accurate post-mortem records associated with clinical descriptions were furnished in four cases by Aberle (8). He was followed by Girard (9) and King (10), who each observed one case of moveable kidney. King himself made an attempt even at that early date to remove the moveable tumour, but failed to find it after opening the abdominal cavity. The woman concerned felt herself, strange to say, better after this abortive attempt to extirpate her kidney than before.[1]

But a somewhat more general acquaintance with this malady dates from the publication of the excellent work on kidney diseases by Rayer (11), who himself observed several cases of moveable kidney. Then followed the publication of

[1] A similar relief from symptoms is not unknown after other unsuccessful essays in abdominal surgery.—TRANSLATOR.

single cases by Braun (12), Brochin (13), Urag (14), Petters (15), Oppolzer (16), Hare (17), and Henoch (18), so that Fritz (19) in 1859 was already able to collect 35 cases from the literature of the subject.

The knowledge of the clinical symptoms of moveable kidney was next substantially enriched by Dietl (20), who pointed out the frequency of moveable kidney and particularly of a chain of symptoms caused by it, which had been till then but little regarded. It is true that Dietl only considered this complaint as one which occurred with particular frequency in the Polish race.

Stress was laid by Becquet (21) on the intimate relation between the sexual life of the female and the production of moveable kidney, while Chroback (22) referred to the connection between hysteria and mobility of the kidneys.

We are indebted to Rollet (23) for the first monograph on the pathology and therapeutics of this affection. In 5500 patients of Oppolzer's clinique this author found 22 cases, and described 10 of them. He himself mentions that this number does not nearly represent the total of those actually present, since some of these patients only present themselves in the out-patient department (Ambulatorium) without being admitted to the wards; some to whom this affection causes no special discomfort, never come under medical examination; and, finally, the frequency with which cases occur in the wards is influenced by deliberate choice of the cases admitted.

Among more recent works must again be particularly mentioned the important publications of Durham (24), Lancereaux (25), Trousseau (26), Guéneau de Mussy (27), Fourrier (28), Henderson (29), Howitz (30), Oerum (31), and Keppler (32).

Isolated cases have been published by Gueterbock (33), V. Dusch (34), Keckeis (35), Wilks (36), Edwin Day (37), Gilewski (38), Ehrle (39), Mosler (40), Drysdale (41), Schultze (42), Steiger (43), Pieper (44), Wiltshire (45), Schiff (46), Heslop (47), Flemming (48), Gontier (49), Thun (50), Ferber (51), Mac Evens (52), Herr (53), Tzschaschel (54), Jago (55), Klüpfel (56), Peebles (57), Defontaines (58), Grout (59), Cabarellas (60), Kovatsch (61),

Hertzka (62), Le Ray (63), Stiller (64), Pitois (65), Schenker (66), and Hunter (67).

Lastly, attention must be called to the appropriate sections in the handbooks on kidney diseases by Rayer, Vogel, Rosenstein and Ebstein.

II. Definition and Nomenclature.

By the name wandering kidney is understood that pathological change in the position of the kidney under which it is permanently or temporarily removed from its normal situation and manifests a greater or lesser amount of mobility. This anomaly has been also called "Moveable kidney," "Descent," "Displacement," "Prolapse," "Ectopia" of the kidney. The older authors speak of "Dislocation of the kidney," and, from the analogy of dislocations of the limbs, of "Spontaneous" and "Traumatic" dislocations. The French sometimes use the phrase "rein flottant," the English "floating" or "moveable kidney."

A moveable kidney may become fixed in an abnormal situation and is then called "fixedly dislocated" (fix dislocirt). Kidneys which are dislocated and fixed are mostly congenital anomalies (for instance the so-called "horse-shoe kidney," and the "kidney lying deep down in the pelvis"), and are combined with other malformations, such as abnormal positions of the intestines and low insertion of the vessels. These congenitally dislocated kidneys, which are only in rare cases found moveable, and may now and again indeed give rise to disorders of labour, are generally unimportant clinically, and, having already been often sufficiently described, will not in this place be further considered.

III. Topographical Anatomy.

With a view to the better comprehension of the pathology of acquired moveable kidney, it seems advisable, as in the case of descent and prolapse of the female genital organs, to direct our special attention to the normal position of the

kidney and the forces which maintain it in this position. The accounts of the old anatomists, which have mostly found their way into the modern text-books, give only imperfect explanations on both these points. At one time pathological conditions have been taken for normal, at another too little regard has been paid in the treatment of many kidney diseases (which was till lately considered hopeless) to certain intimate anatomical relations between them and the neighbouring organs.

Thus, Haller (68) says that the kidneys may lie on both sides of the vertebral column alongside of some four vertebræ between the eleventh (thoracic) and fifth lumbar, while Vogel (69) says that the kidney in its normal situation may project as far as the iliac crest or even farther into the false pelvis.

Among recent productions may be named the valuable works of Rayer (70), the excellent representation of the topography of the abdominal cavity by Sappey (71), Luschka (72), and Rüdinger (73) ; and the works of Pansch (74), and His (75). These works, as well as investigations on the dead subject, have been followed in the ensuing description.

Position of the Kidneys.

The kidneys lie against the posterior wall of the upper part of the abdomen, and are partly within the bony cavity of the thorax close to the transverse processes of the vertebral column, their inner border having an average distance of seven and a half centimetres from the middle line. They extend on either side from the lower border of the eleventh thoracic vertebra to the lower border of the second lumbar vertebra. Pansch is correct when he speaks of an "average" position of the kidney, for its boundaries are not absolutely fixed, the variations from the average boundaries are, however small, and differences of even the height of a vertebra are to be considered pathological.

The majority of authors say that the right kidney lies lower down than the left. Thus Bauhin[1] (76) says: "Horum dexter proxime Hepati subiacet propter eius molem

[1] Incorrectly quoted by Landau.—TRANSLATOR.

in homine altero est inferior, ipsi tanquam nobiliori cedens, et ad tertiam lumborum vertebram suo fine descendit." Spigel[1] (77) says: "Incumbunt renes sima sui parte musculorum, quos ψόας Hippocrates vocavit, . . . capitibus. . . . Inaequalis etiam utriusque positio est, cum laevus dextro elatior sit, non tamen dimidia sui parte."

These authors are followed by those of later date in ascribing the cause of the lower position of the right kidney to the liver. Few only, as Legendre, Luschka, Rüdinger, Sappey and His, assert that both kidneys are nearly on the same level. Pansch (78), indeed, found the left kidney slightly lower than the right in every third subject. Certain it is that the difference of level between the right and left kidney, if any difference exists at all, is but trifling, as observations made on the cadaver make certain. The less regard need be paid to the *influence of the liver* on the position of the kidney, since, as recent investigations by Braun (79) and His (80) show, the liver is normally so extremely soft (like fat or cellular tissue) that it yields to the movements and displacements of neighbouring organs. The flexibility and softness of "fresh liver-tissue" is so great that it is much nearer the truth to say that the shape of the liver depends on the volume of adjacent organs, as is shown by their impressions on it (faciecula renalis, &c.), than to say that the liver is capable of regulating their shape. I lately had an opportunity of satisfying myself of the softness of the liver in the living body, in the course of two operations for hydatids of that organ, and it felt under these circumstances more like a flaccid cyst than a solid gland.

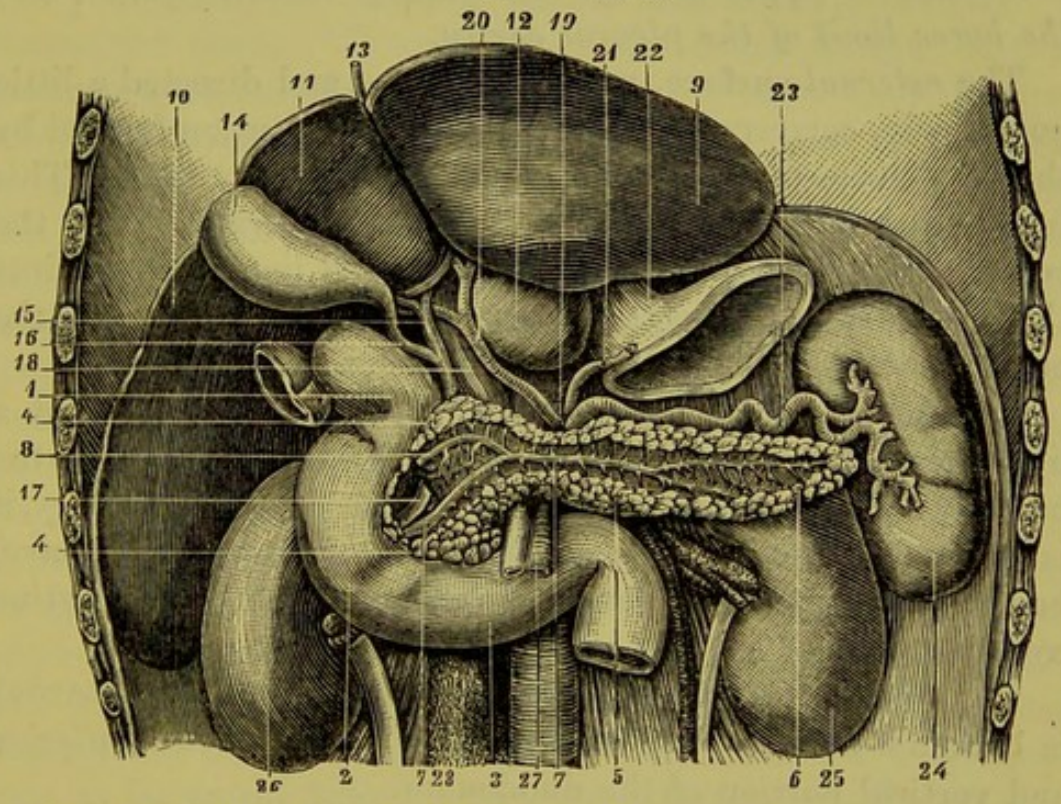
Relations of the Kidneys to adjacent organs.

The *anterior* surface, in the fœtus lobed, in the adult smooth, convex, directed a little forwards and outwards, is covered by the peritoneum in its whole extent. On the *right* side two-thirds or three-fourths, or sometimes even the whole of the kidney lies beneath the liver and therefore to a greater or less extent beneath the ascending colon or hepatic flexure of the colon (flexura coli dextra), and more or less close to

[1] Incorrectly quoted by Landau.—TRANSLATOR.

the vertical portion of the duodenum and the vena cava inferior. On the *left* side the anterior surface of the kidney is covered in its upper third by the spleen, and in its lower two-thirds by the descending colon or splenic flexure of the colon (flexura coli sinistra). Its inner border abuts on the extremity of the cardiac end of the stomach. Part of it lies beneath the pancreas, which is indented by it and the renal vessels.

FIG. 1 (*after Sappey*).



1. Superior horizontal limb of the duodenum turned to the right side with the pyloric end of the stomach.
2. Vertical limb of the duodenum.
3. Inferior horizontal limb of the duodenum, bounded on the left by the superior mesenteric artery and vein.
4, 4. Head of the pancreas.
5. Body of the pancreas.
6. Tail of the pancreas.
7, 7. Its main duct.
8. Accessory duct.
9. Left lobe of the liver.
10, 11. Right lobe of the liver.
12. Lobus Spigelii.
13. Obliterated umbilical vein.
14. Gall-bladder.
15. Hepatic duct.
16. Cystic duct.
17. Common bile-duct, opening, together with the pancreatic duct, into the Ampulla Vateri (common gall and pancreatic duct) and the duodenum.
18. Portal vein, covered on the right by the common bile-duct, and on the left by the hepatic artery.
19. Cœliac axis.
20. Hepatic artery.
21. Coronary artery of the stomach.
22. Cardiac end of the stomach.
23. Splenic artery.
24. Spleen.
25. Left kidney.
26. Right kidney.
27. Inferior mesenteric artery and vein.
28. Vena cava inferior.

The *posterior* surface, which is nearly flat, inclined a little inwards and broader than the anterior, lies against the crura of the diaphragm which separate it from the last rib, the last intercostal space, and the lowest part of the pleural cavity. It is separated from the quadratus lumborum muscle, against which it also lies, by the anterior layer of the aponeurosis of the transversus abdominis muscle, and by the two branches of the lumbar plexus (? the ilio-hypogastric and ilio-inguinal nerves, Translator). *The upper third and sometimes the upper half of each kidney lies therefore above the lower limit of the pleural cavity.*

The *external* surface, which is convex and directed a little posteriorly, corresponds to the angle of separation formed by the two layers of the fascia propria of the peritoneum. This border also is separated above by the diaphragm from the twelfth rib and last intercostal space, and lies below against the aponeurosis of the transversus abdominis muscle and the outer border of the quadratus lumborum.

The *internal* border (Hilum) which is concave, directed a little forwards, rounded above and below, and concave in the middle, abuts on the psoas major. According to Sappey it is covered below by coils of the small intestine. I have, however, frequently found nothing but large intestine covering it.

The *internal* border (? of the right kidney.—Translator) is bounded above and to the right by the vena cava inferior and vertical portion of the duodenum.

The *upper end* of the kidney is rather greater in circumference, more curved and nearer to the vertebral column, than the lower. It corresponds to the intervertebral disc between the eleventh and twelfth thoracic vertebræ.

The *lower end* is bounded by the intervertebral disc between the second and third lumbar vertebræ.

The average *length* of the kidneys, according to Sappey, amounts in women to 12·2 centimetres, their *breadth* to 6·9 centimetres, their *thickness* to 2·8 centimetres; according to Pansch they are 10·3—11 centimetres long.

The *weight* of the kidneys varies considerably, averaging, according to Sappey, 170 grammes; of forty kidneys the lightest weighed 107 grammes, the heaviest 284 grammes.

Their specific gravity, as I have found by several determinations, is greater than that of any of the solid abdominal glands.

Mode of Fixation of the Kidney.

The kidney, firmly surrounded by a tunica propria, lies comparatively loose in the envelope, called by Riolan (81) *membrana adiposa*, by Haller (82) *capsula adiposa*, the name now in common use. This capsule, however, in the fœtus and young subject contains no fat, but consists entirely of connective tissue. It arises from the lamina fibrosa of the fascia propria peritonei which on reaching the kidney divides into two layers, one of which runs with the peritoneum transversely over the anterior surface of the kidney, while the other passes on the posterior surface beneath the pelvis and vessels of the kidney, in which situation it joins the anterior layer again. Englisch (83) describes this membrane lying behind the renal vessels as a layer consisting of closely compressed compact connective tissue running inwards, and passing partly into the adventitia of the aorta, partly into the fascia lying over the pars lumbalis diaphragmatis. According to Englisch this layer contributes in a special manner to the fixation of the kidney and acts as a proper *ligamentum suspensorium renis*. At the upper end of the kidney these two layers join again and separate the kidney from the supra-renal body; from the lower end of the kidney they pass as far as the brim of the pelvis, becoming thinner and thinner in their course. The anterior layer is intimately connected with the peritoneum by a fine connective tissue destitute of fat; the posterior, on the other hand, is somewhat loosely united to the posterior abdominal wall and all surrounding parts. The capsule of the kidney is connected to its tunica propria by loose connective tissue with large undulations. It is not until the eighth to the tenth year that the wide meshes of the connective-tissue capsule begin to be filled with fat, the abundance of which frequently stands in no sort of proportion to the panniculus adiposus elsewhere. It provides the kidney with a soft cushion, protects it against the pressure of adjacent viscera, and by virtue of its softness and mobility permits a moderate amount of change of position.

The capsula adiposa, connected as it is with the peritoneum in front and secured as it is to the abdominal walls behind and at the sides, represents the kidney's *immediate means of fixation*, and justly deserves the title of *ligamentum renis* conferred on it by *Bartholin*.

In addition to the above, the kidneys are maintained in position *indirectly* by the *mesocolon* which is short and tense, attached to the posterior abdominal wall, and is sometimes only indicated; this to a certain extent opposes its descent inwards and downwards after the manner of a barrier. Any movement of the kidney outwards and downwards, or outwards and upwards, is prevented by the renal vessels which are tensely, and the aorta and vena cava which are almost immoveably fixed.

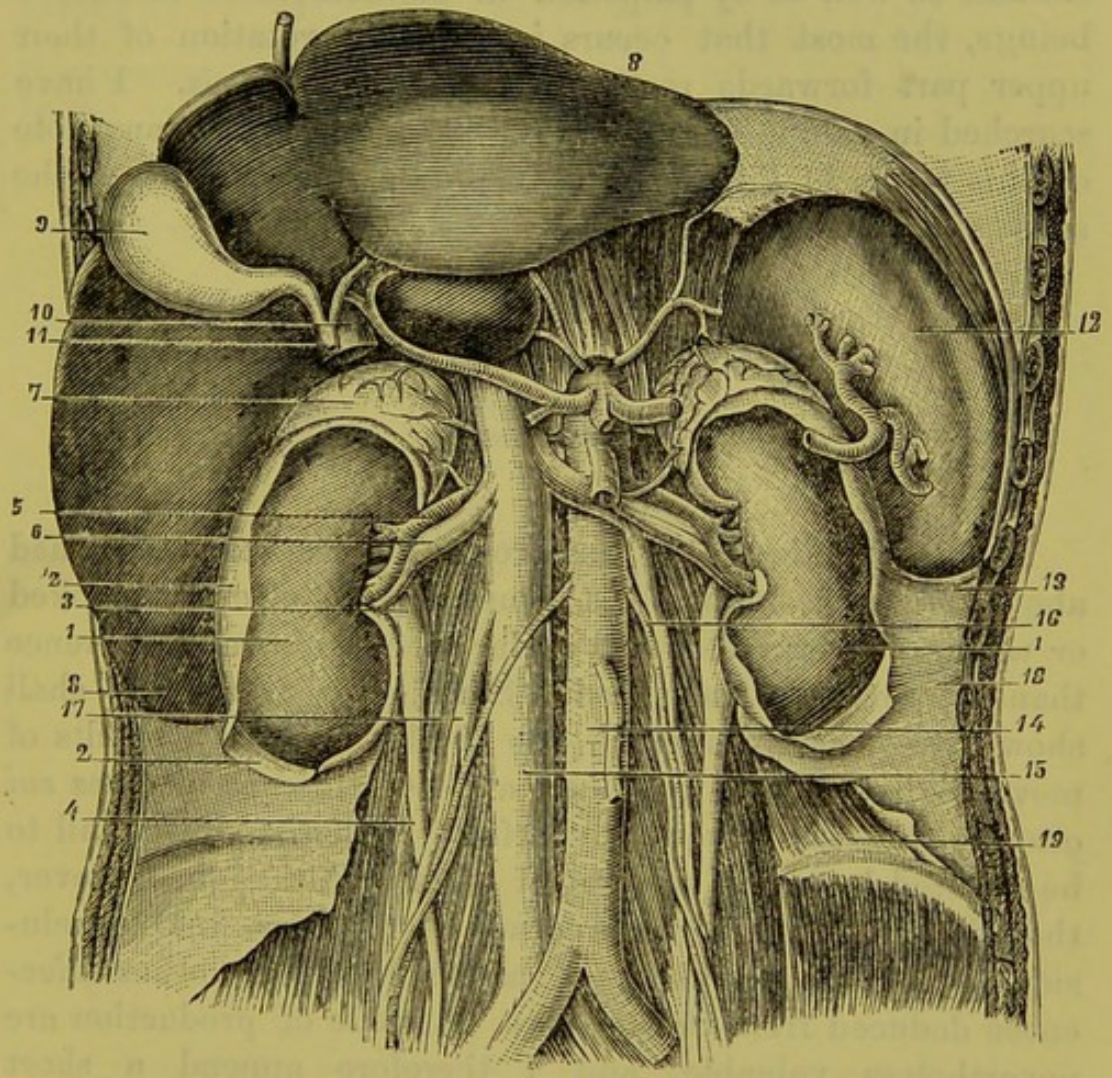
The kidney is still further protected by lying close to that very part of the vertebral column which is not affected to any considerable extent by flexion, extension, or lateral movements of the body, this region being according to Meyer (84) the section from the ninth thoracic to the second lumbar vertebræ which must be regarded as a nearly immoveable mass. Lastly, the enclosure of the greater part of the kidneys within the bony cavity of the thorax protects them from lateral and anterior pressure. *Haller* happily compares the spot where the kidneys lie to a nest in the following words:[1] "Renes ponuntur ad utrumque latus spinæ dorsi . . . . quasi in cavato sibi nido."

Apart from these *anatomical factors* there are also *physical conditions* which oppose the descent of the kidneys, especially the *adspiratory force of the diaphragm* and the *intra-abdominal pressure* produced by the normal act of straining. If one of these factors is eliminated in experiments on the dead subject as may be done by opening the abdominal cavity or removing the diaphragm, the kidney is seen almost invariably to sink slightly by virtue of its weight. In spite of this, the anatomical fastenings are so strong, that *Sappey* found as a result of 24 experiments in which the cadaver was raised upright, that the kidneys remained immoveable in 21, and in 3 only sank 2 centimetres. Though the descent of the kidneys is ascribed by *Sappey* in these experiments solely to their weight, it

[1] Incorrectly quoted by Landau.

must not be forgotten that their weight cannot come into operation until the abdominal pressure is eliminated by opening the abdominal cavity and removing the abdominal viscera.

FIG. 2 (*after Sappey*).



1, 1. The two kidneys.
2, 2. Capsula fibrosa, by which they are fixed to the posterior wall of the abdomen.
3. Pelvis of the kidney.
4. Ureter.
5. Renal artery.
6. Renal vein.
7. Suprarenal body.
8, 8. The liver, raised, in order to show the relations of its lower surface to the right kidney.
9. Gall-bladder.
10. Terminal portion of the portal vein.
11. Origin of the common bile-duct.
12. The spleen, turned outwards, in order to show the relations of its inner surface to the left kidney.
13. Semicircular pouch on which the lower end of the spleen rests.
14. Abdominal aorta.
15. Vena cava inferior.
16. Left spermatic artery and vein.
17. Right spermatic vein opening into the vena cava inferior.
18. Subperitoneal fibrous layer or fascia propria, dividing at the convex border of the kidneys into two folds, to form their sheath.
19. Lower end of the quadratus lumborum muscle.

Thus during life the kidneys remain almost immoveably fixed. Even deep inspiration does not depress them, although they lie against the crura of the diaphragm, to a great extent above the lowest limit of the pleural cavity. Under these circumstances, as is shown by experiments on animals as well as by palpation in suitable cases in human beings, the most that occurs is a slight rotation of their upper part forwards round their transverse axis. I have searched in vain in the physiological handbooks for any note on this point, in *Pansch* alone I found a short remark to the same effect as that just made.

IV. Statistics.

Notwithstanding the numerous publications mentioned above, moveable kidney is a complaint still often overlooked or wrongly interpreted, and of far more frequent occurrence than is generally imagined. Since, moreover, as I shall show, many maladies which are really secondary results of moveable kidney are imagined and described as diseases *sui generis*, statistics never so carefully prepared cannot fail to be affected by many sources of error. Although, however, they do not for this reason permit us to draw final conclusions as to the frequency of moveable kidney, other inferences deduced from them as to its mode of production are nevertheless valuable, and I therefore append a short collection of cases in point, of which I have personally observed 42.

The complaint has been most frequently observed in patients between 30 and 40 years of age, as is shown by the following data :

Author.	No. of cases.	Age.						
		1—10	10—20	20—30	30—40	40—50	50—60	60—70
Aberle . .	4	—	—	—	2	—	—	2
Rayer . .	7	—	—	1	2	4	—	—
Dietl . .	9	—	—	2	3	4	—	—
Rollet . .	22	—	2	6	11	1	1	1
Keppler . .	11	1	—	2	5	2	1	—
Steiner . .	3	3	—	—	—	—	—	—
Hirschsprung .	2	2	—	—	—	—	—	—
Landau . .	42	—	—	4	20	10	7	1
	100	6	2	15	43	21	9	4

It affects *women* by preference.

Author.	No. of cases.	Women.	Men.
Aberle . . .	4	3	1
Rayer	7	7	0
Dietl	9	8	1
Rollet	22	18	4
Henoch . . .	6	4	2
Lancereaux . . .	8	8	0
Jago	5	5	0
Kowatsch . . .	5	5	0
Schultze . . .	3	3	0
Guéneau de Mussy .	12	11	1
Thun	4	3	1
Klüpfel . . .	3	3	0
Fourrier . . .	6	6	0
Steller . . .	3	3	0
	97	87	10

Ebstein's collection consisted of 96 cases, 82 in women and 14 in men; *Fritz's*, of 35 cases, 30 in women, 5 in men; *Lancereaux's*, of 64 cases, 55 in women, 9 in men; *Hare's*, of 23 cases, 20 in women, 3 in men. The above numbers added together give 314 cases, 273 in women, and 41 in men, a proportion which may be taken as nearly representing the truth, although in this total certain cases are counted more than once. This proportion would turn out still more in favour of the women if we were to include in the above collection the data of authors whose practice lies exclusively

among women. Thus *Howitz* observed 34 cases, *Chroback* 19, and I myself 42. *Dietl* stated the proportion of women to men as 100 to 1, but this is certainly an exaggeration.

The *right kidney* is the one most commonly found moveable.

Author.	No. of cases.	Kidneys moveable.		
		Right.	Left.	Both.
Aberle	4	4	—	—
Rayer	7	5	1	1
Dietl	9	8	—	1
Rollet	22	18	3	1
Henoch	6	5	—	1
Guéneau de Mussy	12	11	1	—
Jago	5	5	—	—
Schultze	3	3	—	—
Kowatsch	5	5	—	—
Thun	4	3	1	—
Klüpfer	3	3	—	—
Fourrier	6	5	—	1
Keppler	11	10	1	—
Oerum-Howitz	34	28	4	2
Landau	42	39	1	2
	173	152	12	9

If we add to these 5 other cases of double moveable kidney (*Philippson, Wiltshire, Heslop, Schiff, Schenker*, and *Hunter*, having observed one case apiece) we get 178 cases of moveable kidney, 151 right-sided, 13 left-sided, and 14 bilateral.

Author.	Cases collected.	Kidneys moveable.		
		Right.	Left.	Both.
Hare	23	18	5	—
Lancereaux	43	31	5	7
Ebstein	91	65	14	12
	157	114	24	19

An opposite proportion is observed in congenital malposition of the kidney, which, as we learn from the statistics of *Willis* and *Gruber*, is far more frequently found on the left side.

Most of the women with moveable kidney had had several children, as is shown by the subsequent table of my observations. Out of 42 cases observed by me, only 2 concerned women who had never borne children.

With regard to the station in life and occupation of patients with moveable kidney, it must be remarked that most of them belong to the labouring class, as would be naturally expected to be the case with hospital in-patients and out-patients, who furnish our material for observation. This complaint is, however, by no means confined to the poorer class, as is shown by the six cases published by Henoch, which one and all concern persons of fairly good position.

V. Pathological Anatomy.

Little attention has hitherto been paid at post-mortem examinations to the changes produced by moveable kidney. It is true that the old classical anatomists, *Eustachius, Bauhin, Morgagni, Ruysch,* and *Haller,* quote cases of kidneys lying low down, horse-shoe kidneys, abnormalities in the renal vessels, &c., but they do not give much more information about the conditions in acquired moveable kidney as regards pathological anatomy, than the recent textbooks and handbooks on renal diseases and special pathology. *Cruveilhier* contents himself with the statement that the kidneys not only leave their situation but also rotate on their axis. *Rokitansky* is even less explicit in specially mentioning the congenital abnormalities of position.

Moreover, in the monographs of *Rollet, Le Ray,* and *Defontaines,* we look in vain for an explicit account of the conditions as regards pathological anatomy, so that we are driven to study isolated post-mortem records as the only means of arriving at a better understanding of the subject. How scanty these are, however, is best recognised by the fact that *Durham* only found two cases of displacement of the kidney in 1600 autopsies, and that of these one was congenital. In the same way *Schultze* found only five cases of moveable kidney in 3658 autopsies in the Charité Hospital of Berlin

between 1859 and 1866, and of these he unfortunately gives no description. By the kindness of Geheimrath Professor Dr. Virchow I was permitted to look through the post-mortem records of the Charité between the years 1870 and 1879 containing about 6000 autopsies, and among them I found moveable kidney only four times noted, and even then without any special statement about it.

This slender result, however, can surprise no one who remembers that moveable kidney as such never produces a fatal result, and rarely gives any indication either to the clinical physician or to the pathological anatomist to specially investigate on the dead subject the amount of the mobility of the kidneys. Since, in addition to this, a moveable kidney, even in well-marked cases, is frequently in the habit of returning to its normal position during life under the conditions of dorsal decubitus, and still more so immediately after death (see below), even well-marked cases are very apt to escape our observation during an autopsy conducted in the usual manner, particularly as it usual to remove the kidneys from the abdominal cavity before removing the intestines and other abdominal viscera.

In the last place, however, moveable kidney sometimes gives rise to complications of such a nature that its presence can no longer be seen but only inferred in the cadaver; this is the case in many peri- and para-nephritic abscesses, hydro- and pyo-nephroses, thromboses of the vessels, &c., which are regarded as diseases *sui generis*, whereas their proper place belongs to the pathology of moveable kidney.

Considering, therefore, the rarity of post-mortem records it would seem not superfluous to enumerate them in this place, scattered as they are, and collected with much labour. In the performance of this task it is evident that the most accurate and serviceable accounts date from the period antecedent to the microscope, whereas in the autopsies dating from more recent times, the coarser naked eye changes which concern us in this place are generally neglected.

(1) *Moveable Kidney on the right side, violent gastric pain. Death in consequence of apoplexy.* (Aberle, 1820).

A man, thirty-two years of age, who had suffered for many years from severe abdominal and digestive disturbances, had been much distressed for the last three years by a tumour in the abdomen, situated in the right hypochondriac and umbilical regions, which drove him to the use of various, and sometimes injurious remedies. This supposed tumour could be squeezed upwards at the patient's pleasure, when he lay in a particular position in bed, especially in the morning. Under these circumstances, a firm, smooth, not tender body, of the shape and size of a hen's egg, could be felt more or less to the right, somewhat above and near to the navel, which however, when not fixed from above by the pressure of the hand, slipped away again from the examining finger, often quite suddenly, and retired to the right side, backwards and upwards beneath the liver. By degrees the tumour grew and could be pushed in front of the bodies of the lumbar vertebræ. As the diagnosis remained doubtful, the physicians refrained from active measures; the patient however fell a victim to a consumptive disease and died with symptoms of apoplexy.

At the autopsy, with the exception of the softening of the right corpus striatum, &c., the several organs were found healthy; "the tumour however was formed by the right kidney which was found very moveable in its normal position in the right loin, from whence it could be very easily displaced in front of the bodies of the lumbar vertebræ beneath the duodenum downwards and forwards, that is to say, in the very same direction in which the supposed tumour appeared and disappeared again during life. Its vessels were comparatively long; the cellular capsule of this kidney (fascia renalis) was completely destitute of fat, the hepatic flexure of the colon was unusually distant from the liver, and depressed somewhat towards the middle of the abdominal cavity."

The kidney seemed to *Aberle* congenitally predisposed to mobility, and this tendency appeared to him to have been encouraged by the repeated attempts to render the tumour

apparent. The length of the vessels seemed to him to prove the congenital predisposition.

(2) *Moveable Kidney on the right side. Remarkable displacement of the large and small intestine.* (Aberle, 1841.)

At the autopsy of a woman sixty-six years of age who had died after several apoplectic attacks, the opening of the abdominal cavity at once disclosed a body on the right side of the umbilical region glistening through the peritoneum which lay over it, destitute of fat and quite loose. This body was kidney-shaped and on closer examination proved to be in fact the right kidney lying somewhat obliquely in front of the psoas in such a position as to turn its concave border somewhat upwards. Besides this, the ascending colon and right part of the transverse colon were so much depressed from the right hypochondrium towards the hypogastric region, that no ascending colon could be found in this region, but the cæcum passed at once into the transverse colon, which ran down towards the pelvis and then up towards the spleen in the shape of a V to pass through the splenic flexure into the descending colon. Again, on turning the liver up, the duodenum itself, somewhat depressed, was very distinctly seen through the anterior layer of the obliquely placed transverse mesocolon, between the liver and mesocolon. The stomach descended more obliquely than usual towards the duodenum from left to right. The small intestines and part of the great omentum were for the most part depressed in the abdominal cavity. The left kidney was in its normal position.

I have found a similar observation with regard to the situation of the colon in *Sandifort* (85) in the section; De præternaturali diversarum partium statu, in cadavere mulieris viso.[1] "Colon ex caeco enatum, juxta renem dextrum adscendens, et ad hepar pertingens, mox reflectebatur, juxta caecum, ad pelvis marginem usque descendens, inde ad ventriculum et lienem adscendebat, denique, iterum descendens, pelvim intrabat. . . . Ren dexter sanus, sinister morbosus."

[1] Incorrectly quoted by Landau.

The colon therefore lay in the same position as in the former case except that the same displacement had been here produced by a hydronephrosis on the left side.

(3) *Moveable Kidney on the right side; compression of the vena cava; thrombosis of the vena cava; œdema of the right lower extremity; death in consequence of tubercular pleurisy* (Girard, 1837).

A woman, forty-seven years of age, had suffered for twenty years from febrile symptoms and chest troubles which showed themselves by violent attacks of coughing, shortness of breath, and palpitation. She lost flesh rapidly, and six months previously, after a particularly violent attack of coughing, became conscious of severe abdominal pain. For the last month the right leg had been greatly swollen. The patient died with the symptoms of consumption.

At the post-mortem, besides pleurisy on the right side, cavities, &c., the right lower extremity was swollen to twice the size of the other, the abdominal viscera healthy, the left kidney in its normal position. The right kidney showed a remarkable abnormality. The peritoneum, instead of merely covering its front surface, enveloped the whole kidney except the hilum and thus formed a sort of true mesentery about two inches long. The kidney also floated in the abdomen at the level of the third lumbar vertebra and at the inner side of the ascending colon, which, distended with gas, pressed the kidney forcibly against the vena cava inferior. The vein showed at this spot a considerable constriction with a dilatation below to nearly double its size. From the heart to this constriction it was healthy in its course; but below this point it showed true areolar or cavernous tissue like that of the penis, which filled the whole calibre of the vessel, and reached to within two or three inches of the groin. The femoral and saphenous veins were of the normal diameter. The right kidney itself presented no special point of interest.

(4) *Moveable Kidney on the right side; neuralgia of the leg; death from entero-colitis and peritonitis* (Rayer, 1841).

A woman, fifty-one years of age, who had been employed at hard work in a tobacco factory for thirteen years, and had lost flesh rapidly, stated that ever since a fall she had suffered from severe pains in the right leg and in the right loin, radiating as far as the labium majus. Nothing but rest on her back relieved the pains, and as soon as the patient tried to turn in bed they returned violently in the region of the right kidney.

Bimanual examination easily detected the kidney, which quickly slipped out of the fingers, the patient at the same time experiencing a pain radiating to the right knee. Constipation is present; the urine contains no albumen.

Ten days after she came into the hospital symptoms of peritonitis appeared, and she died of marasmus two months later.

At the post-mortem the voluminous and very congested liver was seen to extend low down, and a narrow and thin band of it, which lay against the right lateral wall of the abdomen, reached as far as the iliac crest. Its growth had displaced the right kidney, which lay superficially at the sides but more anteriorly in front of the vertebral column—pressed from above downwards but still more from behind forwards—in the obtuse angle formed by the iliac vein and vena cava, both of which were closely apposed to it. Externally it was bounded by the prolongation of the liver, but only at its outermost border; its anterior surface was exposed only in the lower half, the upper half being covered by the liver; posteriorly it lay on the projecting angle formed by the muscles occupying the lower part of the abdomen and the upper part of the pelvis, and rising partly below and partly above the iliac crest. Its inferior extremity reached as far as the middle of the fibro-cartilage between the fourth and fifth lumbar vertebræ.

It follows from the above relations that the lower end of the right kidney lay in the immediate neighbourhood of the anterior abdominal wall, and on a plane obliquely inclined

from before backwards; this explains the ease with which it could be felt with the hand and pressed backwards. Its mobility was increased by extreme looseness of its connections and peritoneal investment; the condition of the vessels was similar, except that the transition to them was more direct and shorter.

The left kidney occupied its normal position (?). It was packed away much deeper at the side and behind the vertebral column, and projected downwards as far as the fibro-cartilage between the third and fourth lumbar vertebræ, thus lying not much higher than the right which seemed to be displaced more markedly forwards than downwards.

The right kidney weighed 80 grammes, the left 150, the right was 0·4 centimetres shorter than the left. The shape of the right was slightly altered, its upper end was pressed against the liver, and actually flattened by contact with it; besides this it showed an anterior convexity and a posterior concavity, corresponding to its position on an oblique plane, double-inclined and convex anteriorly.

The left kidney showed marked increase of its tubular substance.

The right supra-renal body was found in its normal position.

(5) *Double moveable Kidney* (William Roberts, 1852) (86).

In a phthisical, very thin woman, both kidneys were very plainly felt through the abdominal walls, the right much lower than the left.

At the autopsy the right kidney was found lying loose one and a half inches below the liver, and attached only by its vessels and ureter; it was entirely destitute of fat. The left kidney had a normal position, but lay an inch lower (than usual). The artery and vein of the right kidney were half an inch longer than those of the left.

(6) *Moveable Kidney on the right side; schirrhus of the uterus; hydronephrosis* (Braun, 1853).

In a woman, fifty years of age, suffering from cancer of

the womb and ascites, a solid moveable tumour was made out during life in the umbilical region. Death occurred with symptoms of coma.

At the autopsy a large steatoma was seen on the right side near the umbilicus, enveloped in a yellowish white mass. On opening this, the right kidney could be seen lying in it, enlarged, pale, soft, in a state of fatty degeneration, the cortical substance being hardly brownish-red and bloodless. The renal vessels tore when it was disturbed, and the renal artery alone showed any remaining firmness of texture; the ureter was distended to the size of a finger, and filled with fluid down to the bladder.

Braun's opinion was that the mobility of the kidney was due to the pressure and maceration of the abdominal glands—that this was the cause of the ascites—and that the tenesmus and relaxed state of the abdominal contents had largely contributed to the displacement of the kidney.

(7) *Moveable Kidney on the right side; adhesions between it and the liver, gall-bladder, and transverse colon; hydronephrosis; uterine infarct; multilocular ovarian cyst on the right side* (Urag, 1857).

In a very thin woman, sixty-five years of age, a tumour could be felt under the anterior edge of the right lobe of the liver. It ran obliquely above and inwards, downwards and outwards; its shape was oval, the upper border convex, the side flatter; it seemed elastic, well defined, moveable, sinking and rising with respiration, and in shape resembled the kidney. It would not rotate on its axis, but could be pushed as far as the middle line, into the right renal region, and also somewhat downwards, though these manipulations were painful to the patient. Compared with the left lumbar region the right was sunken and tympanitic on percussion; if the tumour was pushed into the right loin it became full in appearance and dull to percussion. The quantity of urine secreted in twenty-four hours amounted to 1190 cubic centimetres, its reaction was acid, it was slightly turbid, deeply yellow without deposit, its specific gravity was 1015.

The woman died in consequence of bronchiectasis. At

the post-mortem the right kidney was found lying in immediate contact with the anterior border of the right lobe of the liver and adherent to it as well as to the gall-bladder and transverse colon by dense connective tissue, the adhesions permitting the kidney to be pushed back into its normal situation and also as far as the middle line or into the umbilical region (mesogastrium), though not without dragging on the liver. The outer border of the kidney looked upwards, the upper extremity inwards, the lower extremity outwards, the hilum downwards; the kidney was twelve centimetres long, seven broad, four thick; the peritoneum investing it, as well as its capsule, thickened. The capsule was distended irregularly and in the shape of vesicles (blasig), it felt elastic and fluctuating. The substance of the gland had entirely disappeared, and in its place was found a colourless clear thin fluid, enclosed by the empty septa (Fächern), of the capsule which was shining and smooth internally. The renal vessels were elongated and contracted, the pelvis thickened and four and a half centimetres broad, the ureter as far as the bladder was two centimetres in diameter, its mucous membrane reddish-grey, thickened and granular. The left kidney was firmly seated in its normal position, it was in an early stage of hydronephrosis.

The size of the uterus corresponded with the third month of pregnancy. The posterior wall, which was four centimetres thick was chiefly answerable for its increased bulk. Its tissue was yellowish-red, soft, infiltrated with pus. The cavity was small, and occupied by glairy mucus. The posterior wall of the bladder was closely adherent to the uterus, and on pressing the uterus pus escaped into the bladder from several points; the opening of the right ureter was closed, the opening of the left narrowed. In the right ovary were five serous cysts, the smallest as large as a chestnut, the largest as big as a lemon; a cyst the size of a hazel-nut lay at the brim of the pelvis.

Urag attributes the hydronephrosis to the pressure of the uterus on the ureter.

(8) *Moveable Kidney on the left side; neuralgia of the leg hydronephrosis mistaken for an ovarian tumour.* ('Bulletins de la Société Anat.,' 1854, s. le Ray, p. 32.)

In a woman forty years of age, much reduced in health, who complained of violent boring pains in the lumbar region and left leg, a large hard tumour was found to the left of the linea alba and reaching nearly to the false ribs. Bimanual examination detected the same tumour in the left fornix vaginæ. The uterus was descended and moveable; on pressing it upwards very restricted movements were produced in the tumour which was large and hardly moveable by itself. The tumour was taken to be a malignant tumour of the left ovary.

At the autopsy it was found to be the kidney elongated and enlarged to three times its size, lying with its lower extremity on the fundus uteri and the left broad ligament. It lay in the iliac region a little obliquely from above downwards and from without inwards. It was nineteen centimetres long by seven broad. It was decolorised, lobed externally, soft, and giving marked fluctuation at the level of the hilum, or rather over the whole inner border. Section disclosed large, inter-communicating spaces, which were in fact the dilated infundibula and pelvis. These dilatations contained a turbid serous fluid without any gritty or calcareous deposit. The mucous membrane of the excretory ducts was very thick and covered in its whole extent as far as the bladder with false membranes, giving it the appearance of shagreen or of the back of a calf's tongue.

The ureter was also hypertrophied, its calibre uniform and nowhere occluded except at the hilum, where it was closed by false membranes. The renal substance was also changed, the cortical part being amyloid and uniformly greyish-white. The pyramids of Malpighi were distinguishable only in the upper and middle parts of the kidney, by their triangular outline and pale red colour, but their striated appearance was almost completely lost.

It is unfortunate that no observation is made, in this as in most of the accounts in point, as to the origin and condition of the renal vessels, nor as to the position of the colon

and its relation to the kidney, so that it is impossible to set at rest the doubt whether this might not have been a case of congenital displacement.

(9) *Moveable Kidney on the left side, with extreme mobility; displacement of the colon* (Durham, 1860).

At the autopsy of a woman thirty-four years of age the left kidney was seen to project remarkably. The descending colon lay much nearer the middle line than usual and instead of forming the sigmoid flexure on the left side, it ran across the lumbar vertebræ into the pelvis to the right of the sacrum. Slight pressure caused the kidney to slip into its usual position, and closer examination proved it to be moveable not only with extreme ease but also to a great extent.

When the patient moved in various suitable ways and under gentle manipulation the kidney glided away with equal ease across the vertebral column and under the ribs, or even somewhat to the right side into the iliac fossa. On closing the abdominal cavity and palpating the lumbar region the kidney was felt as a smooth, oval, half-elastic tumour, which slipped away when pressed by the fingers.

The peritoneum was abnormal. Instead of passing over the anterior surface of the kidney it only just touched the lower part of its inner edge, and then after forming the descending mesocolon, again came into contact with the outer border of the kidney. Moreover, the lesser cavity of the peritoneum (sac of the omentum) reached so far towards the left side as to cover the posterior surface of the spleen, and so far downwards as to be in contact with the upper border of the kidney. The kidney had no distinct mesentery (mesonephron). In the loin hardly any fat was found but only loose cellular tissue. The absence of fat partly explains the mobility of the kidney. The renal vessels had their normal origin, but were perhaps longer than usual. The left supra-renal body moved with the kidney only to a limited extent.

(10) *Moveable Kidney on the right side. Adhesions to the liver; parametritis of the left side* (Lancereaux).

In a woman aged forty-one who was a drunkard, had suffered from frequent vomiting and cough, and was very thin, a moveable kidney could be very easily made out on the right side of the abdomen.

After the patient's death from erysipelas of the face the right kidney was found not against the quadratus lumborum muscle but tightly pressed against the right side of the vertebral column. It was much smaller than the left kidney and slightly lobed. Its right border was adherent to the liver which was rather large.

The posterior wall of the uterus was adherent to Douglas's pouch by old false membranes, which were confined to the right half of the posterior surface of the right broad ligament. The inflammation had extended along the ureter as far as its upper extremity. The right ovary which was involved in this focus of inflammation, was two or three times as large as the left, the tube was sinuous, its end was closed and adherent to the peritoneum opposite the posterior fundus vaginæ, and enlarged to the size of a nut.

(11, 12) *Moveable Kidneys on the right side* (Lancereaux).

In a consumptive woman, aged sixty years, the right kidney was found astride (à cheval) of the second and third lumbar vertebræ, covered with peritoneum. The vessels and ureter appeared normal, somewhat elongated; the liver was congested and seemed to have pressed the kidney downwards, though it could be presumed that the kidney had changed its position previously. The right ovary was unchanged, but the uterus was studded with fibrous tumours.

A woman who died with stenosis of the mitral valve showed a condition precisely similar.

(13) *Moveable Kidney on the right side* (Mosler, 1866).

A woman, aged thirty-six, died of double pneumonia after amputation of the thigh. *Grohe*, who made the autopsy, found the following conditions:

Liver enlarged. On turning it up, the right kidney, which was, however, very deeply situated, at once came into view. The kidney was rotated half round so as to bring its outer convex border almost entirely horizontal and looking downwards and on a level with the crest of the ilium, the hilum looking upwards and inwards. The right renal vein ran obliquely upwards and opened into the vena cava inferior at a somewhat acute angle. The ureter which was entirely covered at its origin by the kidney, first describing a slight curve, ran upwards over the trunks of the great vessels, and then passed towards the pelvic cavity as usual. The diameter of the vein, artery, and ureter was normal, no special alterations attributable to the abnormal position of the kidney could be made out. The only difference between the kidneys was that the right was somewhat smaller than the left. The right kidney was $4\frac{5}{8}$ in. long, $2\frac{3}{8}$ in. broad, $1\frac{6}{8}$ in. deep; the left was $5\frac{2}{8}$ in. long, $2\frac{3}{8}$ in. broad, $1\frac{3}{8}$ in. deep.

(14) *Moveable Kidney on the right side* (Jago).

In a woman who had lost much flesh from obstinate vomiting, a moveable kidney was discovered on the right side. Pressure against its lower end made it slip away from the hand upwards, and the upper end could be pressed to right or left. Pressure on the upper end drove the tumour plainly, but only a little, downwards. If seized with the whole hand it could be pushed upwards, but the hilum could not be reached. If pressed downwards, it was so smooth that it slipped away.

At the autopsy the kidney could be displaced from the loin to the extent of two inches above and three inches below, this mobility not being restrained by the vessels. The peritoneum was adherent to the whole anterior surface of the kidney, and to part of its lateral and posterior surfaces, but did not enclose the emerging vessels so as to form a mesentery (mesonephron). The peritoneum passed somewhat loosely from the kidney to the adjacent organs in the loin.

(15) *Moveable Kidney on the right side; caries of the thoracic spine; formation of a mesentery* (mesonephron) (Henderson).

A woman, aged fifty, had long observed a moveable tumour in the lower abdomen, which could be felt in the right side of the hypogastric region, disappeared beneath the liver on slight pressure, and reappeared on bending forwards. There was caries of part of the vertebral column of some years' standing.

The patient died of pneumonia. At the autopsy the bodies of the fifth and sixth thoracic vertebræ were found to be so far destroyed that two fingers could be introduced into the vertebral canal. The right kidney was suspended and quite moveable in a prolongation of the peritoneum like a mesentery; it was quite normal in size and structure. The left kidney was loosely attached, but not moveable. The liver appeared normal, except that it was rather small.

(16) *Moveable Kidney on the right side; caries of the fourth lumbar vertebra* (Ebstein).

In the body of a woman fifty years of age and much emaciated, the right kidney was found lying across the vertebral column on the usual level with the hilum looking backwards and upwards, the convex border looking downwards and forwards, the capsule of fat entirely absent; the kidney which was of normal shape could be displaced as far as the brim of the pelvis.

(17) *Moveable Kidney on the left side; caries of the first to the third lumbar vertebræ* (Cullingworth) (87).

In the body of a ballet-dancer, thirty-two years of age, who had had nine children, the psoas muscle was found almost completely destroyed; the left transverse process and the left half of the body of the second lumbar vertebra had disappeared, the tips of the transverse processes of the first and third lumbar vertebræ were also carious, the kidneys

and their calices dilated; on the inner and posterior side, where they were connected to the lumbar vertebræ, an opening was found forming a communication between the diseased vertebræ and the renal tumour.

The ureter had been occluded by a perforated piece of carious bone; and this perforation could only have happened subsequently to the dislocation downwards of the kidney.

Other post-mortem records were useless on account of excessive brevity or of inaccuracy. Those which have just been quoted establish the fact that moveable kidney both in life and after death is found by preference on the right side and in women. Again, moveable kidney presents no constant condition either during life or after death, but varies in respect of its position and degree of mobility. The first observation of *Aberle*, in which the kidney felt during life to the right of the umbilicus was found after death in its normal position, is very instructive. In this case, unless special attention had been given during the autopsy to the question of its mobility, the moveable kidney would certainly have been overlooked, as perhaps often happens.

The kidney is usually covered by small intestine and lies near the vertebral column in the region of the second to the fifth lumbar vertebræ, and, besides being displaced from its normal position, is rotated on one or two of its axes. In some cases its outer border is turned upwards, its upper end inwards, its lower end outwards, so that the hilum looks inwards instead of downwards (*Urag*); in others it lies in an exactly opposite position, transverse in the abdomen with the hilum upwards (*Mosler*). It is rare for the lower end to be directed forwards (*Rayer*); most frequently it lies obliquely from above and externally downwards and inwards.

In correspondence with this, the position and relation of the renal vessels and ureter to each other and the neighbouring organs is changed. The lower down the kidney lies the acuter the angle between the renal vessels and the vena cava and aorta must be, and the more curved must be the ureter; the more the kidney is rotated round one of its axes, the more must the calibre of the vessels and ureter be narrowed by torsion. Little attention unfortunately has

hitherto been paid to these changes in the cadaver, the vessels being only now and then described as elongated and narrowed.

The kidney which is moveable is in many cases remarkably small and light, in which case the other is hypertrophied by way of compensation. In *Rayer's* case the moveable kidney weighed 80 grammes, the other 150 grammes. It is usual for the kidney which is not moveable to be more deeply situated than normal. In some cases the kidney substance is itself degenerated, sometimes by fatty change, sometimes by the contraction of connective tissue within it, and it is sometimes in a state of hydronephrosis. Quite recently numerous cases of sarcomatous moveable kidneys have also been reported, as for instance by Wolcott (88), Kocher (89), Jessop (90), Czerny (91), Lossen (92), and Barker (93). Under such pathological circumstances as these, indeed, the mobility of the kidney is so far the lesser evil that it is not considered.

Hydronephrosis of moveable kidneys has been mentioned it is true, but not brought into any closer connection with the mobility of the kidney. *Simon* indeed stated expressly so late as 1876, that up to that time no case of hydronephrosis in a moveable kidney had been described.

The capsula adiposa shows a pretty constant condition, being destitute of fat in many cases where there is no great degree of general emaciation. The capsule of the kidney is then like an empty bag, which is bulged forwards by the kidney as far as its size and the looseness of the cellular tissue permit. By this means a kind of mesentery of the kidney, a mesonephron, is found, which is sometimes very long (*Girard, Oerum-Howitz, &c.*). This mesentery has been erroneously regarded by many authors (*Portal, Rollet, Ebstein, &c.*) as a congenital cause of moveable kidney.

Again, the hepatic and splenic flexures of the colon are found almost constantly displaced, being either bodily depressed downwards and inwards together with their mesenteries, or having the two layers of the mesocolon separated by the kidney which has in a sense migrated between them. A similar effect is produced by hydronephrosis, or, in the case of the broad ligament, by the growth of tumours of the

ovary or uterus within its substance. In this respect, therefore, hydronephrosis and moveable kidney behave alike, as the foregoing post-mortem records show, except that in the case of hydronephrosis the two layers of the meso-colon are kept apart by its constant growth, whereas in the case of moveable kidney it is the movements of the kidney which separate them (conf. the observations of *Aberle* and *Sandifort*). The displacement of the colon has also been erroneously regarded as a congenital cause of moveable kidney.

Moveable kidney produces no effect on the solid organs near it, thus even the supra-renal bodies are constantly found in their normal position.

Among other pathological changes observed in the dead subject are adhesions, contracted by the kidney with neighbouring parts. A favourite spot for these adhesions is the lower border of the liver, the gall-bladder and transverse colon.

It is rare for a moveable kidney to produce thrombosis of the vena cava inferior by compression (*Girard*).

VI. Ætiology and Pathogenesis.

If an attempt were made to explain the causes and mode of production of moveable kidney from post-mortem appearances only, the scarcity and incompleteness of post-mortem records would yield but imperfect results. Besides this, it would be impossible thus to decide the question whether the changes discovered after death were causes or consequences of moveable kidney. In this way indeed the length of the renal vessels, the presence of a mesentery of the kidney (mesonephron), and the displacement of the colon have been considered actual causes of moveable kidney, while accurate observation shows that they are just as much results of moveable kidney, or that both pathological conditions are possibly consequences of a third common cause. Finally, there may be present during life certain anatomical disturbances which have an important bearing on the pathology of moveable kidney, and yet cannot be recognised either by post-mortem examination or by examination of the fully formed patho-

logical specimen, such for instance as compression, torsion of vessels, and pressure on nerves.

We have already seen that the kidney, lying as it does in the capsula adiposa, against the quadratus lumborum muscle, and for the most part within the bony enclosure of the thorax, is maintained in its position directly by physical pressure, as for instance by the intra-abdominal pressure, as well as by its anatomical attachments. [1]As soon, therefore, as one of these factors fails, the kidney may become moveable.

Anatomical Causes.

Many authors (*Rayer, Rollet*) have mentioned the pressure exercised by neighbouring organs when enlarged (especially tumours of the liver and spleen) as important causes of moveable kidney; but this is erroneous. It is true that moveable kidneys have been found in patients with ague or splenic leukæmia or hypertrophy or tumours of the liver, but the well-established fact that they are not constantly found in these diseases proves that the mobility of the kidney cannot be laid to their charge. Since, moreover, tumours of the liver and spleen grow downwards over the anterior surface of the kidney and not in the direction of the long axis of the kidney, such tumours would tend to maintain the kidneys in their position rather than to depress them. The only tumours which could displace the kidneys by virtue of

[1] [The logic of this sentence as it stands seems doubtful; the author probably means that all the factors are necessary and no more than are necessary to maintain the position of the kidney. But this again is not proved. It seems more probable, from the analogy of other organs, that one or more factors are essential and generally sufficient, the others coming into play only in case of failure of the first. Thus in a railway train the screw-couplings are generally sufficient, but the chain-couplings are added in case of their failure. Or take the case of the uterus, whose descent is not prevented by the perineum unless its ordinary means of attachment fail; the perineum generally does not deserve to be described as one of the means of preventing descent of the uterus. This may be seen any day in women whose perineums are completely gone, the uterus nevertheless remaining high up in the pelvis. The question belongs to a large and important department.—Translator.]

their position are tumours of the suprarenal bodies and pancreas, examples of which are furnished by *Rayer* and *Bonnet*.

A similar effect is produced on the mobility of the kidney by tumours of its own substance, such as sarcomas, carcinomas, and hydronephroses. In this case the mobility is produced by the great stretching and relaxation which the capsule—the most important attachment of the kidney—sometimes suffers through the increase in weight and size produced by these tumours.

This is seen typically in cases of caries of the last thoracic and first lumbar vertebræ, and in psoas abscesses (Henderson, Ebstein, Culligworth), in which the kidney is deprived of its firm base of support.

One of the most important and frequent causes of mobility is absorption of fat from the capsula adiposa and relaxation of the peritoneum. The absence of the fat round the kidney can of course only produce mobility in cases in which it was previously present in considerable quantity and then became rapidly absorbed. If, however, the fat is only slowly removed, an accommodation takes place, and the kidney maintains its position just as it does in very young subjects who have never had any fat in the capsules of their kidneys. In the other case, however, after a rapid absorption of the fat, the capsule looks like a relaxed wide-meshed envelope, easily capable of being *bodily* dragged down from the posterior wall of the abdomen, and within which it is easy for the kidney to descend. We see here just the same conditions as we do in the skin, which can be lifted up from the muscles in folds whose size is greater the more rapidly and thoroughly the panniculus adiposus has been absorbed.

It generally happens, however, that at the same time as the fat round the kidney is being absorbed, that in the peritoneal structures and the abdominal walls is also becoming absorbed, so that the means by which the kidney is indirectly secured are also deprived of their strength, and under these circumstances[1] slight bearing down efforts, such as those

[1] [Here for the first time in this treatise we meet with the very important but very obscure question of the "Intra-abdominal pressure." That there is such a force, that it varies, that it has laws of its own, that its effects are

which occur during defæcation, are sufficient to dislocate the kidney downwards.

We ought not therefore to be surprised at finding among those suffering from moveable kidney a large number who have recovered from acute febrile diseases, such as enteric fever and ague, or who are still suffering from chronic and rapidly emaciating affections such as phthisis, &c. Thus out of nine of *Dietl's* cases, severe ague and enteric fever had preceded the moveable kidneys in four.

A conspicuous part in the production of moveable kidney is also played, especially in women, by affections of the *abdominal walls,* which are exposed normally in pregnancy and pathologically in the numerous cases of tumours of the genital organs, to serious alterations in compactness, firmness

important, and that it is not always positive, has been definitely proved by Matthews Duncan and Schatz; but beyond this very little is practically known. And at the outset difficulties meet us. In the first place it seems certain that hernia, descent of the uterus, and descent of the abdominal organs are conditions due to closely allied if not identical causes (this will be discussed again later on, p. 357). In this class of cases the intra-abdominal pressure would seem to be increased. But increased intra-abdominal pressure is equivalent to diminished specific gravity or increased buoyancy of the intra-abdominal organs, which (in the case of the kidneys for instance) would mean diminished tendency to descend.

With regard to the effort of straining as being an exciting cause of descent of the kidneys this explanation seems more than doubtful; for if the kidney is squeezed downwards by the diaphragm with a certain force, it is squeezed upwards by the abdominal muscles with an equally great force, besides the increased buoyancy produced by increase of the intra-abdominal pressure which would tend rather to lift than to depress it.

The effect of jerks and falls and perhaps vomiting is a question of impetus and belongs to a different category; their effect in dislocating the kidney, especially when the abdominal muscles are not braced, is easily explained.

The only possible mode of reconciling facts with regard to the conditions of the intra-abdominal pressure in our present state of ignorance would be to regard the kidney (and other abdominal viscera) as generally having a tendency to descend, even when their buoyancy was at its greatest, and to regard the apparent increase in the intra-abdominal pressure in hernia, descent of the uterus, &c., rather as the result of diminished resistance to a generally positive pressure, with which the weight of the viscera would co-operate. Against this view would have to be put the occasional high position of floating pelvic tumours and of the pregnant uterus. It is not, however, proved that the intra-abdominal pressure is the same in all parts of the abdominal cavity even at the same time.—Translator.]

and elasticity. True it is that it is normal for the abdominal walls which have been physiologically stretched during pregnancy, to make as good a recovery as the womb; but in cases where the lying-in has gone wrong, where labours and abortions have succeeded each other rapidly, they become flabby, shrivelled, and thin, and *pendulous belly*, which is here so injurious, is apt to follow, with or without divarication of the recti muscles. If now the intra-thoracic pressure is increased (*i.e.* if the diaphragm is forced down), as in lifting, in difficult defæcation, or severe efforts of any sort, the efficient means of fixing the abdominal contents furnished by well-braced abdominal muscles fails, and the descent of the kidney is opposed by diminished resistance. But the subjects of pendulous belly are just the persons who are also predisposed to severe bearing down efforts, inasmuch as they are usually much constipated. Finally, however, where pendulous belly is present, the action of the intra-abdominal pressure, which normally presses with equal force on all the viscera, is reversed, inasmuch as the bowels which occupy the loose sac formed by the abdominal walls exercise traction on the superjacent parts, including the kidneys, when the patient stands upright. The[1] fatter and heavier the abdominal walls, the greater the pendulous belly and the greater therefore this traction. This is the obvious explanation of the occasional great difficulty of breathing in persons with pendulous belly, the traction of the viscera impeding the normal expiratory movement of the diaphragm. But by the frequently recurring distension of the abdomen, whether physiological or pathological, the peritoneum also is relaxed, and in pendulous belly the anterior peritoneal layer of the capsule of the kidney, and with it the kidney, is subjected to direct traction.

The injurious effect of the above-named causes is confirmed by clinical observation. The vast majority of women affected with moveable kidney have borne many children, and, as shown by the appended table of my own observations, it is striking how frequently their deliveries had followed each other closely. Among the forty-two cases observed by me

[1] [It is, however, a fact that hernia and allied conditions are commoner in thin, flabby individuals than in fat ones.—TRANSLATOR.]

only two are nulliparæ, and of these one had carried a large ovarian tumour for eighteen years, and had acquired pendulous belly after I had removed it. Similar observations of cases in which the abdomen had long been distended by tumours or ascites are contributed by *Oerum-Howitz* and *Rollet*. Ruptures are frequently found in women along with moveable kidney, as observations by *Rayer*, &c., show.

Menstruation, again, has been named by *Bequet*, *Lancereaux*, *Fourrier*, and others among the influences contributing to the production of moveable kidney. According to these authors every menstruation produces congestion of the kidney and its capsule, and therefore increase of its volume, through a connection between the ovarian and renal plexuses, which connection, however, has yet to be proved. The relaxation of the capsule, its distention at next menstruation, and so on, gradually enlarge it to such a degree that the kidney becomes moveable within it. Although clinical symptoms show that a connection between moveable kidney and menstruation exists, and that the renal and uterine vessels are closely connected (as the investigations of *Virchow* (94) prove), a direct relation of cause and effect between menstruation and moveable kidney must be rejected, for, on the above theory no woman who menstruates should fail to have a moveable kidney.

There are, however, a series of other influences, hitherto too little regarded, which demonstrate the dependence of moveable kidney on the sexual department of a woman's organism, besides such causes as repeated pregnancy, tumours of the lower abdomen, &c., which have already been mentioned:—*these are the numerous displacements of the generative organs, the descents, prolapses, and inversions of the vagina and uterus.* In the first place these affections have important causes in common with moveable kidney, such as impeded involution of the generative organs post partum, relaxation of the peritoneum, and rapidly repeated deliveries; this is proved by their pathology. In the second place, however, descent of the genital organs favours the descent of the kidney by direct traction. Since the female generative organs are directly connected with the kidneys by the peritoneum as well as by the ureter, which runs close to the body of the uterus and in the substance of the upper third of the

vagina, this traction is increased by the bladder which is usually prolapsed in these affections. It either happens that the kidney yields to this traction, or that the ureter becomes closed by it.

Finally, a whole series of diseases of the female sexual organs contributes directly to the production of moveable kidney by inducing hydronephrosis which relaxes the capsule of the kidney. In consequence of the intimate mutual relations existing, as we shall see, between moveable kidney and hydronephrosis on the one hand, and between affections of the female generative organs and moveable kidney, as we have already shown, on the other hand, it seems imperative to consider very briefly the relation between certain affections of the female generative organs and the production of hydronephrosis.

Relations of Sexual Disorders to the production of Hydronephrosis.

Even *Walter* (95) explained the frequency of hydronephrosis in women by the circumstance that they possess more organs capable of pressing on the ureters and thus giving rise to accumulation of urine, than men.

Morgagni (96) had already called attention to the mutual relations between the pregnant uterus and the kidney, remarking on a case in point: "Inter caetera autem detrimenta quae mulieribus afferunt cruciatus nephritici, non dubito, quin abortus, aut non infrequens et fœtus, et matris interitus sit referendus. Cum enim uterus crescens ureteres premendo, minus per hos facilem reddat urinae defluxum, et, quod consequitur, nonnihil in renibus eam moretur. . . . ." &c.

Cases, again, are not rare in which an autopsy has shown compression of the ureter by tumours of the uterus and ovary.

A far more frequent cause of hydronephrosis is cancer of the uterus, in which hydronephrosis is almost one of the regular phenomena, as *Virchow, Säxinger*, and others have shown.

Freund[1] (97) and *Hildebrandt*[2] (98) have convincingly

[1] [*Freund*, Deutsche Naturforscher-Versammlung, Karlsbad, 1862; 'Verhandlungen der gynäkologischen Section,' S. 119.

The whole reference to this paper (which I succeeded in finding in the library of the Royal Society, after a fruitless search in those of the Royal Medical and Chirurgical and Obstetrical Societies, the College of Surgeons, and the British Museum) is as follows:

"Amtlicher Bericht über die sieben und dreissigtste Versammlung Deutscher Naturforscher und Aerzte in Karlsbad im September, 1862," Karlsbad, 1863, S. 295.

"According to Dr. Freund hydronephrosis is often found in cases of retroflexion of the uterus: its causes are:

"1. Actual narrowing of one or both ureters, *e.g.* from chronic changes in the pelvic cellular tissue.

"2. Dislocation of the uterus may cause kinking (Knickung) of the ureters. The ureter is often fastened to the side of the uterus after perimetritis; this would explain the symptoms of hydronephrosis during life, namely, violent pains in the loins passing downwards along the ureters, pains in the lower extremities, headaches, frequent desire to pass water, dyspeptic troubles—which have been hitherto, according to Dr. Freund, wrongly attributed to hysteria. The symptoms of hydronephrosis are intermittent, the quality of the urine resembles that in catarrh of the bladder. Dr. Freund believes that pyelitis is a frequent sequela; he mentioned a case which ended in cure. After kinking of the ureters hydronephrosis only occurs on the right side; after narrowing of the ureters, on both sides.

"As the 3rd cause, Dr. Freund cites atrophy and hypertrophy of the pelvic contents; the former often extending to the bones, the general suffering small. The origin of both is problematic."

This seems nothing more than a collection of general statements; the 'Official Report' contains not a single fact in favour of the theory laid down in the text.]

[2] [*Hildebrandt*, 'Volkmann's Sammlung Klinischer Vorträge,' Band i, Abth. 3, Gyn. No. 5, "Ueber retroflexion des Uterus."

"Frau H., who had suffered from annoying urinary troubles besides the usual symptoms of retroflexion, was sent to me by her family doctor to be examined for a somewhat voluminous soft tumour above the left Poupart's ligament. I found retroflexion, and gave as my opinion that the tumour was to be regarded as a dilatation of the ureter in consequence of the retroflexion. My diagnosis was verified easily and promptly. I first replaced the uterus with the sound, then introduced the cathether and drew off the water, pressing on the abdominal walls at the same time. The tumour diminished as I did so from the size of a child's head to that of an apple."

This case seems far from proven. To have proved Hildebrandt's point it would surely have been as well to pass the catheter *before* replacing the uterus; by his method he has destroyed the proof that the retroflexion had any connection with the urinary symptoms.

I have quoted both passages at length to enable readers to judge for

proved that the common *retroflexions of the uterus* produce hydronephrosis by kinking (Knickung) and downward dragging of the ureters.

A rarer cause of hydronephrosis in connection with the female generative organs is mentioned by *Stadtfeldt* (99), viz. the compression of the ureter by *parametric cicatrices*. I find a similar observation in *Sandifort* (100). Significant cases in point have been quite recently communicated by *Fränkel* and *Maass* (101) and by *Schottelius* (102). The latter found in a woman forty-three years of age the pelvis of the left kidney dilated into a sac holding six litres of fluid, the ureter somewhat dilated and tortuous, but admitting the passage of a probe as far as the bladder. The hydronephrosis was due to a displacement of the uterus, and fixation of the left ureter by dense connective tissue, the result of former parametritis, to the cervix, above the opening of the ureter into the bladder.

Parametritis may therefore produce obstruction of the ureter in two ways, either through direct compression of the corresponding ureter by the deposit, or by contraction of the cicatricial tissue drawing the uterus to its own side and thus dragging on the opposite ureter and occluding its calibre.

A *hæmatometra* may also produce hydronephrosis by compression of the ureter in the same way as a parametritis, as *Tüngel* (103) has shown.

One of the most important causes of hydronephrosis however, though hitherto little regarded, is *descent of the female generative organs* first indicated by *Virchow* (104). It seems all the more necessary to consider this cause in the present place, inasmuch as we have already recognised descent of the female generative organs as a cause of moveable kidney itself; so that the relations of descent of the female generative organs to moveable kidney are twofold, contributing as it does directly through traction on the one hand, and indirectly through the production of hydronephrosis on the other hand, to the mobility of the kidney.

We owe the first observation in which descent of the

themselves how far they consider that retroflexion of the uterus has been "convincingly proved" to be a cause of hydronephrosis. Descent of the uterus, with or without retroflexion, is another matter.—TRANSLATOR.]

female generative organs was recognised as a cause of hydronephrosis and confirmed by an autopsy, to *Virchow* himself, who found in a woman, forty-three years of age, the subject of an irreducible procidentia, the ureters *much constricted as far up as the sacro-iliac synchondrosis*, and the pelvis of the kidney dilated. This condition was explained by *Virchow* from the circumstance that the base of the trigone, that is the spot where the ureters open, was drawn forwards beneath the symphysis pubis, necessitating compression and therefore obstruction to the passage of urine. *Virchow* remarks, "The possibility of such obstruction, and the production of hydronephrosis in cases of prolapsus uteri of this kind, is well worthy of attention in practice, though not hitherto mentioned. It stands on the same footing as the hydronephrosis, which so very often accompanies cancer of the womb, and which is also usually disregarded." This hint by Virchow has, however, attracted little notice, for apart from his own observation I have been able to find only one solitary notice of the same kind by *Philipps* (105), who found in a 4 para thirty-five years of age (who had suffered since her first confinement from procidentia of the uterus and died comatose), the capsule of the kidney adherent, the glandular substance much contracted, the pelvis dilated, the pyramids flattened and changed into cavities, hardly a quarter of the glandular substance left, the mucous membrane of the urinary passages thickened, the ureters dilated to the thickness of a finger. The ureters and bladder contained milky purulent urine, the muscular tissue of the bladder was hypertrophied, the uterus lay in front of the vulva as large as an apple. The procident mass consisted of the neck and part of the posterior wall of the bladder in front, and of the posterior vaginal wall and greatly thickened peritoneal covering of Douglas's pouch behind. The orifices of the ureters were found beneath the pubic arch and had thus been exposed to a considerable pressure.

Although in this case the mechanical cause of the production of the hydronephrosis doubtless consisted in compression of the ureters against the pubic arch, as is proved by the dilatation of the ureters down to their vesical orifices, in other cases it seems to consist in occlusion of the

ureters by traction (such as may be produced by stretching an india-rubber tube) higher up than the point of application of the traction, as can be seen in *Virchow's* case.[1]

Physical causes.

We have so far studied the *primary* changes in the *direct* or *indirect means of fixation* of the kidney as producing its mobility, but in all cases pressure from above or traction from below must contribute to its development. These *physical* influences are competent, by themselves and *without any previous anatomical changes of importance,* to produce moveable kidney.

Thus *acute injury* is frequently cited as a cause. In this case, of course, just as in cancer of the breast, the bare statement of the patient that a tumour developed in the abdomen after a fall or a blow, &c., should not be implicitly believed. In the case of moveable kidney as well as in cancer of the breast, it is an external occurrence which first directs a patient's attention to the existence of the malady. The occurrence of this *luxatio traumatica renis* is established by trustworthy observations. Thus observations are quoted by *Rayer, Henoch, Ferber* and *Le Ray,* in which this complaint was acutely produced by a fall from a carriage or from horseback, or by a blow on the side. If it is remembered that isolated ruptures of the liver, spleen or kidney have been found after severe injuries, the possibility of the occa-

[1] [Attention should be called in this place to a remarkable class of cases in which marked dilatation of the ureters and hydronephrosis occur. These may be described as cases of urinary obstruction from irritation. The first division of the class concerns the subjects of incontinence of urine. Three cases are recorded by *Dr. Alexander James,* 'Edinburgh Medical Journal,' 1878, p. 135, in which death occurred, and in the two of these in which a post-mortem examination was obtained dilatation of the ureters and double hydronephrosis was found. The second division concerns the subjects of extroversion of the bladder, in which affection a similar condition is generally found (*Champneys,* 'St. Bartholomew's Hospital Reports,' vol. xvi, 1880, p. 111) in spite of the absence of any structural obstruction to the ureters. In both of these cases the obstruction is spasmodic, and the comparison of the two classes shows that its seat must be the orifices of the ureters. The bearings of this on the previous remarks in the text are obvious.—Translator.]

sional occurrence of isolated ruptures of the capsule of the kidney by a blow or its "contrecoup" will not be doubted. I myself have seen two patients in whom careful investigation proved the sudden appearance of an abdominal tumour, in one case after carrying a heavy weight with the trunk bent sideways, and in the other case after a fall, in both cases without any predisposing anatomical cause.

In a similar fashion other authors have seen moveable kidneys actually develop *in the course of labour* after severe bearing down; but in these cases it is questionable whether a moveable kidney already produced had not been pushed up during pregnancy by the growth of the uterus, and merely reappeared after delivery.

This complaint, however, is more frequently induced by *repeated injury*, especially by the shock of cough in bronchitis, pleurisy, whooping-cough, and particularly when favoured by other factors, such as the rapid emaciation of phthisis. Even *Riolan* and *Portal* drew attention to the importance of this influence, and they have been confirmed by the observations of *Le Ray*, *Defontaine*, *Olivier*, *Keppler*, and *Rayer*. I myself cannot doubt, when I consider that the kidney lies above the lowest part of the pleura, that every pleurisy with effusion must necessarily depress the kidney, so that under these circumstances, violent concussion of the diaphragm may very easily produce mobility of the kidney.

But repeated exertions, such as prolonged and severe labour, lifting great weights, carrying heavy children, violent straining at stool, may act in the same way as fits of coughing in loosening the attachments of the kidney and thus contributing to its mobility.[1] Literature contains many vouchers for their occurrence. Thus a patient under my care, in whom no predisposing anatomical cause of moveable kidney could be ascertained, had been used to carry heavy burdens on the hips, with the trunk bent sideways.[2]

[1] [The steady squeeze, presumably equal in all directions, produced by any of the above methods is surely a very different thing from shocks, such as that of coughing, blows, or falls. This has been remarked in a previous note—TRANSLATOR.]

[2] [This unsymmetrical position may have been a very important factor in the case.—TRANSLATOR.]

Finally, the *stays* have been quoted from the most various quarters as an important factor in the production of moveable kidney. *Cruveilhier* especially remarks; "J'ai rencontré plusieurs fois chez les femmes qui usent de corsets fortement serrés, le rein droit dans la fosse iliaque du même côté. Ce déplacement arrive, lorsque par la pression exercée par le corset *sur le foie*, le rein est forcé de l'espèce de loge qu'il occupe à la face inférieure de cet organe, à peu près comme un noyau entre les doigts qui le pressent."

If we consider, however, that many women who have moveable kidney have never worn stays and that only a few of those who wear stays get moveable kidney, it follows that this article of clothing, as such, is guiltless of the production of moveable kidney. Neither can the laced bodices (Schnürleibchen[1]) on which blame is laid by *Müller-Warneck* be allowed to rank as causes. This author, as well as *Bartels*, assumes that lacing of the bodice will directly compress the kidneys; but this assumption is incorrect, for the bony thorax, within which the kidneys lie, and round the lowest part of which the laces are braced, is far too rigid to pass on the pressure to the subjacent organs. The damage which the articles of apparel mentioned above do nevertheless inflict consists far more in this, viz. that too tight lacing round the thorax prevents its expansion on inspiration and obliges the diaphragm to descend deeper by way of compensation and thus to depress the subjacent organs, the liver, spleen and kidneys. Again, the bodices (Schnürmieder[1]) may produce an injurious effect directly on the kidney in cases in which the kidney has already become moveable and lies against the anterior abdominal wall. It is especially important to remember that, when once a kidney has become moveable, a number of other factors contributing to the further development of its mobility come into play, which, so long as the kidney remains in position, have absolutely no effect in producing its displacement. This is true, for

[1] [*Schnürleib, Schnürmieder*, are articles of clothing unknown in England. They are laced bodices of simple construction, and worn still very generally. The lower orders wear them often outside their other garments. Similar bodices are worn outside by the Italian peasantry, and form part of the proper dress of Margaret in 'Faust.'—TRANSLATOR.]

instance, of the movements of the spinal column, which, as we have already seen, is to be regarded as immoveable only in the region occupied by the kidney in its normal position. Besides, a well-fitting pair of stays, which is only used for supporting the breasts and strings of the garments, is, as we shall see, much more calculated to prevent than to produce mobility in a normally placed kidney.

It now remains to state plainly the causes which especially contribute to the mobility of the *right kidney*. This position is all the more forcible inasmuch as, with the exception of sudden violence affecting one side, the causes of mobility hitherto considered act equally on both sides of the body.

(*a*) The assumption that the right kidney is the larger and heavier is disproved by numerous determinations by *Rayer* and *Sappey*.

(*b*) *Guéneau de Mussy* assigns the usual tendency of the uterus to rise in pregnancy towards the right side as a cause, without further explaining the connection.

(*c*) *Lancereaux* thinks that there is possibly a special connection between the right ovarian and renal plexus, producing at every menstrual period congestion and consequent expansion of the capsule of the right kidney.

(*d*) *Cruveilhier* in this connection also makes stays answerable for depressing the right kidney, on the ground that their pressure squeezes the kidney beneath the liver, just as a slippery cherry-stone is flipped between two fingers, whereas the left hypochondrium, occupied as it is by the spleen and cardiac end of the stomach, bears the pressure of the bodice with impunity.

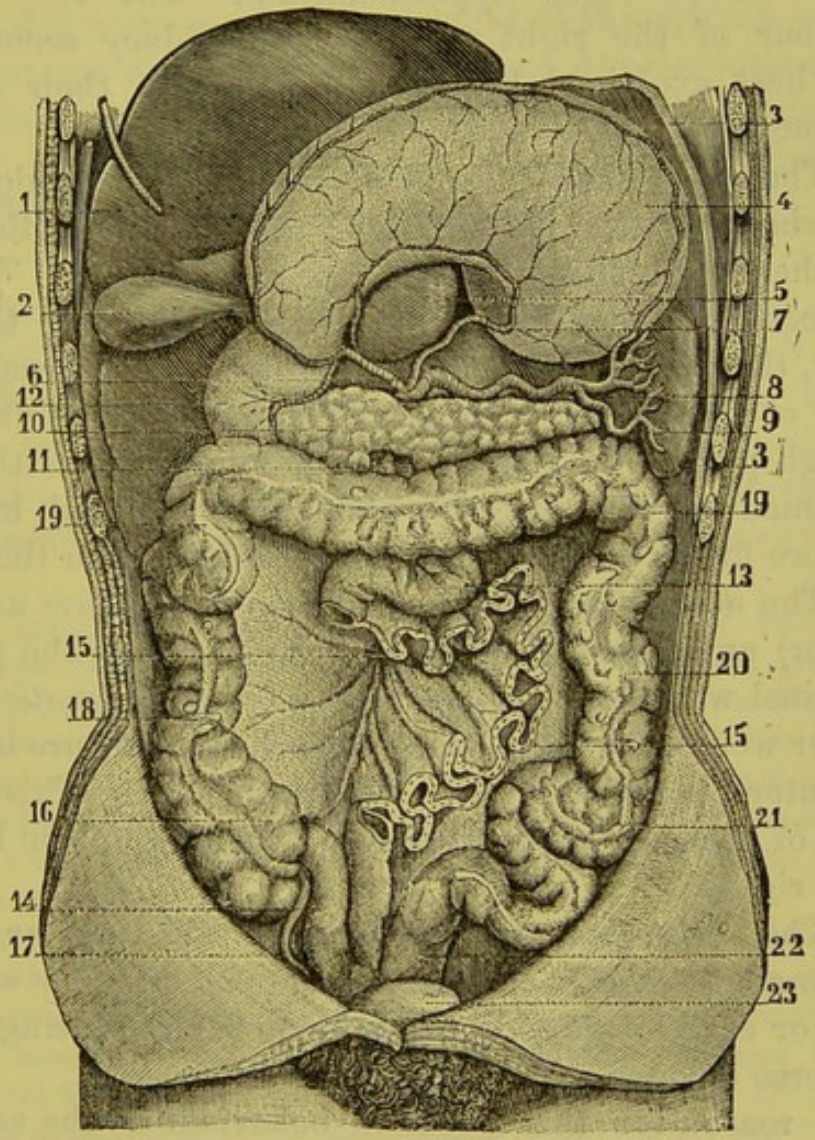
(*e*) According to *Müller-Warneck* also the liver, and therefore the "right kidney which normally lies distinctly the lower down," are specially affected by laced bodies. This supposition is, however, not to the purpose, for normally the right kidney lies very slightly if at all lower down than the left, so that the left kidney being deprived of the protection of the bulky liver must be much more exposed to pressure than the right.

(*f*) Other authors make the liver alone answerable for the frequency of moveable kidney on the right side, but finally acknowledge with *Rollet* that: "The reason why the right

kidney becomes moveable far oftener than the left is not as yet sufficiently explained."

(*g*) It seems, however, to me that the explanation of this peculiar relation ought not to be sought, as has hitherto been the case, in causes which would seem to imply that the right

FIG. 3 (*after Sappey*).



1. Lower surface of the liver.
2. Gall-bladder.
3. Section through the diaphragm.
4. Posterior surface of the stomach turned up.
5. Lobus Spigelii.
6. Cœliac axis and hepatic artery.
7. Coronary artery of the stomach.
8. Splenic artery.
9. Spleen.
10. Pancreas.
11. Superior mesenteric vessels.
12. Duodenum.
13. Upper end of jejunum.
14. Lower end of small intestine passing into colon.
15. Mesentery.
16. Cæcum.
17. Vermiform appendix.
18. Ascending colon.
19, 19. Transverse colon.
20. Descending colon.
21. Sigmoid flexure.
22. Rectum.
23. Urinary bladder.

kidney is exposed to greater pressure than the left. Other things being equal, the left kidney would be much more easily displaced laterally in the left hypochondrium which is not occupied by firm abdominal glands, than the right, for the right kidney could only be displaced by more intense pressure or traction from under the liver, which occupies the whole of the right hypochondrium. But the different behaviour of the right and the left kidney seems to me to be best explained by the differences in their mode of attachment.

1. The *upper end of the descending colon* lies higher and is attached to the ribs further to the left and further down (seitlicher und tiefer) than the ascending colon. Whereas the ascending colon lies against the middle of the right kidney, the descending colon lies against the external convex border of the kidney. Hence it follows that the left kidney, although placed on the same horizontal level as the right, is fastened to the posterior abdominal wall both higher up and more firmly (höher hinauf und stärker) than the right.[1]

2. The *descending colon* and the *splenic flexure* are tauter (straffer) and shorter in their attachment to the posterior abdominal wall than the *ascending colon* and *hepatic flexure;* in other words, the mesocolon of the splenic flexure is shorter and tauter than that of the hepatic flexure. This indirect means of attachment is therefore stronger on the left than on the right.

3. The *ascending colon* does not form a right angle with the *transverse colon* as the *descending colon* does, but an obtuse angle, or even forms a dependent loop before passing into the transverse colon.

The reason for this peculiar behaviour of the ascending colon, which I have seen a few times in children, and many times in adults, and which *Sappey* has described as normal, appears to me to be the upward movement of the fæces in

[1] [This sentence, which is somewhat obscure, seems to mean that the colon (which by means of its attachments to the posterior abdominal wall on the one side and to the kidney on the other, is one of the defences of the kidney against downward displacement) is itself attached to the posterior abdominal wall higher and more extensively on the left side than on the right.—Translator.]

the ascending colon contrary to the action of gravity, and only effected by the peristaltic movements of the bowel. By this means the spot where the ascending becomes the transverse colon is gradually dragged downwards so much as to form in extreme cases a fairly long loop, though it usually passes obliquely or with a curve into the transverse colon.

But during the passage of the fæces from the transverse into the descending colon, the weight of the column of fæces and peristaltic action work in the same direction, no stagnation takes place in the splenic flexure, which maintains its original form. This difference between the hepatic and splenic flexures explains also in its turn how the right kidney has more room to descend than the left.[1]

4. The descent of the left kidney is opposed by *the position of its vessels* above the *inferior or horizontal part of the duodenum*. Moreover, the position of the aorta on the left side of the spinal column makes *the left renal artery* shorter than the right; this shorter attachment by means of the renal artery (which is naturally considered in this capacity before

[1] [This explanation seems highly questionable. In the first place it belongs to the category of "fæcal theories" which are pressed into service on very small provocation, and have, generally speaking, no positive evidence in their favour. Among the unsymmetrical conditions which they have been called in to explain may be mentioned varicocele (left), uterine obliquity and torsion (to the right), and torsion of ovarian tumours, which this is no place to discuss; but with regard to the question now before us the following objections may be urged:

a. It is probable that the question of ascent or descent of the fæcal column has very little to do with it. Take a familiar instance—a monkey climbing up a rope and then down it, the rope being connected with a spring weighing machine; how much difference in tension will there be? The only point of difference, addition or deduction, in the case of the colon will be the rate of movement of the fæcal column. How fast is that?

b. If this explanation were correct we ought constantly to find the ascending loops of intestine full, and the descending empty, or at least one fuller than the other. Where is this evidence?

c. This explanation assumes a permanently perpendicular position. This ceases for about one third of each day at least. Again, here we ought to have evidence of a difference between the position of fæces in bed-ridden and other people. Where is this evidence? Is it not, on the contrary rather, these very bed-ridden people who are predisposed to moveable kidney?

In the absence of direct proof, and in the face of the above considerations, we think this explanation ought not to be entertained.—TRANSLATOR.]

the thin-walled vein) renders the mobility of the left kidney the more restricted. Less stress must be laid on the circumstance pointed out by *Oerum-Howitz*, that the left suprarenal vein opens into the renal vein, while the right suprarenal vein opens directly into the vena cava, the left kidney (as opposed to the right) having thus an additional indirect connection with its suprarenal body (see Figs. 1 and 2).

5. The left renal vessels are closely connected by cellular tissue with the head and neck of the pancreas, which thus furnishes an additional attachment to the left kidney, of which the right is destitute; for the right renal vessels lie bare, except for their peritoneal covering (see Figs. 1 and 2).[1]

These reasons seem to me to explain sufficiently why descent of the left kidney, though not impossible, is considerably rarer, other things being equal, than that of the right.

The *direction* in which the kidney is permitted to move is prescribed by the normal attachments and normal position of the kidney, and is nearly constant. Since the insertion of its vessels (*i.e.* the aorta and vena cava at the level of the first lumbar vertebra) is a fixed point, it follows (presuming the most favourable conditions for mobility, namely the complete separation of the renal vessels from the posterior abdominal wall) that the right kidney is capable of moving

[1] Conf. *E. Zuckerkandl*, "Beiträge zur Anatomie des menschlichen Korpers." 'Wiener. med. Jahrb.,' 1883, S. 58; 'Centralblatt med. Wiss.,' 25 Aug., 1883, S. 611.

If, after removal of the capsula adiposa, the right kidney is raised from the posterior abdominal wall, a membrane composed of connective tissue and varying in thickness is seen, which passes into the parietal peritoneum at the borders of the kidney and suprarenal body. This fascia is entirely unconnected with the aponeurosis which covers the anterior surface of the quadratus lumborum. Thus, the right kidney is enclosed in a capsule (besides the capsula adiposa and fibrosa), the anterior wall of which is formed by the peritoneum, and the posterior wall by this post-renal fascia. In the case of the left kidney the colon runs along its side and not in front of it, as it does on the right side. Thus, the front of the capsula adiposa of the left kidney is covered not only by the parietal peritoneum, but also by the descending mesocolon, and these are joined together. This, however, applies only to the portion of the left kidney which lies beneath the transverse mesocolon, while above this the pancreas lies. The left kidney thus appears decidedly more firmly fixed than the right, and this is perhaps the reason why the right kidney is oftener moveable than the left.—[Translator.]

within the segment of a sphere of which the centre will be the entrance of the right renal vein into the vena cava inferior, and the left within the segment of a sphere of which the centre will be the origin of the left renal artery from the aorta. Hence, it follows that the presumption of some authors, among whom is *Sappey*, that a moveable kidney moves vertically downwards towards the corresponding iliac fossa, is not correct. It is much nearer the fact that it can only move downwards, forwards, and inwards; its movement upwards and outwards being impossible on account of the presence of the spleen, liver and diaphragm, not to mention other reasons.

The *degree* of its mobility depends not only on that of the relaxation of its capsule and vessels from the posterior abdominal wall, but also from the resistance of the organs lying below it, the amount of the pressure of the abdominal walls, and the weight of the kidneys.

Again, the kidney, as it descends, never maintains its original relation to external dimensions (wird sich . . niemals sich selbst parallel nach unten bewegen), but is compelled to rotate, as it descends, on one or more of its *axes*, by virtue of the obliquity of the planes over which it glides, as well as by changes in the attitude of the body. The more it descends, the more transversely will rotation round its centre cause its long axis to lie; the farther forward it advances, the more will its outer border and its upper end look forwards.

The *deepest possible position* of the kidney is determined by the length of its vessels, which, together with the ureter, suffer manifold torsions and kinks during the displacement of the kidney, varying according to its depression and rotation round its axes, as indeed might have been assumed *a priori*.

VII. Symptoms.

The opinions of authors as to the clinical consequences of moveable kidney are very various. *Rosenstein* observes: "More important than congenitally displaced kidney is

moveable kidney, although it properly possesses rather *a negative than a positive interest*, so that a knowledge of the facts in point is more important as a means of avoiding errors in diagnosis than on its own account, since *the affection itself causes no great inconvenience*, and treatment is powerless to relieve it."

This view is adopted by most of the handbooks and text-books, while *Keppler*, on the contrary, regards even uncomplicated moveable kidney as a deadly disease, which should be extirpated whenever it gives rise to symptoms.

It is therefore most important, considering such opposite opinions, to obtain clear views of the symptoms and consequences of moveable kidney.

It is evident from the study of its pathogenesis that acquired moveable kidney is but very rarely to be described as the primary lesion. We often find with it maladies, which have either arisen from similar causes to those which produced the moveable kidney itself, or have themselves produced the moveable kidney. Moreover, the degree of mobility and the anatomical consequences of moveable kidney, as well as individual susceptibility, are different in different persons, so that moveable kidney calls up no idea of a typical disease but rather the idea of a disease of many various forms. It is, therefore, more necessary to individualise with regard to this than with regard to almost any other complaint.

It is rare for a moveable kidney to leave the patient quite unaware of its presence. This is most likely to happen in the case of persons who are otherwise very healthy, and who, as we know, sometimes feel no inconvenience from other displacements, such as those of the womb, or from ruptures; or it may happen in those who are very ill, as for instance in consumptive patients, in whom the symptoms of moveable kidney are obliterated by the principal complaint.

Generally, however, moveable kidney exercises an injurious moral and physical effect on the patient in its capacity as a foreign body, by dragging and compression of nerve-trunks, vessels and viscera, and by functional disturbances.

Symptoms arising from the Nervous System.

Patients who are the subjects of moveable kidney are usually affected with hypochondriasis, especially if they discover a tumour in their abdomen by accident, and this frame of mind increases if the malady is diagnosed as a malignant growth by the doctor, or if, in spite of a contrary opinion on his part, the patient takes it for one. This unfortunate humour finds tangible pabulum if the patient feels pains in the abdomen, whether due to the tumour or other causes, and often grows so much on her that she readily decides of her own accord to have the moveable kidney removed. This condition, which is seldom found even in the case of malignant tumours, can easily be explained when one considers that these patients are reminded by a moveable kidney more than by any other tumour—at every step, on dressing and undressing, even on turning in bed—of the presence of an apparently serious malady ; and in this view they are often confirmed by doctors.

Some patients complain of a feeling of pressure and weight, of dragging and drawing in the lower abdomen, they feel "as if something had been unhooked in their belly." Others feel beating, or a more circumscribed gnawing griping pain in the region of the navel. These unpleasant sensations are compared by women who have had children to fœtal movements, and they sometimes think themselves pregnant, especially if they wish to be so. Among other characteristic expressions may be mentioned the feeling as if one of their sides was dropping off, or as if something was turning round in their belly. Often, but wrongly, these complaints are regarded as hysterical. No doubt among the women who have moveable kidney some are hysterical, but we should not agree with *Chroback* (who in nineteen cases saw hysteria eight times) or with *Lancereaux* (who saw it in cases of moveable kidney four times), in considering the moveable kidney as the cause of the hysteria. It is intelligible that the imagination of an hysterical patient will be unusually excited by the presence of a moveable tumour in the abdomen, but the painful and unpleasant sensations caused by moveable kidney ought not

to be regarded as hysterical, inasmuch as we find them of the same kind and intensity in women who are not hysterical, and in men. One sign that these painful sensations are not hysterical is that they are increased by sudden movements or great bodily exertions, but in the recumbent position and during rest they disappear.

Besides these vague, insufficiently localised sensations of pain, one not uncommonly observes *circumscribed neuralgias* in remote spots, arising reflexly through dragging on the nervous plexuses and ganglia which plentifully surround the kidney. Now the capsule of the kidney normally lies upon the two last branches of the lumbar plexus, and is surrounded by a rich network of sympathetic ganglia. Thus we often find pains down the front of the leg as far as the knee, between the ribs, in the loin, and in the abdomen. Sometimes these radiate as they do in liver affections to the scapula, sometimes along the ureter as far as the labia majora. These neuralgias are usually confined to the affected side, more rarely they are transferred to the opposite side. *Güeneau de Mussy* observed intercostal neuralgia of the left side in a moveable kidney of the right side; and among my observations are two in which there was lumbar and intercostal neuralgia on the left side, the right kidney being moveable. On pressing firmly against the kidney, which is easily manipulated, both patients alike complain of circumscribed pain in the region of the opposite kidney.[1]

Menstruation produces increase of pain and impairment of the general comfort. We then observe the same phenomena as those described by *Matthews Duncan* under the name of aching kidney, these complaints consisting essentially of dull pain in the region of the kidney radiating thence into the bladder and sacrum. As a matter of fact this pain can almost constantly be found in women with moveable kidney at the menstrual period, even when they are free from it betweenwhiles. Whether it is induced more by menstruation or by the moveable kidney cannot now be decided. The fact, however, that it sometimes quite ceases after menstruation and after the menopause, proves the existence of a

[1] [The same transference is sometimes observed in the case of the ovary.—Translator.]

vasomotor connection between the generative organs and the kidney, that is between the ovarian and renal plexuses, as was supposed by *Lancereaux* and *Fourrier*.

It is rare for the pain in the renal region to cease during menstruation, as it did in some observations by *Grout* and *Le Ray*.

The recumbent position alleviates the diffuse pains as well as the circumscribed neuralgias, as set forth by *Müller* (106) with regard to the lumbar neuralgia which frequently occurs with moveable kidney.

Symptoms arising from the Great Vessels.

Although the above symptoms arise principally from pressure and traction of the great nerve-trunks and small nerve-fibres, we sometimes meet with symptoms of *pressure by the kidney on the great vessels*, especially the easily compressible vena cava, with subsequent thrombosis from pressure and œdema of the corresponding lower limb. A very instructive case is communicated by *Girard* (see above, Case 3), only it is doubtful whether in this case the intestines distended with gas, or perhaps some adhesions had not pressed the kidney against the vein. I have observed one case of moveable kidney on the right side with subsequent œdema of the right lower leg, for which no reason could be found except the pressure of the kidney on the vein. The œdema began to disappear as a pregnancy, which had supervened, advanced.

Symptoms arising from the Digestive Tract.

Very many persons affected with moveable kidney suffer from gastric disturbances, varying from slight epigastric pains and nausea, to phenomena which sometimes amount to the well-marked signs of chronic gastric and intestinal catarrh, jaundice, or even attacks of peritonitis.

These gastric disturbances depend on complaints which happen to coincide, or they are consequences of causes similar to those which produce moveable kidney. When, therefore, diseases accompanied by rapid emaciation, such as enteric or severe intermittent fever, or rapidly succeeding labours,

prolonged suckling, or generative affections of all sorts have preceded or still persist, we are hardly justified in calling moveable kidney the cause of the gastric disturbances. A primary chronic gastro-intestinal catarrh or a gastric ulcer may, however, so impair nutrition that these affections may be justly regarded as furnishing a cause for the mobility of the kidney, rather than as being themselves caused thereby. When, therefore, *Keppler* attributes the disturbances of nutrition, emaciation, even death to a moveable kidney, it is necessary for us to be emphatically warned against these practically injurious exaggerations, as has been done by *Oerum-Howitz* with reference to the cases described by Keppler. The real ill-consequences of moveable kidney, even in its bearings on the digestive tract, are only obscured by such performances.

As a matter of fact moveable kidney may contribute to gastric disturbances, as might be assumed beforehand from the nervous and vascular connections between the renal and other abdominal organs. This connection is completely confirmed by clinical observation, inasmuch as persons with moveable kidney experience epigastric pains and digestive disturbances which are otherwise inexplicable. It is often possible by pressing on the prolapsed kidney to produce epigastric pain, nausea, and retching, in these patients, to reproduce in short exactly the same condition which they will tell you unasked is that from which they are suffering.

A very plausible theory has lately been suggested by *Bartels*, of Kiel, to explain the frequent coincidence of dilatation of the stomach and subsequent gastric and intestinal catarrh with moveable kidney on the right side. According to this, the right kidney displaced forwards and inwards by the pressure of tight lacing (as described by *Müller-Warneck* (107)) compresses the fixed descending portion of the duodenum which lies between the hilum of the right kidney and the vertebral column, and thus produces a mechanical obstacle to the escape of the chyme from the pylorus, and dilatation of the stomach with its well known consequences.

Oser (108) has rightly opposed this hypothesis, and especially on clinical grounds. He thinks it is self-evident that a moveable tumour which has a tendency to sink either

downwards or backwards as the patient stands or lies, can hardly exercise such a pressure on the bowel as to produce obstruction while the abdominal walls are lax. In spite of the opportunity of observing very many cases of moveable kidney, he never once found hypertrophied stomach, which is usually found in cases of stenosis of the pylorus. Finally, he proved the complete competence of the pylorus by directly distending the stomach with air, in cases of moveable kidney in which another method had seemed to prove its incompetence, and, therefore, the truth of *Bartels'* hypothesis.

On the whole *Oser* considers the causal connection between displacement of the kidney and distention of the stomach as not proven, and only allows the possibility of this connection in cases in which the displaced kidney is so firmly fixed in its new position that it is able to compress the duodenum.

The principal objection, however, to *Bartels'* assumption is its inadmissibility on anatomical grounds. The right kidney even *when in its normal position* lies with only its *upper half* parallel to the descending portion of the duodenum, as Fig. 1 shows, and, when moveable, lies with its upper end below the inferior horizontal part of the duodenum, so that the two organs are actually not in contact. Besides, the specific gravity of the kidney, which alone can be considered as contributing to real compression (the intra-abdominal pressure being probably somewhat the same as that of water), is far too low to exercise such compression, so that a force is still needed to press the kidney against the gut, and this force is wanting.

Moreover, since chronic catarrh of the stomach is found even when the moveable kidney is on the left side, in women who do not lace, and in men who have moveable kidney, it is evident that another explanation of general application must be sought for the connection between gastric disturbances and moveable kidney. Such an explanation seems to me to be easily found in the fact that the posterior parietal layer of the peritoneum covers at once the anterior layer of the capsule and vessels of the kidney and passes over the duodenum, so that if the kidney becomes displaced forwards, inwards and downwards, a tendency to dragging and kinking of the limbs of the duodenum both on the right and left side

must be produced, especially as these limbs, but particularly the flexure between the duodenum and jejunum, are fixed by somewhat firm connective tissue to the vertebral column, and are comparatively unyielding (see Fig. 1). Of course this kinking should not be represented as permanent, as the kidney changes its place with various movements of the body, and in the recumbent position generally returns to its normal position, thus instantly relieving the dragging produced through the peritoneum. When, however, this dragging and the consequent obstruction to the calibre of the gut are frequently repeated as the erect position is assumed, and when other influences, such as pendulous belly, which acts in a similar manner, supervene, distention of the stomach and chronic catarrh of the stomach will not fail to appear.

With respect again to the *jaundice* which occurs in moveable kidney, direct compression of the common bile duct by the right kidney has been cited as the cause of the jaundice by *Litten* (109), who observed in *Frerichs'* clinique (in the case of a woman, thirty-seven years of age, suffering from frequent attacks of pain in the right hypochondrium induced by a moveable kidney on the right side) a jaundice recurring twice within two weeks and only lasting a few days. *Litten*, who was unable to find a similar observation on record, put aside the idea of gastro-duodenal catarrh and of gall-stones as a cause for the jaundice, and assumes a temporary compression of the common bile duct.

Stiller (110) had already alluded to the possibility of this connection, when he replied to the assumption by *Bartels* of compression of the duodenum by the moveable kidney, that, if the descending portion of the duodenum were really compressed by the kidney, this same pressure must produce jaundice by pressing on the diverticulum Vateri (the common gall and pancreatic duct) in this situation. This objection, however, according to *Stiller* is enfeebled by the anatomical consideration that the right kidney in becoming moveable must sink down along the right or outer surface of the duodenum, and the compression can hardly be so complete as to close the orifice of the common bile duct which lies on the opposite or left surface. Besides (says Stiller) the subject is at present too novel for us necessarily to accept the

assumption that as cases in point are collected we shall have to recognise this as an additional cause of jaundice.

Although I have seen jaundice supervene on three occasions in women affected with moveable kidney (in one of them four times within a short space, lasting on each occasion a few days), I cannot regard these cases as a clinical proof of temporary compression of the common bile duct, as this is impossible for the same reasons as the direct compression of the duodenum. How, moreover, can we explain an isolated compression of the common bile duct which opens on the left side of the duodenum by a moveable and not fixed kidney on the right side! I am much more inclined to refer the frequent appearance and disappearance of the jaundice, which I also saw in my case, to a temporary occlusion of the duct by mucus or a gall stone, particularly as this peculiar appearance and disappearance of jaundice is not a very rare occurrence even among women who are not the subjects of moveable kidney, as I have often had occasion to observe.

It is, however, certain that women who have moveable kidney are more prone than others to jaundice, but only because they more frequently suffer from gastro-duodenal catarrh, for the reasons above enumerated, and because a moveable kidney not infrequently contracts adhesions to the gall-bladder. Thus, in a case observed by me (see below), in which there was jaundice which persisted several weeks, the cause for the jaundice appeared to be connected with a firm adhesion between the moveable kidney and the lower surface of the liver and gall-bladder (mit dem unteren Leberlappen und der Gallenblase).

Still more serious disturbances are said to be produced by moveable kidney through compression and consequent obstruction of the colon. *Rollet* relates a case in *Oppolzer's* clinique of a woman, twenty-six years of age, and apparently consumptive, who had a moveable kidney, and who had suffered many years previously from small-pox, later from enteric fever, and in early years had been in the habit of masturbating. Inasmuch as she in the meantime, up to six months previously, had suffered (especially after bodily exertion such as dancing) from violent colic, and since her admission into the hospital had frequently suffered from

obstinate constipation often lasting six or seven days, from frequent colic, swelling of the abdomen, retching, desire to vomit, and sensitiveness of the abdomen, the following diagnosis was made: Intestinal obstruction, caused by pressure exercised by the kidney on the ascending colon. This case, however, is so far from proving obstruction of the colon by the kidney that it ought not to be let loose on literature without a query, until it is proved by an appropriate autopsy that the improbable assumption is true, viz. that the colon can be permanently compressed by a moveable kidney. The origin of such a compression in adhesions formed by the moveable kidney is not unlikely. Certain it is, however, that a moveable kidney is competent to produce (by mechanical injury and dragging on the bowels) colics and dull boring pains in the abdomen, which are not otherwise easily explicable, and which we not infrequently find in patients with moveable kidney, without any other discoverable cause.

The Symptoms of Renal Incarceration so-called.

Patients with moveable kidney are subject, generally after violent exertion or rapid movement, to a sudden piercing pain in the abdomen, which forces them to lie down. Soon the whole abdomen becomes sensitive and distended, and the side (especially in the region of the prolapsed kidney) becomes painful on palpation. A profound feeling of general discomfort follows. The patient is weak, giddy, palpation of the renal region easily makes her faint, a cold sweat covers the brow, the pulse is small, the respiration superficial. Sometimes there is a desire to vomit, actual vomiting often supervenes. The urine is dark and scanty, often coloured red from the admixture of blood. The attack is not always ushered in by a rigor, in fact the pyrexia is slight throughout. The observer detects (as far as the tension, resistance, and tenderness of the abdominal walls allow him) in the region of the prolapsed kidney a large tumour, confined to the corresponding side, over which percussion is impaired, and which is hardly moveable.

In the course of one or two weeks, however, this tumour

entirely disappears, as all observations agree in stating, the threatening symptoms having reached their acme on the fourth to the sixth day. One of the first signs of recovery is the copious excretion of clear urine of low specific gravity.

Dietl, who first directed attention to this hitherto unknown train of symptoms and gave it the name of renal incarceration, considers its essence to consist (as in the case of ruptures) in incarceration of the kidney in the surrounding connective tissue and peritoneum, with subsequent circumscribed peritonitis with effusion. He is followed by nearly every writer. Thus *Rollet* gives the following explanation: "In order to understand the symptoms of renal incarceration we need only consider the physical processes which must take place when the kidney (under bodily exertion, violent straining, shock, or congestive swelling at the menstrual period, &c.) changes its place and is displaced from its normal position with more or less force. Apparently it must first make its way within the connective tissue surrounding it, and in which no vacant space exists. It is inevitable during this that parts of the surrounding connective tissue, and even the peritoneum passing over it, should be dragged and pressed, and the vascular arrangements disturbed in some manner. In consequence of the irritation and vascular disturbance in the surrounding connective tissue, produced by changes in the position of the kidney, the connective tissue becomes the seat of inflammatory swelling. The kidney is all the more likely to stick in the narrow meshes of the sub-peritoneal cellular tissue and to become really incarcerated there, as its retreat into the normal situation is in a moment cut off. The symptoms caused by the incarceration of a moveable kidney tally with this representation of facts. If the incarceration of the kidney is not reduced early enough, either spontaneously (by the recumbent posture, observation of strict diet, and softening fomentations), or artificially by gentle or, if the diagnosis is quite certain, even powerful pressure on the kidney in the contrary direction, not only does the cellular tissue surrounding the kidney become inflamed, but the inflammation easily spreads to the peritoneum and a more or less violent circumscribed peritonitis supervenes, with inflammation of the capsule and parenchyma of the kidney."

Ebstein accepts this view, and considers the clinical symptoms as produced by the production of a more or less considerable peritoneal effusion.

According to *Gilewski* the incarceration of the kidney is produced by the wedging of the kidney between the last rib and vertebral column, and by its being held there by the morbid contractions of the abdominal muscles. In a case observed by him this mode of incarceration is said to have been favoured by a curvature of the spine. The kidney thus incarcerated pressed the ureter against the vertebral column, produced pyelitis, acute hydronephrosis, and the symptoms above described.

Oerum-Howitz accept this explanation only in part.

To me, neither the character of the acute attacks described (pointing to the incarceration of the kidney), nor the clinical symptoms themselves (referred to the supervention of circumscribed peritonitis, or, according to *Gilewski*, to acute hydronephrosis) appear to be correctly explained.

It is *a priori* unlikely that the kidney should become incarcerated in the cellular tissue which is everywhere so yielding, and especially in the neighbourhood of parts so soft as the intestines. But granting that it could become wedged for a moment between the vertebral column and ribs ; the force to hold it fast in this position would fail, for one cannot possibly assume with *Gilewski* a contraction of the abdominal muscles lasting for days. A wedging of the kidney between the vertebral column and the anterior surface of the arch of the rib in the healthy skeleton is, however, impossible, because its longest diameter is less than the distance between the parts mentioned. On the supposition of an incarceration it would be most remarkable that the train of symptoms under discussion should, so far as at present observed, invariably end in recovery without any active treatment, and it is not apparent how an incarcerated kidney could free itself from its incarceration without external interference. Finally, the incarceration of a kidney would certainly be a far more frequent occurrence than it actually is, considering that dragging of the cellular tissue and peritoneum takes place in every case of moveable kidney.

Taking the analogy of a strangulated[1] hernia the symptoms consequent on incarceration of the kidney have been explained in this bearing also by the supposition of a circumscribed peritonitis. This idea has been strengthened by the resemblance of the clinical symptoms to those of circumscribed peritonitis due to other causes, such as perimetritis and perityphilitis, by the supervention of great distention, which was taken for effusion, and finally by the fact now and then observed that during and after an attack the mobility of the kidney is restricted.

The invariably favourable course, however, of this supposed peritonitis with effusion, as well as the circumstance that the effusion, which is sometimes "enormous," disappears in from six to eight days, cannot fail to throw doubt on the correctness of this explanation. Besides, the peculiar outline of impaired resonance which is found diffused around the moveable kidney, does not admit of easy explanation on the assumption of *circumscribed* peritonitis.

The opinion of *Gilewski* that acute hydronephrosis produced by incarceration of the kidney constitutes the essence of the process under discussion, appears again unlikely, inasmuch as the weight of a normal kidney is far too small to be able to overcome by its pressure the pressure under which the urine is secreted into the ureter; besides an *acute* hydronephrosis is not competent to distend the pelvis of the kidney to the size found in those cases, and, moreover, it does not, as we know from numerous experiments, produce the symptoms we have named. A dog whose ureter has been tied, secretes, as *Cohnheim* (111) says (when the operation is not followed by inflammation, the general health remains unimpaired and the same food is taken), the same quantity of urine and that of the same specific gravity as before. And this not only in the first days and weeks after the operation, but also later, when the occluded kidney is attacked by

[1] [Great difficulty has been felt in deciding between "strangulation" and "incarceration" as the equivalent of "Einklemmung;" but the latter word has been chosen, as tallying best with the facts, as a word used synonymously with Einklemmung frequently in the text, especially in connection with the name of *Dietl*, the author of the term. But, in the present passage, the reference is obviously to the general class of Strangulated Hernia.—TRANSLATOR.]

hydronephrotic atrophy, becomes anæmic and ceases to secrete. But in the case of moveable kidney we see from the commencement disturbances in the secretion of the urine, amounting even to hæmaturia. In a woman, the subject of an uretero-vaginal fistula (112), moreover, I produced an unilateral hydronephrosis by the use of the probe, but this was not followed by the symptoms enumerated above. In unilateral hydronephrosis again produced unintentionally by sewing up the ureter in the course of the operation for vesico-vaginal fistula, I have seen symptoms develope which were essentially different from those under discussion.

We must, therefore, look out for another explanation of the production and of the clinical symptoms accompanying so-called renal incarceration, and *this I find in an intense local disturbance of circulation in the moveable kidney, caused by twisting or kinking or acute angular insertion of the renal vessels, especially the vein, in consequence of the change of position and rotation of the kidney.*

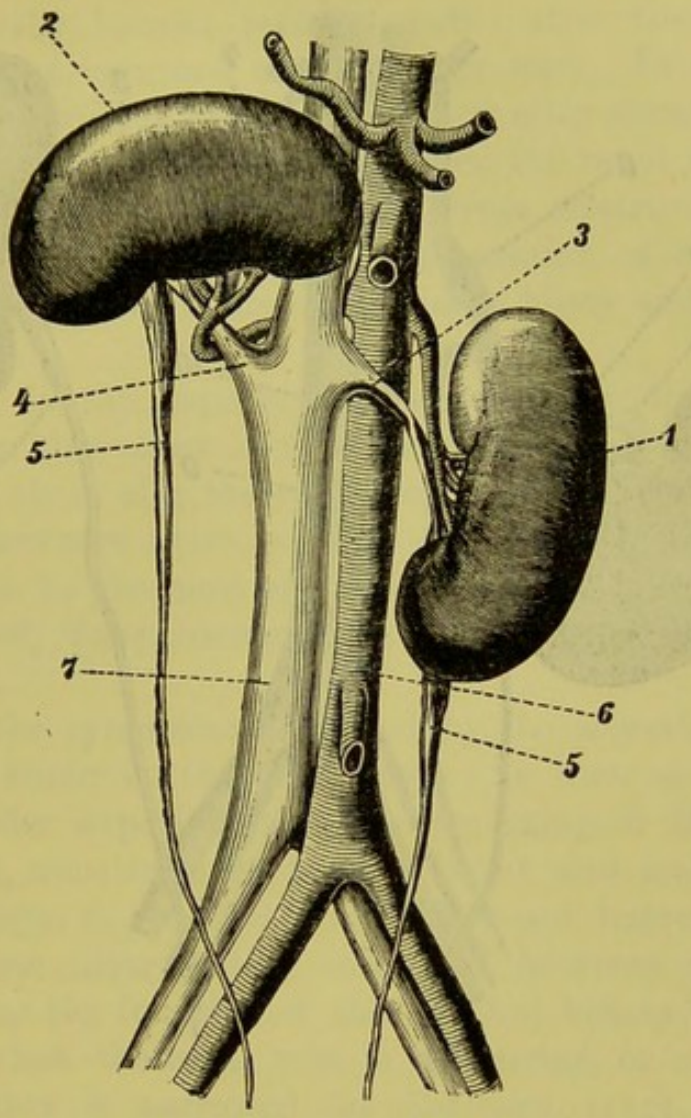
Although the authority of *Cohnheim* (113) (who thinks that renal obstructions due to *local* obstacles to the return of blood in the renal veins play hardly any part in pathology) is opposed to this supposition, its correctness seems to me to be supported by a series of important reasons.

We have already discussed the relation of the renal vessels and ureter during descent and rotation of the kidney, a relation which is of great importance for the train of symptoms before us. We have also already found the acute angular course and torsion of the renal vessels established by some autopsies. In order, however, to bring these relations more prominently into view I have got some preparations of kidneys made artificially moveable in the dead subject drawn, these preparations showing some kinds of twisting and kinking of the vessels (see Figs. 4 and 5[1]).

But my hypothesis is supported by experiment and by observation of the clinical changes even more strongly than by these drawings.

[1] I have to thank *Dr. Guttmann*, Director of the State Hospital at Moabit for furnishing me with a subject, and *Dr. Smidt* for help in preparing the dissections. Figures 4 to 8 are drawn after nature by *Herr Grohmann*.—AUTHOR.

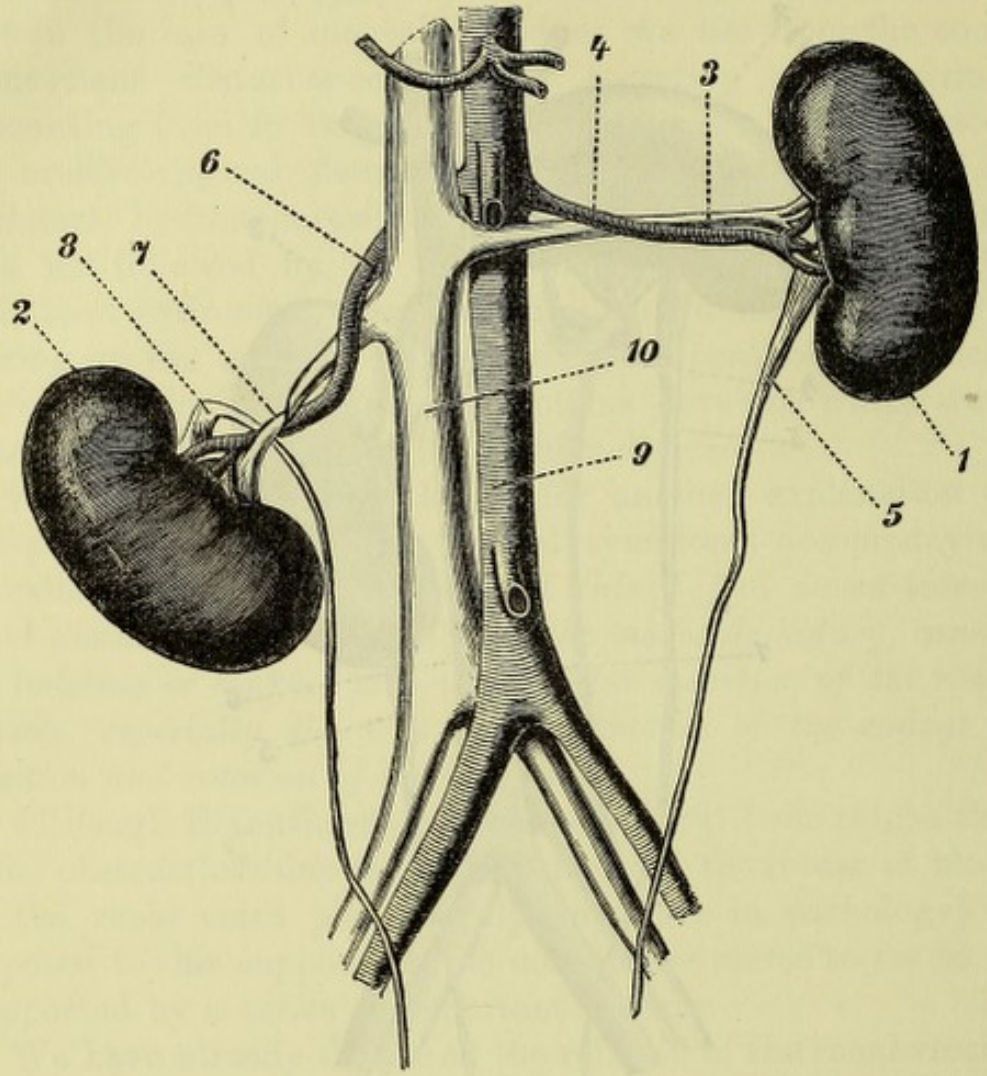
FIG. 4.



1. Left kidney.
2. Right kidney.
3. Left renal vein kinked and twisted.
4. Right renal vein compressed.
5. Ureter.
6. Abdominal aorta.
7. Vena cava inferior.

There are, perhaps, few pathological processes which have been so accurately investigated as the coarser disturbances of circulation in the renal vein. *Max Hermann* and *Ludwig* (114) found that after tying the renal vein the tubuli uriniferi became completely closed in consequence of the obstruction to the return of the blood, so that the secretion of urine ceased. If the renal vein became pervious again, the secretion of urine became quickly reestablished. *Litten* and *Buchwald* (115) who completely tied the renal vein, saw the kidney very quickly swell up, as the following example shows. Even within four hours of the ligature of the vein, the weight

Fig. 5.



1. Left kidney.
2. Right kidney.
3. Left renal vein.
4. Left renal artery.
5. Left ureter.
6. Right renal artery.
7. Right renal vein twisted and kinked.
8. The ureter bent round and wrongly inserted.
9. Abdominal aorta.
10. Vena cava inferior.

and measurements of the two kidneys bore the following proportions to each other:

Rabbit's Kidney.

	Side not ligatured.	Side ligatured.
Weight . .	4·7 grammes.	6·7 grammes.
Length . .	2·9 centimetres.	3·2 centimetres.
Breadth . .	1·4 „	1·5 „
Thickness .	1·5 „	1·6 „
Breadth of cortex	0·25 „	0·3 „

No inflammatory processes were remarked. The ligatured kidney usually became rapidly smaller after the sixth day, and very soon returned to its former size. In some cases the ligatured kidney became atrophied after some weeks.

Cohnheim (116) observes that when the renal vein is tied, the kidney swells up so rapidly from obstruction to the return of blood and from the supervention of œdema, that in less than an hour it may be twice as heavy and big as the other.

Just as in the experiment, we find clinically in human beings a sudden onset of the symptoms of incarceration so-called; in them also there developes in a very short time a tumour sometimes twice as large as the kidney itself, undistinguishable by the most accurate palpation from the kidney, and generally disappearing without leaving a trace in six or eight days.

Again, the symptoms furnished by the secretion of urine tally with those in the experiment; in men as well as in animals under experiment at the beginning of the attack a dark urine, sometimes containing blood, and scanty for the first few days, is secreted, which does not become replaced by a profuse clear watery urine till recovery has set in. Inasmuch as the function of the affected kidney is naturally impaired when the renal vein is obstructed, or even as soon as its calibre is narrowed (as *Robinson* (117), *Perls* and *Weissgerber* (118) found), the quantity of urine of course diminishes at once. The subjective symptoms are likewise very easily explained by this hypothesis, since, when one kidney suddenly ceases to act the other does not at once supply its function, so that for a short time slight symptoms of uræmia are produced.

The favourable issue lastly tells in favour of my hypothesis; for in animals, even after complete ligature of the renal vein, we see the kidney recover its function completely by means of the establishment of a collateral circulation, to which the vasa aberrantia which are often present, and the veins of the capsule of the kidney and of the supra-renal bodies contribute.

It is difficult in an individual case to decide from the gravity of the symptoms, whether we have to deal with

complete obstruction of the vein with subsequent thrombosis, or kinking, or only considerable narrowing by twisting. If the patient immediately assumes a quiet horizontal posture, we have at once the most favourable conditions for the organisation of a thrombus, or the untwisting of a twist, or the straightening of a kink in the vein.

Finally, my hypothesis explains certain processes, the causes of which have been hitherto obscure. Among these I count the atrophy of the moveable kidney comparatively often found post-mortem, which has been observed by *Litten* and *Buchwald* after ligature of the renal vein in animals, especially when a delay or imperfection in the reestablishment of the collateral circulation has suspended the function of the whole or of certain parts of the kidney. Similarly it is probable that the colloid degeneration of the epithelium, observed by these authors, and due to the same causes, stands in intimate relation to the discovery of colloid material in the cyst of a hydronephrosis, in which, also through pressure, the function of the kidney has been considerably impaired.

But it is certain that the *perinephritic and paranephritic abscesses* of moveable kidneys mentioned by *Riolan* himself, and finally the so-called putrefaction or formation of abscesses in the kidney itself, are best explained by thrombosis of the renal vein. A thrombus in a larger or smaller branch of the renal vein remains of course harmless provided it is not infectious and the kidney is healthy. But if these conditions necessary for the harmlessness of a thrombus are absent, especially if there is pyelitis, or if the collateral circulation leading to recovery does not become properly established, *abscesses* in or near the kidney, or even *peritonitis* and *pyæmia* easily supervene.

We cannot be surprised that obstruction of the renal vein, producing œdema of the kidney, &c., will cause dragging on the peritoneal investment of the capsule, and secondarily produce pain like that of peritonitis, as in twisted ovarian pedicle. In the next place slight adhesions between the kidney and neighbouring organs may form ; but the essential part of the process is produced neither by incarceration of the kidney nor by circumscribed peritonitis. Moreover, these adhesions are often produced by the same chronic process as affects

tumours of the ovaries and uterus, and without symptoms of inflammation; the process consisting in friction of the peritoneal surfaces, abrasion of the epithelium, and subsequent adhesion of the abraded surfaces, so that the presence of the adhesions does not always indicate previous inflammatory changes.

It would perhaps seem strange, on the above hypothesis explaining the origin of the symptoms of incarceration, that these symptoms are not more frequently observed. This would certainly happen often, if there were not other causes to prevent the danger of frequently occurring complete or long persistent occlusion or narrowing of the renal vein. These causes receive additional support from the fortunate circumstance, that the renal artery and vein are not equally long, owing to the difference between the course of the aorta and vena cava inferior, so that spiral twisting of both vessels in their whole extent (like that for instance in the umbilical cord) is here rendered impossible.

We must further remember that the part of the right renal artery between the aorta and the right border of the vena cava inferior, and the part of the left renal vein between the vena cava and the left border of the aorta (see Fig. 2) are very firmly fixed by connective tissue to the main trunks, *i.e.* to the vertebral column, and are therefore protected against kinks in part of their course, though a comparatively small part. But it is just here that a series of small veins from the capsule of the kidney, and on the left side from the suprarenal body open, and these effect a collateral circulation easily and quickly, if the circulation in the renal veins becomes obstructed. This end is favoured by the double renal vein found especially on the right side, the frequency of which was pointed out even by *Riolan* and *Haller* (119). It must also be remembered that if the renal vein is kinked or twisted, the renal artery, although provided with very thick walls and a large calibre, will be narrowed at the same time, so that the diminished supply of blood will slightly alleviate the excessive engorgement of the renal vein.

Finally, however, quiet horizontal decubitus, and a return of the kidney to its normal position will very often relieve

the disturbance of the renal circulation under discussion, before the supervention of thrombosis or long persistent kinking of the renal vein.

All these causes explain the reason why, in spite of the probability that kinks and twists often occur in the vessels of a moveable kidney, it is rare for disturbances of the circulation to persist long and produce the symptoms of incarceration so-called, and it is also rare for narrowing or occlusion of the renal vein to arise. Decisive post-mortem proofs are hardly to be expected, since even the ligature of a renal vein never of itself causes death, and on the other hand if fatal complications occur, the vascular changes will be obliterated.

Symptoms arising from the Secretion and Excretion of the Urine.

It is denied by almost all writers that moveable kidney produces any effect on the secretion and excretion of the urine. *Rosenstein* (120) especially insists on the absence of all symptoms of diuresis. *Henoch* (121) lays stress on the healthy nature of the urinary secretion established by *Oppolzer* himself, only one of whose patients complained of frequent desire to pass water when in the erect position, which *Henoch* thought might have depended on other causes. *Trousseau* (122) observes : " Quant à la sécrétion urinaire, elle n'est en aucune façon influencée, il en est ainsi de la miction." On the other hand I have frequently observed disturbances in the secretion and excretion of the urine in patients with moveable kidney, and believe that they play an essential part in the pathology of this disease. These disturbances are shown not so much in colicky pains radiating from the renal region along to the bladder, as in alterations in the quality and quantity of the urine, which is sometimes scanty, high coloured and depositing a copious sediment, at other times becoming more copious and profuse than normal. Hæmaturia is also seen in cases of moveable kidney, a phenomenon which in the absence of every other cause must be considered to depend on the moveable kidney. A particularly striking case of this kind has been recorded by

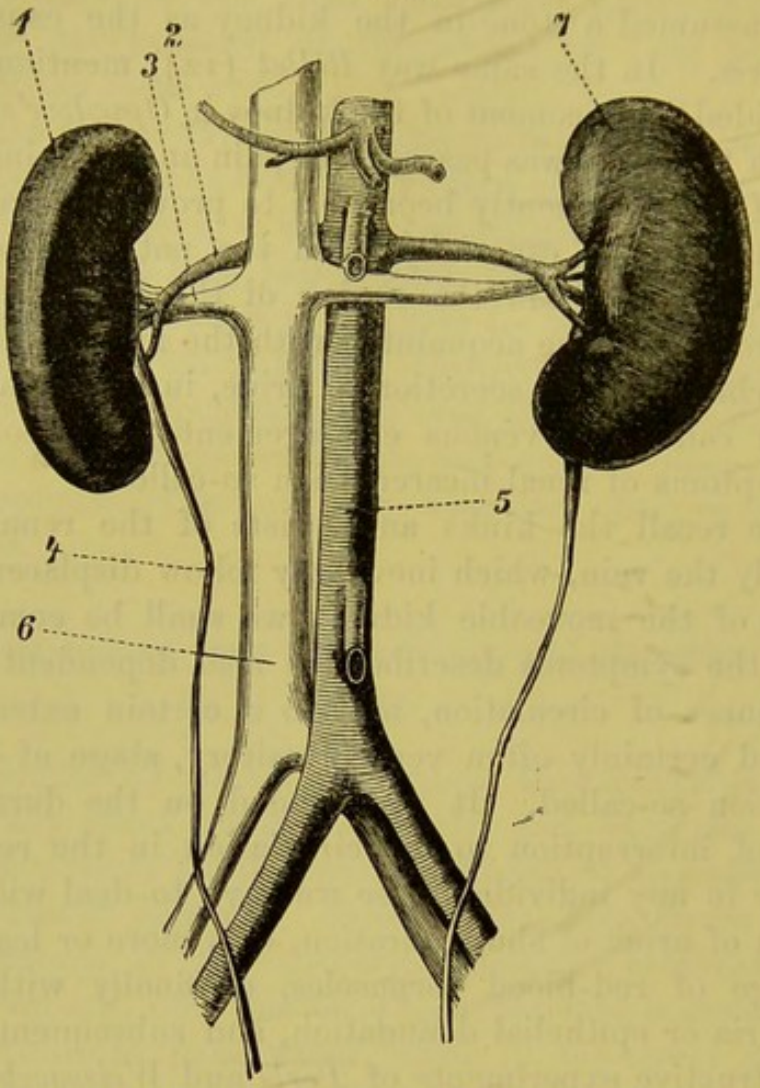
Ehrle (123) in *Niemeyer's* clinique, who, without any cogent reason, assumed a stone in the kidney as the cause of the hæmaturia. In the same way *Rollet* (124) mentions a case of left-sided displacement of the kidney in *Oppolzer's* clinique, in which the urine was passed with pain and contained blood. I myself have frequently been able to prove the presence of blood in the urine, especially when the patients complained of considerable pain in the region of the kidney. But we have already become acquainted with the most intense form of disturbance in the secretion of urine, in the form of suppression caused by venous engorgement, when considering the symptoms of renal incarceration so-called.

If we recall the kinks and twists of the renal vessels, especially the vein, which inevitably follow displacement and rotation of the moveable kidney, we shall be compelled to regard the symptoms described as also dependent on local disturbances of circulation, and to a certain extent as the first, and certainly often very transitory, stage of renal incarceration so-called. It will depend on the duration and degree of interruption to the circulation in the renal vein, whether in any individual case we have to deal with a suppression of urine of short duration, or a more or less copious discharge of red-blood corpuscles, or finally with regular hæmaturia or epithelial denudation, and subsequent pyelitis. The instructive experiments of *Perls* and *Weissgerber* and of *Robinson*[1] already mentioned, who found exactly the same changes as those above-named in the secretion and excretion of urine in the animals under experiment, point in the same direction, namely, that here an obstruction to the circulation in the renal vein takes place. In the same way frequent impediment as well as total obstruction to the circulation in the renal vein is capable of producing atrophy of the kidney. Whether such local disturbances in the circulation may also lead to parenchymatous nephritis, as *Frerichs* (125) and *Leudet* (126) imagine, must remain an open question.

By far the most important disturbances however take place with reference to the *excretion* of the urine. We have already seen that this is disordered even by local disturbance of circulation in the vessels, and this is confirmed by

[1] ["Robertson" in the text. But see p. 297.—TRANSLATOR.]

Fig. 6.

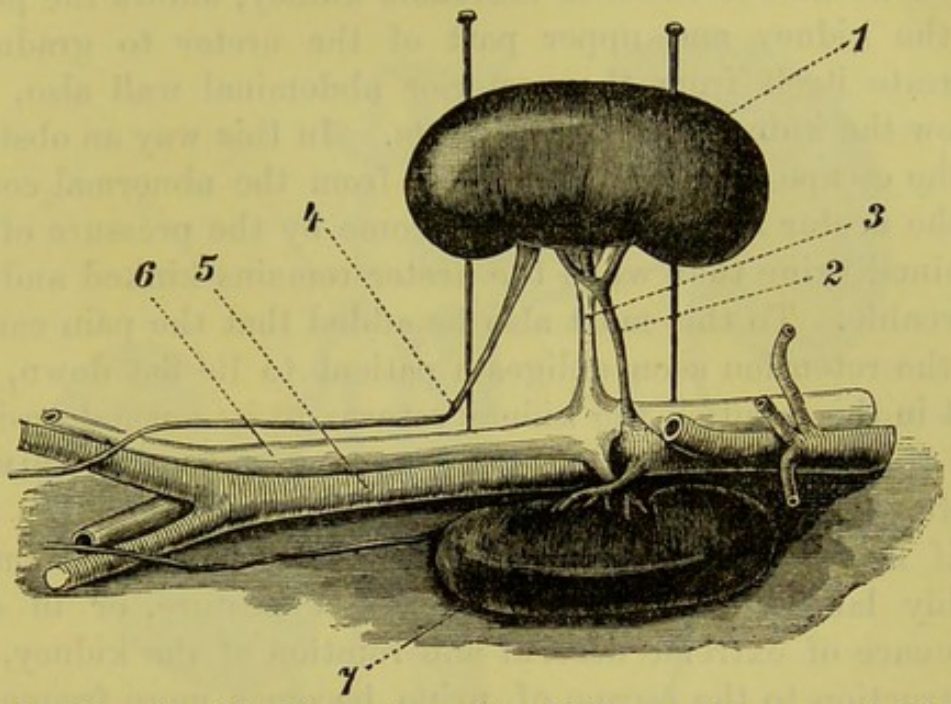


1. Right kidney, twisted on its horizontal axis, and with its outer border looking downwards.
2. Right renal artery.
3. Right renal vein.
4. Right ureter, kinked and twisted.
5. Abdominal aorta.
6. Vena cava inferior.
7. Left kidney in its normal situation.

numerous experiments involving the ligature and artificial narrowing of the renal vein. In this relation however the disturbance of the circulation, even when it occurs more or less contemporaneously with the disturbance of the circulation, is *secondary*, and caused by the alteration in the secretion. But I must affirm, contrary to the general opinion, that the *secretion of the urine* especially, may be *primarily* suppressed by narrowing or occlusion of the ureter.

As soon as the kidney descends, the origin of the ureter, which is normally placed at the lowest point of the renal pelvis, and therefore at the point most favourable for the

FIG. 7.



The last dissection (Fig. 6) seen in profile. References the same as to Fig. 6.

escape of the urine, moves to a higher position, and, if the kidney is very low down, to the very highest point of the pelvis. But as the kidney rotates (as it usually does) the ureter will also become twisted on its axis. In the former case kinking of the ureter results, in the latter torsion, in either case obstruction to the flow of urine must result.

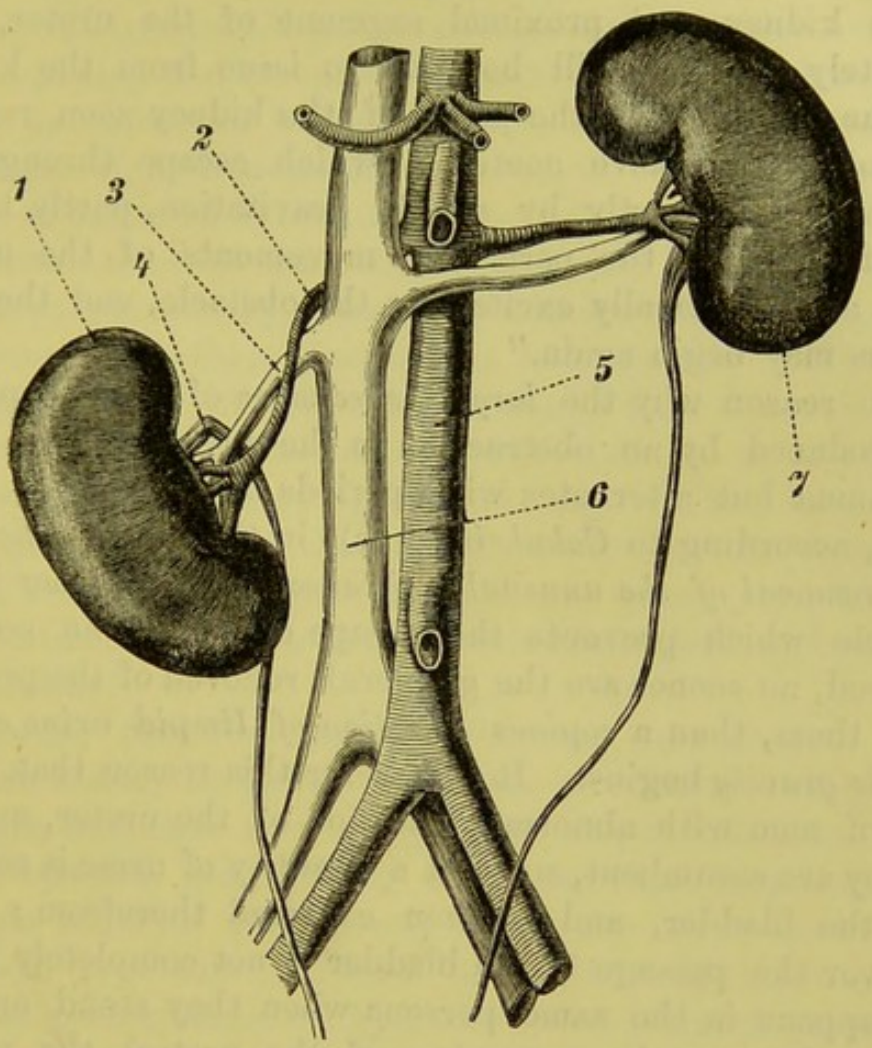
Here, as in the case of the renal vessels, I have endeavoured to render the torsion, kinking and bending of the ureter intelligible by the help of drawings. Figs. 6 and 7, in which the ureter is bent and twisted, represent the same dissection from two different views; Fig. 6 from the front, Fig. 7 in profile. The very perceptible upward bend of the ureter, which we have already seen in Fig. 5, is repeated in Fig. 8 below. If this is compared with *Mosler's* post-mortem account, (see above p. 258) the resemblance between the course of the ureter as there described and as here drawn, cannot fail to be seen.

It is in fact probable that the colicky pains often observed in patients with moveable kidney depend on temporary obstructions and occlusions of the ureter, and the simultaneous local disturbances in the circulation. The suppression of urine is however usually soon relieved, inasmuch as the loosening of the capsule of the kidney from the posterior abdominal wall,

which we find in cases of moveable kidney, allows the pelvis of the kidney and upper part of the ureter to gradually separate itself from the posterior abdominal wall also, and follow the kidney in its movements. In this way an obstacle to the escape of the urine arising from the abnormal course of the ureter is more easily overcome by the pressure of the retained urine than when the ureter remains kinked and not moveable. To this must also be added that the pain caused by the retention soon obliges a patient to lie flat down, and that in this position the kidney returns to its normal position and the kinked or twisted ureter at once rights and untwists itself.

If however, under the influence of severe or prolonged bodily labour, especially in the erect posture, or in consequence of extreme descent and rotation of the kidney, the obstruction to the escape of urine becomes more frequently repeated, and increases in intensity, the continual change between tense repletion and complete evacuation of the pelvis of the kidney must result by degrees in its diminished elasticity and increased distension, and finally in the condition described by *Cohnheim* as the consequence of so-called kinking and improper insertion (and, I would add, torsion) of the ureter—that is, in hydronephrosis. According to his clear description, when, for instance, the ureter is inserted into the kidney opposite the upper part of the pelvis, in the erect posture the urine cannot overflow into (? out of) the pelvis of the kidney until the pelvis of the kidney is full to that point. "The pelvis of the kidney is enclosed by a yielding and expansible wall, which gradually dilates under the pressure of the urine collecting within it, until the sac reaches such a size as to be capable, on its part, when full, of completely compressing the ureter, which lies immediately in contact with it. But only in the erect position! For as soon as the owner of this kidney lies down, as for instance during sleep, the urine flows away continuously, and without any obstacle, out of the sac into the bladder. In the case of kinks, on the other hand, the change is produced by the segment of the ureter situated above the kink, rising somewhat as the distension becomes greater, in such a way that the point of bending comes rather to resemble the point

Fig. 8.



1. Right kidney.
2. Right renal artery kinked.
3. Right renal vein.
4. Right ureter bent, and inserted into the highest point of the pelvis of the kidney.
5. Abdominal aorta.
6. Vena cava inferior.
7. Left kidney in its normal situation.

where a funnel passes into its neck. But as soon as the greater part of the urine has escaped, the ureter becomes kinked again at the old spot. But even in the case of simple narrowing, however little the obstacle itself changes, there is by no means always the same impediment to the secretion of urine. Suppose a case in which a considerable obstacle arises suddenly during a period of plentiful secretion, a moment will be reached (not, indeed, so rapidly as in complete obstruction, but in a relatively short time) in which the disorder of relation between secretion and escape will

result in so great a repletion and distension of the pelvis of the kidney and proximal segment of the ureter, that absolutely no urine will be able to issue from the kidney into the pelvis. But the pelvis of the kidney soon relieves itself of its excessive contents, which escape through the narrowed spot, partly by simple gravitation, partly by the powerful aid of the peristaltic movements of the ureter, which are abnormally excited by the obstacle, and then the process may begin again."

The reason why the *largest specimens* of hydronephrosis are produced by an obstruction in the ureter, which is not permanent but alternates with periods of patency, is to be found, according to *Cohnheim* (127), in the *state of the urine after removal of the unusual resistance.* "No sooner is the obstacle which prevents the escape of the urine secreted removed, no sooner are the glomeruli relieved of the pressure upon them, than a *copious secretion of limpid urine of low specific gravity* begins. It is just for this reason that, in the case of men with abnormal insertion of the ureter, as long as they are recumbent, so great a quantity of urine is secreted into the bladder, and is then excreted therefrom; when however the passage to the bladder is not completely patent (as happens in the same persons when they stand or walk, and as happens in narrowing of the ureter) the profuse secretion fills and over-distends the pelvis of the kidney and the ureter with greater and greater quantities of urine. And because the secretion of urine never ceases as long as any escape from the distended renal pelvis is possible, years and tens of years may pass in the same process, though of course not without a very considerable expansion of the parts affected."[1]

[1] The same kind of "hysterical" urine is secreted under a variety of circumstances, including any retention in the bladder sufficient to raise the pressure in the ureters. A familiar instance is furnished by the frequently-observed phenomena of Retroversion of the Gravid Womb, the prominent symptom of which is retention of urine. The urinary symptoms do not cease with the evacuation of the bladder, nor with the simultaneous reduction of the incarcerated uterus, but a "copious secretion of limpid urine of low specific gravity" continues for some time, and often for some days, the quantity being enormous. The same set of facts is discussed in a paper alluded to in a previous note (Champneys, 'St. Bartholomew's Hospital

Although however, no further demonstration is needed to prove the impossibility of so marked an alternation between tense repletion and complete evacuation of the pelvis of the kidney as *Cohnheim* has assumed in the above exposition; the origin of hydronephrosis by means of mobility of the kidney as assumed by me contradicts this assertion.

So little attention has hitherto been paid to the production of hydronephrosis by moveable kidney, that *Lancereaux*, *Trousseau* and others describe it only by the way as an occasional cause of moveable kidney, and *Gilewski* only mentions acute hydronephrosis, and that in an erroneous connection with the symptoms of incarceration so-called. The reason of this is that the formation of the hydronephrosis entirely obliterates the primary causes, not only in their clinical relation, but also in their anatomical appearances on dissection.

In order therefore to establish my hypothesis, (that moveable kidney furnishes one of the chief causes of hydronephrosis, and that very many cases whose causes are considered obscure are nothing else than hydronephroses in moveable kidneys) not merely upon the above somewhat theoretical arguments, I shall here give additional proofs from the pathogenesis of hydronephrosis founded on pathological conditions, experiment, and clinical symptoms.

(1) *Proofs derived from the Pathogenesis of Hydronephrosis.*

That form of hydronephrosis, in which no cause of compression or obstruction of the ureter is found after death, was known even to ancient writers.

Schönlein (128) disagrees with the assertion of *P. Frank*

Reports,' vol. xvi), treating of the dilatation of the ureters in Extroversion of the Bladder, the author's theory being that of obstruction to the flow of urine by spasm of the vesical orifices of the ureters caused by irritation of the extroverted bladder. Similar urine is often secreted by children suffering from incontinence, and is probably due to the same cause. In all these cases dilatation of the ureters and renal pelves is produced by the same cause, viz. a considerable but not absolute obstacle to the escape of urine.—TRANSLATOR.

(that the dropsical kidney arises from mechanical causes, one of the ureters being compressed and preventing the escape of the urine, which is retained in the kidney and gradually distends it), and expressly remarks that cases are met with, and indeed are the majority, in which the ureters are quite patent. "But the conditions of the malady are still unknown."

Cruveilhier propounded the following explanation for these cases, which has also been accepted by *Englisch* (129): "If an organic sac or tube has once been expanded and its tissues overdistended so as to lose some of their elasticity, the distention of the walls[1] remains, and the collection becomes permanent." In the fœtus an obstruction of this kind may be produced by the adhesion of the contiguous epithelial layers.

According to *Rosenstein* the cause of hydronephrosis in the absence of any discoverable impediment, is in many cases *obscure*.

Virchow (130) observes that those cases are the strangest of all in which extreme hydronephrosis is present, in spite of patency of the ureter. *Virchow* has frequently examined such cases, and has each time found a valvular obstruction caused by a folding of the wall due to an oblique origin of the ureter out of the pelvis of the kidney.

Simon (131) has also observed this sort of hydronephrosis, which according to him most frequently gives occasion to operative proceedings, and to which, although practically the most important, but little attention has hitherto been paid. In two cases examined by him the completely pervious ureter arose not only at an acute angle from the pelvis of the kidney, but its upper part even lay in the wall of the pelvis for a length of seven to ten centimetres, yet not in the proper wall of the renal pelvis, but between this and its peritoneal investment. The valvular closure at the renal orifice of the ureter (ostium pelvicum) was so complete, in spite of the perviousness of the ureter, that, even when the sac was filled with water at a considerable pressure, not a drop escaped from the ureter.

Cohnheim (132) alludes to the abnormal insertion of the

[1] Wunde, a misprint for Wände.—TRANSLATOR.

ureter as an important cause of hydronephrosis, when he says he *considers it to be a congenital malformation.* The ureter, instead of rising, as usual, from the lowest point of the pelvis, rises higher up, so that in the erect position the urine cannot escape until the pelvis is full up to the level mentioned. The idea that these were cases in which the ureters had originally been double, and that one of them had subsequently become degenerated and disappeared, is excluded by the fact that where there are two ureters there are also two pelves, and that these are very rarely amalgamated. "*The reason for the occasional abnormal condition of the transitional spot between the pelvis and ureter is in fact unknown,* and moreover in a dissection nothing can usually be ascertained with accuracy, as the distension of the pelvis, which is never absent under these conditions, and is often quite enormous, has already completely changed the original conditions. Moreover, we sometimes meet with quite sharp kinks in the course of one of the ureters, which must properly be considered congenital, in the complete absence of bands of connective tissue caused by adhesions and cicatrices."

Although however all writers regard the angular insertion and valvular closure of the ureter as the cause of the hydronephrosis, which view appears to me to be confirmed by the examination of the new-born infant, *Simon* considers hydronephrosis to be the cause of the morbid changes in the course of the ureter. Owing to some impediment or other, as even *Cruveilhier* assumed for the case of the fœtus, a hydronephrosis forms, and the ureter, *which was not enlarged above the impediment,* becomes displaced at an acute angle at its pelvic orifice (ostium pelvicum) as the tumour grows larger. This causes a valvular closure, by which the lower half of the distended renal pelvis compresses the upper part of the ureter. Here, therefore, the same mechanism is in operation as that by which, in the case of diverticula of the alimentary canal, the part immediately below the diverticulum is kinked and occluded with a valvular closure. As the distension of the pelvis of the kidney increases (which can only proceed horizontally forwards and outwards, the vertebral column offering an obstacle on the inner side), the substance of the kidney becomes pressed outwards and backwards together

with its calices. Through these changes in position, the upper portion of the ureter, which normally lies on the inner side, comes to point more forwards and outwards, and comes to be placed between the anterior internal wall of the distended renal sac and the peritoneum which invests it.

Englisch similarly gives his opinion against the assumption of a primary abnormal course of the ureter as a cause of hydronephrosis.

When, however, *Simon*, starting from this explanation, goes on to assert that the ureter, occluded by a stone, or compressed by a parametric cicatrix, or hindered in its evacuation of urine by the distension of the bladder, collapses above the impediment, this hypothesis is actually impossible, for the result of urinary obstruction is to produce *distension* above the point of compression. Thus, the conclusions drawn from this explanation appear unjustifiable. The origin of hydronephroses of this kind is therefore inexplicable, either from the artificial explanations of *Cruveilhier* and *Simon*, or from the general hypothesis of the congenital character of the morbid insertion and valvular occlusion of the ureter.

Moveable kidney, however, appears from the above considerations calculated to produce urinary obstruction and consequently hydronephrosis, by frequently repeated displacement and twisting and kinking of the ureter. This view is confirmed by the fact that the majority of hydronephroses of obscure origin are seen in women of considerable age, and on the right side.

(2) *Proofs derived from Anatomical conditions.*

When dealing with the post-mortem appearance in moveable kidney (see above), we mentioned some cases in which an abnormal position of the hilum (which sometimes pointed upwards and sometimes forwards), and of the ureter (which sometimes ran upwards in a curve above the pelvis of the kidney) were actually shown. Although hydronephrosis was not simultaneously present in all these cases, we cannot conclude from this circumstance, as *Mosler* has done, that it cannot be caused by these abnormalities, but only that it

does not always necessarily result therefrom. In addition to those given above I have found the following pathologico-anatomical reports which confirm my hypothesis.

Sandifort (133) reports a case of hydronephrosis in which the ureter, which was pervious, was twisted on its axis: "Ren dexter sanus; sinister morbosus. Hic quippe, expansus insigniter, ultra quinque pollices longus, tres cum dimidio latus, et pressioni cedens, singularem et pelvis et totius corporis monstrabat expansionem, vere hydropicam . . . Ureter . . superiori in loco quidem parum prominulus, sed dein contractus et quasi contortus erat, ad vesicam sanam descendens, et toto hoc in tractu minime expansus, verum potius contractus, non tamen tantopere, quam superiori in loco, ubi haec constrictio tanta erat, ut, compresso maxime rene, non nisi paucæ liquoris flavi urinosi prodirent guttulæ. Fisso uretere contractus maxime ureter et ferme occlusus apparuit, ubi ex pelvi originem trahit." The ureter was therefore pervious.[1]

Haller (134) reports: "In a woman who had died of dropsy, a tumour had grown some years since below the navel, and was regarded by the surgeon as a rupture. After death it was found that this lay beneath the peritoneum and omentum, it was membranous, white, and filled with water, an ureter arose from it, and traces of renal papillæ were found in it. A *remarkable instance of alteration in the position and structure of the kidney.* The other kidney was quite healthy and lay on the right side."

The two following cases are still more striking:

Hare (135) found double hydronephrosis in the body of a woman, 38 years of age, in whom a large tumour in the left as well as in the right side had occasionally appeared and disappeared. That on the right side was still full, the left nearly empty. The only impediment to be found was that *both ureters were twisted on their own axis.* As soon as the ureter was separated from the surrounding tissues and untwisted, the urine collected on the right side flowed freely away. Hare regarded the cause of the twisting as congenital.

P. Wilse (136) saw in a woman aged 39 (who had had

[1] Incorrectly quoted by the author.—TRANSLATOR.

eleven children and had suffered at times from violent gastric disturbances), a tumour in the left side of the abdomen which *sometimes disappeared*. After death, hydronephrosis of the left kidney was found. The left ureter was neither obliterated nor occluded by a stone; on the other hand it was curved upwards at the point where it sprang from the pelvis, and thus formed a valve which closed the orifice of the ureter as soon as a certain collection of urine took place, but if the accumulation became greater, was unable to maintain the occlusion. A temporary hydronephrosis was thus produced, which explained the attacks.

The analogy between this and the condition of the ureter in cases of moveable kidney strikes one at once (see Figs. 5 and 8), but in neither case was it thought of. Besides these, we find in pictures of hydronephroses the ureter twisted on its own axis.

(3) *Proofs derived from clinical observations. Intermittent Hydronephrosis.*

Cases of Hydronephrosis associated with and caused by moveable kidney certainly occur oftener than has been hitherto imagined. Even *Simon* (137) in 1875 was acquainted with not a single example of hydronephrotic moveable kidneys, although his own explanation of the origin of hydronephrosis from temporary disturbances frequently repeated ought certainly to have led him to regard moveable kidney as the cause in doubtful cases. Since then hydronephroses in wandering kidneys have been observed with certainty (namely by operation), by *Pernice* (138), *Ahlfeld* (139), *Czerny* (140) (two cases), *Wagner* (141), and *Landau* (142). All these observations concerned women and the right kidney.

The connection between moveable kidney and hydronephrosis is recognised most unmistakeably by a symptom which must be regarded as quite pathognomonic in the production of hydronephrosis by moveable kidney, namely, the periodical filling and emptying of the sac of the hydronephrosis. This phenomenon, which attracts our notice

by the frequent appearance and disappearance of a tumour, and which can be called (as *Cole* (143) and *Morris* (144) have done), intermitting, or temporary, or relapsing hydronephrosis, has been described in moveable kidney by no one but *Eger* (145), so far as I can see. I myself have also seen a case in point (146). The refilling and disappearance of the hydronephrotic sac could be repeatedly proved. In the woman especially on whom I operated, the torsion or valvular closure of the ureter was relieved by changes in posture and by diminution of the pressure of the hydronephrotic fluid following moderate aspiration, the escape of the urine becoming re-established. Both observations are appended below.

The reason why intermittent hydronephrosis is so seldom observed may be that, as long as the alternation between repletion and evacuation goes on, the hydronephrotic sac has still a small volume and therefore easily escapes recognition. But as soon as it becomes larger and more easily palpable, the valvular closure or the abnormal insertion of the ureter is generally so firmly established that the hydronephrosis remains permanent. Thus it happens that intermittent hydronephrosis in moveable kidney is often overlooked or otherwise interpreted.

Tulpius (147) is the first to report on this peculiar form of "ischuria," which he observed in a patient each time at full moon. It lasted in his case five days on each occasion, and only yielded to a venesection. He was unable to explain this peculiar condition during the life of the patient:[1] "Sola anatome, post obitum instituta, eruit illic feliciter veritatem in profundum demersam; et ostendit distincte, quî angusta, renis sinistri, pelvis, excrevisset in eam amplitudinem, ut suppleret commode vicem, vesicae urinariae.[1] Quae propterea tam fuit vacua, quam ren repletus."

Sauvages (148) described this form of retention of urine, connecting it also with the moon, *i.e.* with the "Ischuria lunatica Tulpii."

Johnson (149) observed in a woman pregnant for the eighth time a remarkably changeable condition of the urine,

[1] The case is reported under the heading "Ischuria lunatica" and the original contains "uniariae"—a misprint for "urinariae."—TRANSLATOR.

which was either scanty and dark or plentiful and milk-white; in the first case its excretion was accompanied by great pain, in the last case it was easily passed. After delivery a large tumour was noticed on the left side, projecting four inches above the surface of the abdomen, and measuring seven or eight inches in diameter. The tumour was tense and smooth over its whole surface; on gentle palpation there was distinct fluctuation; pressure on it caused pain. The diagnosis of an ovarian tumour was made. Soon after the patient died; the autopsy showed bilateral hydronephrosis.

Schönlein (150) distinguishes two varieties of renal dropsy (Hydrops renalis). In the first, in which the ureters are pervious, the patients pass an enormous quantity of pale greenish urine, four to six quarts[1] or more in the twenty-four hours. In the second variety, in which the ureter is closed, the urine passed is scanty, dark, and reddish; the tumour in the renal region and the sympathetic symptoms are much more plainly marked in the second than in the first. "The diagnosis is very difficult. The disease has not hitherto been recognised till after death, in several cases it has been taken for ovarian dropsy."

Rosenstein (151) quotes a case of supposed hydronephrosis which, in consequence of the sudden disappearance of the tumour, he erroneously considers to have been an ovarian tumour, as he remarks: "In cases where the development of the tumour is not observed, there will always be sources of error in diagnosis. The statement of the patient also, of occasional spontaneous evacuation through the bladder, is well worthy of consideration, as this is more likely to happen when an ovarian tumour is adherent to the ureter than in hydronephrosis, where the mechanical occlusion occasioned by the hydronephrosis is hard to overcome."

Hillier (152) relates the case of a woman who showed a tumour in the right side, which occasionally disappeared and then reappeared. As long as the tumour remained she had violent pain and the urine was scanty, as soon as it dis-

[1] A "Maass" is a German measure for beer, different in different States. It seems to be about a pint on an average, but in this place must mean a larger quantity.—TRANSLATOR.

appeared the pain went away and the urine became profuse and turbid.

The similar observations of *Hare* and *Wilse* have been already mentioned above. In the discussion which followed the relation of a case of intermittent hydronephrosis caused by cancer of the bladder, by *Morris* (153), altogether three or four cases of intermittent hydronephrosis were mentioned by *Hare* and *Smyth*, of which I believe, as I also do of those mentioned before, that they were caused by the same mechanism as the hydronephroses observed by *Eger* and me.

(4) *Confirmation by experiment.*

If the moveable kidney, pelvis, and ureter are represented by an india-rubber elastic ball loosely fastened on a firm board, with a thin afferent and efferent tube (154), intermittent hydronephrosis is easily reproduced under exactly the same conditions as in the living subject.

(1) If the bag filled with water is lifted up vertically and bent so that the efferent tube occupies the highest point, evacuation will not take place until the pressure of the water overcomes the elasticity of the india-rubber bag. Analogue: Moveable kidney with the ureter inserted into the highest point of the pelvis.

(2) If the bag is laid horizontally, evacuation will spontaneously take place if the efferent tube is intact.

(3) If the bag is filled in a horizontal position and placed so that the efferent tube forms an angle with the bag, and the tube is then twisted; as the pressure of water increases in this position, the bag will become more and more elevated and distended, and no evacuation will take place until the board on which it lies is considerably tilted to the side, which will cause the bag to turn on its side and thus untwist the efferent duct.

(4) If on the contrary the board is made vertical, the bag if moderately filled in this position will bend towards the efferent duct and form a kink in it. It will depend on the size of the angle and the downward resistance occasioned

by the further influx of water into the expanding bag[1] whether the bag can rise so as to diminish the angle between it and the efferent tube. Analogue: Intermittent hydronephrosis caused by the descent of the kidney and axial rotation of the ureter.

(5) The effect of increased and diminished pressure can also be easily imitated with this apparatus. As in the patient, when the bag is filled, evacuation will at one time be rendered possible by increase, at another by diminution of pressure, according to the position of the bag.

(6) The causation of hydronephrosis by prolapsus uteri is illustrated by hanging a weight on the efferent duct. As the weight is increased the walls of the efferent duct to a greater or less height above the weight are seen to become firmly pressed together and to close the duct.

Phenomena of Moveable Kidney during Pregnancy and Labour.

According to the scanty observations hitherto available, the troubles caused by moveable kidney appear to be rather diminished than increased.

(*a*) The growing womb pushes the intestines gradually up, and with them indirectly the kidney, as I have observed in two cases; and by this means the evil effects of dragging and kinking of the vessels and ureter are usually alleviated, just as is the case when pregnancy supervenes in a case of descent of the generative organs.

(*b*) To this is to be added the circumstance, that during pregnancy there is usually a copious deposit of fat in the panniculus adiposus and in the subperitoneal cellular tissue, which gives additional firmness to the attachments of the kidney.

(*c*) Moreover the circumstance that menstruation (which exercises an untoward influence on the capsule of the kidney both anatomically and also symptomatically in its effect on

[1] *i.e.* On the action of gravity as against the tendency of the elastic bag to erect itself as it becomes tense?—Translator.

the general health) ceases during pregnancy, contributes essentially to the relief of the disturbances caused by moveable kidney. Some observers, as *Hare* and *Oppolzer*, have indeed observed cures of moveable kidney during pregnancy, which cannot be doubted when the same experiences are remembered in the case of prolapse of the vagina and uterus. Whether moveable kidney exercises an injurious influence on the other hand on the pregnant uterus, is as yet unknown. It is however not unlikely that where it has descended very low, or in case of complications such as interruption of the circulation in the renal vein (in the former case mechanically, in the latter by the great disorder of the circulation), it is capable of inducing abortion, as indeed happened in the case related by *Eger*. That this is not inevitable in cases of intermittent hydronephrosis is proved by the observations of *Johnson* and *Eger*, in whose cases also the growing uterus appeared to undo the kinking and twisting of the ureter, and to make it pervious for the urine.

Dearth of observations prevents us from stating *a priori* the exact mutual relations between labour and acquired moveable kidney. The cases of *Chambon de Montaux* (155) (in which a kidney was found incarcerated behind the growing uterus), *Boinet* (156) (who observed a deviation of the uterus, caused by a kidney lying behind the bladder), and *Hohl* (157) (in which a displaced kidney is said to have formed an obstruction to labour), one and all concern cases of congenital malformation and probably of displacement with subsequent fixation. Moreover the case of *Hohl* is so inaccurately and unscientifically described that it cannot be utilised to prove anything.

It is however obvious, as well as established by many cases, that the pressure which occurs in labour is capable of acting injuriously on the kidney. Women therefore who have a moveable kidney must be cautioned against bearing down too strongly during labour.

VIII. Objective Signs of Moveable Kidney.

Moveable kidney is capable of attracting attention both by its absence from its normal position and by its appearance in an abnormal situation. It will therefore be necessary to examine the question what physical signs, both in the anterior and posterior lumbar region are due to it.

Inspection.—In thin subjects with a moderate amount of pendulous belly, the moveable kidney may occasionally be found against the anterior abdominal wall in the erect posture. Its outline can in some cases be also made out when the patient lies flat on the back or on the side opposite to that of the moveable kidney. Sometimes the kidney can be seen to map itself out by a sulcus parallel to itself which moves downwards on deep inspiration along the anterior abdominal wall.

The condition of flattening or depression of the corresponding lumbar region, especially plain in the knee-elbow position, described by the majority of writers (158) as constant, is an untrustworthy and exceptional sign. Its small value is best seen by the fact that in a patient whose right kidney had been removed, and whose great emaciation also furnished the most favourable conditions for its production, I was unable to see this flattening. *Keppler* also failed to find this depression. The fact that in certain positions, especially when the legs are adducted, the so-called lateral lumbar sulcus corresponding to the lateral border of the sacrospinalis muscle normally comes out pretty plainly in thin subjects, has in my opinion given rise to many mistakes of this kind.

Percussion.—The resonance over a moveable kidney in the abdomen is generally dully tympanitic. It is only when the kidney has a long mesentery, and relaxation of the subperitoneal cellular tissue allows the ureter to detach itself far from the posterior wall of the abdomen, that the resonance is absolutely dull (and then of course only on superficial percussion), because in this case no intestines separate the kidney from the abdominal wall. On the left side the resonance is also but seldom quite dull, because the voluminous stomach and intestine filled with air always lie in

front of the kidney, unless this is very low down; and although purgatives may evacuate the air which masks the true percussion sound from the intestine, they cannot do so from the stomach. A special value has been laid on the percussion signs in kidney diseases, caused by the relations of the kidney to the colon. Whereas in the case of tumours of the kidney the colon almost invariably lies on their inner side, and the percussion sound on their outer side is dull (as would naturally be supposed in the case of extraperitoneal tumours), moveable kidney has had the percussion signs of an intraperitoneal tumour assigned to it (*Simon*, *Ahlfeld*), and it has been believed always to lie on the inner side of the colon, and always to be bounded on the outer side by tympanitic resonance. This relation however is only found where the kidney has a long mesentery, and where in addition the flexure of the colon is very long or the kidney has migrated between the folds of the mesocolon. But when the mesocolon is taut and short, even if the kidney is moveable the colon is usually pressed inwards, and the percussion note on the outer side of the kidney remains dull, as in the case of a subperitoneal tumour. A moveable kidney therefore behaves with regard to percussion, at one time as an intraperitoneal and at another as an extraperitoneal tumour.

Here also it is generally believed that the absence of the kidney from its usual position in the lumbar and posterior abdominal region furnishes important percussion signs. According to *Rollet*, *Trousseau* and others, the note over the side corresponding to the displaced kidney is clearer and fuller than on the other side.

The majority of the textbooks and handbooks on percussion say the same. *Piorry* (159), the founder of renal percussion, claims to have percussed out even the slightest changes of size in the kidney.

According to *Guttmann* (160) the diagnosis of a moveable kidney (whenever at least a proof is needed), rests on the circumstance that percussion shows the absence of the kidney from the normal place, by the diminished dulness. "If a kidney is displaced the lumbar region of that side is more resonant than the other side."

Numerous investigations however have convinced me of

the untrustworthiness of this sign. Even strong percussion hardly detects any difference between the two sides in moveable kidney. When one considers that one percusses a thick layer composed of the muscles of the back, the ribs, and the quadratus lumborum muscle, it is not to be expected that the dulness will be changed into resonance by the absence of the kidney. If the kidney is displaced on the right side, which is generally the side in point, its place is at once taken by the posterior surface of the liver, which of course in no way changes the original dulness. Thus neither *Weil* (161), who examined a patient whose kidney had been removed by *Czerny*, nor I under similar circumstances found any difference in resonance. Moreover *Keppler* (162) justly denies, on the ground of careful examination, the so-called physiological kidney dulness.

Pansch (163) again has pointed out, from minute topographico-anatomical investigations, the errors of the clinical observers like *Gerhardt*, according to whose account of the kidney dulness the kidney normally reached as far as the crest of the ilium, whereas it does not reach so far by three to five centimetres. *Pansch* moreover correctly observes that even an area of dulness reaching to the crest of the ilium is not necessarily identical with the proper kidney dulness, since the capsula adiposa lying below the kidney is often as thick as the kidney itself which causes the dulness.

After all we shall therefore be obliged to agree with *Skoda's* (164) statement already mentioned: "The size of the kidney has very little to do with the state of the resonance in the lumbar region. This may be quite dull when the kidneys are very small, and tympanitic although the kidneys are very large. We must therefore when possible press the plessimeter down until we get complete dulness and the *resistance of a solid organ.* By repeating this process at several spots we can judge where the kidney shows a striking enlargement."

As a matter of fact even in the case of moveable kidney the most unmistakeable signs are furnished by palpation.

Palpation.—A moveable kidney is generally to be perceived beneath the free edge of the ribs, rather towards the

middle line and the navel, as a smooth, oval, solid sensitive body. Now and then we succeed in feeling the shape of the kidney, but rarely (as happened in *Frerichs'* clinique) in feeling the pulsation of the renal artery. The right kidney is easier to feel than the left, because the left is surrounded by softer and more yielding parts than the right, and can therefore more easily slip away. The extreme mobility of the kidney is remarkable, for a slight touch or the contraction of the abdominal walls is sometimes alone sufficient to make it change its position, and in the horizontal posture it easily slips back into its normal place. The mobility of the kidney is often so great that one has actually to surprise it in order to catch it for palpation. Once back in its normal position it is sometimes hard to render it apparent again. Some patients can press it into the flanks and bring it forward again by peculiar sidelong movements of the trunk. This however does not always succeed, and so, after making a diagnosis one is often subsequently at a loss. This habit that the kidney has of retreating into its normal position and staying there is so characteristic that in two cases in which a moveable kidney was certainly present and a long incision had been made in the linea alba with the view of removing it, it could not be brought forward into the wound in the abdomen without shaking and changing the position of the whole body (see *Keppler* (165) and *Lauenstein* (166)). On a third occasion even the shaking was in vain, and *King* (167) (whose case it was) was obliged to abandon the operation.

Palpation must be practised with the patient in different postures and always *bimanually* (168). The knee-elbow position which was formerly recommended is least suitable for this purpose, because in this position the kidney tends to fall towards the diaphragm, that is, into its normal position ; the most convenient is the horizontal posture with the upper part of the body moderately raised and the legs moderately bent, a position which is usual in gynæcological examinations on account of the diminution in the intra-abdominal pressure which it causes. It appears useful to place the patient on the left side to feel the right kidney, to press the right hand firmly against the lumbar region and to press the kidney

with the right hand against the left hand resting on the abdomen. In the case of the left kidney *vice versâ*.

By these manipulations it is surprisingly easy in favourable cases to feel the lower end even of the normally placed kidney, and when it is moveable to move it to and fro between the hands. At the same time an impression is gained by practice whether one or other lumbar region is more easily indented than usual or than the other. This condition, constantly to be observed in moveable kidney and important for diagnosis, depends upon the fact that when the kidney is absent from its normal position, the soft parts situated in the flank, the muscles and intestines, are more easily pressed under the arch of the ribs; but is not, as has been erroneously supposed, explained by imagining that the kidney which is normally situated beneath the arch of the ribs betrays its absence *directly* by this diminished resistance.

Palpation itself is generally painful. Sometimes violent pressure makes a patient vomit. *Trousseau* (169) made use of the tenderness caused by pressure on the kidney for diagnosis, comparing it with the tenderness produced by pressure over the other kidney.

Auscultation has not hitherto been utilised for diagnosis in the case of moveable kidney. It is not however to be doubted in this case also a whistling murmur will be audible when twists and narrowing of the vessels occur.[1]

IX. Diagnosis.

The recognition of a moveable kidney depends on the physical signs, especially those derived from palpation, and

[1] The auscultation of abdominal tumours is in so unsatisfactory a state that little can be said about it. A murmur is said to be occasionally heard in ovarian tumours, but what these tumours are, or what are the circumstances under which murmurs occur in them is not stated. It would be a picturesque circumstance for a murmur to develop in an ovarian tumour during twisting of the pedicle; but where is such a case recorded? The uterine murmur in some fibrous tumours is well known. The wisest course in the present chaotic state of knowledge is to follow the advice of the American ambassador, and "Never prophesy unless you know."—Translator.

the knowledge of the numerous symptoms caused by it. The fact that they are so very often overlooked or wrongly interpreted depends not only on want of acquaintance with their subjective and objective symptoms, and on the usual supposition that the patients are hysterical or hypochondriacal, but also on real difficulties standing in the way of their recognition.

When indeed one succeeds in seizing a solid body situated in the abdominal cavity in such a way that one can move it more or less backwards and forwards between the lumbar and umbilical regions, in finding the pulsation of the renal artery and the hilum, there is no difficulty in making a diagnosis. But if a kidney possesses only moderate mobility or is situated within a patient with strong and fat abdominal walls, the physical symptoms afford but an uncertain conclusion, and the subjective symptoms are generally of such a nature as to be capable of other equally justifiable interpretations. Finally, if one even succeeds in feeling a solid round body in the abdomen without recognising the shape of the kidney, there are a large number of other pathological conditions (tumours and so forth) which can easily be mistaken for it on account of the similarity of their subjective and objective symptoms. On examining the statistics of moveable kidney, we find in fact that the same may be said of them as *Simon* (170) asserted for hydronephrosis: "That they consist of a long series of errors of diagnosis followed by an equal number of inappropriate interferences." Now since moveable kidney has no pathognomonic symptoms, it is quite necessary to ascertain the differential signs between it and many pathological conditions which give similar manifestations. Even then it will often be impossible to make a correct diagnosis except by exclusion. An enumeration of all possible sources of mistake would amount to a treatise on the diagnosis of abdominal tumours. Only the most important and frequent will therefore be given here.

Partial contractions of the Recti, Transversi and Obliqui Abdominis muscles frequently give the impression of a smooth oval tumour, which after a certain time disappears on pressure by the palpating hand, just like a moveable kidney. If these patients are put under chloroform, it is true that the influence

of tension of the abdominal walls is eliminated, but the differential diagnosis cannot always be made, because a moveable kidney also usually retreats into its place during narcosis. A conclusion is however arrived at by frequent examination, and especially by carefully conducted strong percussion over the supposed tumour.

It may be very hard to decide whether the tumour is *a tumour of the liver, a liver deformed by tight lacing, hypertrophy of the liver, a liver developed in lappets* (Zipfelbildung[1]) ; or *a moveable kidney without complications,* or *adherent to the lower border of the liver ;* because the same signs on percussion and palpation hold good under both conditions, and the symptoms in both cases may consist of gastric disturbances, jaundice, and vague pains in the lower abdomen. It has already been mentioned that the physical signs deduced from the absence of the kidney from its normal situation are not decisive.

I have observed a patient (see below) who frequently suffers from jaundice, and in whom a diagnosis of moveable kidney adherent to the lower border of the liver would be impossible but for the fact that the moveable kidney could be felt before the formation of adhesions.

Lastly, a conclusion may frequently be arrived at by the presence of some ætiological element, or by the course and consequences of treatment. In this way it took me several months of observation in the case of three patients to diagnose a moveable kidney and exclude an affection of the liver ; in two cases a tumour finally declared itself as a hypertrophic cirrhosis of the liver of obscure origin.

Trousseau (171) relates a case of moveable kidney in which ten physicians had made the diagnosis of a tumour of the liver, an instance to which I could add many others. When *le Ray* (172), referring to the differential diagnosis between affections of the liver and moveable kidney, remarks that a sulcus must intervene between the moveable kidney and the liver, it must be remembered that palpation just at the border of the arch of the ribs is much hampered, and that a similar

[1] There is no English word equivalent to Zipfelbildung. The lappets are generally more or less isolated and moveable over the right lobe. Many of them represent conditions found in the lower animals.—TRANSLATOR.

sulcus may be easily felt on palpating lappets of the liver (Leberzipfeln) and livers deformed by tight lacing.

The most decisive sign is that furnished by the observation of the further course of the affection, and a conclusion is of course quickly arrived at when malignant tumours are present.

A mistake between *moveable kidney and cystic tumour of the liver* (Echinococcus) or *of the gall-bladder* (Hydrops vesicæ felleæ) or *solid tumours of the gall-bladder*, is very likely. The former is ascertained by tapping, the latter only by the course of the disease. How little the statements of textbooks can be trusted (173) when they say that tumours of the gall-bladder are firmly fixed, are situated on the outer border of the rectus muscle, are pear-shaped and give rise to colic, can be seen from the following observation.

On the 24th of February, 1880, I saw Frau E. R—, thirty-six years of age, a patient of Herr Geh. San.-Rath Dr. J. Meyer, complaining of a swelling in the abdomen and violent epigastric pain. She was fairly well nourished, of healthy appearance, she had had five children quickly and had been till lately free from all discomfort.

Her present ailment is said to have been caused by hard work.

Present condition :—Thoracic organs, &c., healthy; well marked pendulous belly. Beneath the right lobe of the liver is a firm tumour of the shape and size of a kidney, remarkable for its extraordinary mobility, and which can be easily pushed downwards, outwards and inwards away from the liver without causing the patient much pain, but (as in *Urag's* case described above) when the pressure is relaxed, returns to its original position nearly in the parasternal line. The tumour itself was painless on pressure, but pressure caused a circumscribed pain in the epigastrium. The functions were normal except that lately the patient had lost her appetite.

The most obvious supposition founded on the above condition was that of a moveable kidney adherent to the liver, which, in spite of its great mobility, was pulled back to the liver by its adhesions as in *Urag's* case. On the other hand it had been pronounced to be a tumour of the omentum.

Meanwhile, after some time an increase in the tumour and

deterioration of the general health were observed, so that thoughts were naturally entertained of a malignant growth. But even then it was impossible to interpret the signs except on the hypothesis that a moveable kidney had become cancerous, especially as the mobility of the tumour remained very considerable. The appearances did not change till June, when pain persisted even during the horizontal position, jaundice appeared, and emaciation advanced so rapidly that it became possible on deep bimanual palpation to feel plainly the lower end of the right kidney in its normal situation. Although the tumour was neither fixed, nor situated at the outer border of the Rectus, nor pear-shaped, nothing but cancer of the gall-bladder could now be diagnosed.

The autopsy, held in the Elizabeth Hospital, Berlin (where the patient attended at last), confirmed this diagnosis.

The instances in which, *vice versâ*, a moveable kidney is taken for an affection of the liver (as has already been mentioned), are still more frequent. It should also be remembered that both affections may occur together. In the case of *Aberle* mentioned above, gall-stones coexisted with moveable kidney; moreover I have seen a similar case myself (see below). If well-marked gall-stone colic, yielding to large doses of morphia, and the passage of gall-stones had not been ascertained, the jaundice which occurred in this case would very likely have been referred to the moveable kidney.

The presence of pain occurring even during quiet decubitus ought to prevent us from mistaking *cancers of the stomach or pancreas* for moveable kidney, even apart from the relative immobility of these tumours.

Such a mistake however is more likely to be made in *cancers of the ascending and descending colon*, which frequently cause nothing for a long time but digestive disturbances (Stenosis), and present to the examining hand a moveable tumour lying beneath the abdominal walls in the region of one or other flexure (I examined such a tumour in the Jewish Hospital of Berlin, which had been wrongly considered to be a moveable kidney). Then again the differential diagnosis is decided by the circumstance that the percussion note over a small cancerous growth in the colon lying immediately

beneath the abdominal walls is always tympanitic; in the case of such tumours when larger and more diffuse, there are so many signs of local and general disturbance that a moveable kidney would hardly be thought of.

In like manner *fæcal masses long retained* in a loop of colon with a long mesentery may simulate a moveable kidney, for the hepatic flexure is a favourite place for fæcal accumulation. A long-continued constipation indeed suggests this complaint, which is at once ascertained with certainty as soon as we succeed in pushing the supposed tumour down into another portion of gut. This however is only rarely possible, and besides, defæcation can proceed almost normally past a fæcal tumour lying in a diverticulum. This condition, viz. that the ingesta lying in proximal portions of the alimentary canal take precedence of those lying in distal portions must be remembered, in order to avoid the mistake of excluding the idea of a fæcal tumour on account of regular[1] defæcation. This can sometimes be easily recognised by the shape of the tumour and its retreat after passive movements into a situation in which moveable tumours are not met with.

A moveable kidney on the left side may be mistaken for a *moveable spleen*. The spleen however during spontaneous and passive movements always lies immediately beneath the abdominal walls, while a moveable kidney when moved upwards retreats from the abdominal wall and slips under the intestines. The note over the spleen is therefore always dull, over the kidney at one time dully tympanitic, at another tympanitic. This sign appears much more trustworthy than the assertion that the spleen must be recognised by its more elongated shape.

It is otherwise in the case of *tumours of the spleen* (*Ague, Leukæmia, Amyloid Spleen*), the size of which indeed forbids their being mistaken for anything but *moveable tumours of the kidney*, but which also, when of a certain size lie close beneath the abdominal walls. In this case however the presence of splenic dulness in the normal situation is decisive, but still more the circumstance that in leukæmia, &c., there

[1] I once saw a woman who had swallowed a small coin, and who, in spite of purgatives and consequent copious daily motions, did not pass it for ten days.—AUTHOR.

are other distinct signs arising with certainty from disease of the spleen.

In the case of the frequently occurring doubt whether we have before us *a tumour of the womb or ovary* or a moveable kidney, the *direction* of the mobility should be tested before all things. If a tumour can be moved towards the lumbar region without causing much pain, a tumour of the generative organs seems excluded. On the other hand a moveable kidney cannot be excluded on the score of slight mobility of the tumour towards the pelvis, for such a tumour can sometimes be moved in that direction. Moreover even tympanitic resonance over the true pelvis does not absolutely disprove the presence of tumours of the generative organs, as the following case observed by me in the gynæcological clinique of *Breslau* shows.

In a woman who had been confined some months previously, a tumour of the shape and size of the kidney was observed at the level of the splenic flexure of the colon. Examination of the generative organs threw no light on its nature. There was tympanitic resonance above the pubes. The autopsy showed the tumour to be an ovarian tumour with a thin pedicle about 20 centimetres long. The tumour owed its unusually high situation to the recently pregnant uterus, and had during the course of pregnancy contracted adhesions with the mesentery and posterior abdominal wall, which held it fast in its abnormal situation in spite of the diminution and descent of the uterus into the true pelvis. As the pedicle which was very likely originally long and thin had been much drawn out, coils of small intestine had naturally taken up their position between the generative organs and ovarian tumour, and thus the tumour, though really intra-peritoneal, had given the physical signs of a sub-peritoneal (retro-peritonealen) body.

On the other hand moveable kidneys have frequently been taken for ovarian tumours, as *Spiegelberg* (174) and others point out. I saw how hard their differential diagnosis from fibrous tumours with long pedicles can be, from two cases, in which it was only in the course of the operation that I was able to make out the connection of the kidney-shaped fibrous tumour with the uterus. In both cases the mobility of the

tumour was extreme, and a certain diagnosis could only be made from the circumstance that in spite of the superficial situation of the tumour and the possibility of grasping it completely, the pulsations of the renal artery could not be felt.

The stock signs given for the diagnosis of tumours of the kidney (175) are useless under the circumstances under discussion.

In making a diagnosis therefore it is recommended to proceed by first disregarding entirely the doubtful tumour itself, and looking to see if the other organs claiming attention are to be found and are of normal shape. With a view to this, bimanual palpation of the *kidneys* should be attempted, the result of which is however seldom trustworthy. But before all things the diagnosis of the presence of the *normal* uterus and both normal ovaries should be attempted by the usual bimanual examination or *Simon's* rectal exploration with the entire hand, which is only of value in this case (176); and these facts can nearly always be ascertained. In making a differential diagnosis between tumours of the kidney and generative organs therefore, the presence of the normal ovaries and of the normal uterus will confirm the diagnosis of a moveable kidney; the presence of only a *single* normal ovary, that of a tumour of the other ovary.

Lastly, there is still a condition under which the wrong diagnosis of a moveable kidney is likely to be made, namely, that of so-called phantom tumour, whose symptoms consist of a permanent and extreme distention of the abdomen with unusually tense abdominal walls, and the ætiology of which is still obscure. Women affected by this complaint generally feel themselves pregnant, say they feel the movements of the child, and so on. The thickness and firmness of the abdominal walls and their contraction on the slightest touch renders palpation useless without anæsthesia, then indeed it is usually possible to recognise the nature of the apparent tumour and to exclude moveable kidney. It is still more frequent for moveable kidney to simulate so-called spurious pregnancy, which will however hardly be mistaken after a careful examination of the generative organs. Other possibilities of error, such as isolated collections of fat in the omentum

and tumours in the abdominal walls need only be borne in mind to avoid mistakes.

The Diagnosis of the Complications of Moveable Kidney.

A moveable kidney may be supposed to be *adherent* when the kidney lies more or less immoveable in an abnormal situation, but still within an area not much farther removed from the first lumbar vertebra than the length of the renal vessels. It is however extremely rare for a displaced kidney to become fixed in an abnormal position; the statements of some writers that the kidney contracts adhesions now and again with the neighbouring parts and becomes surrounded with deposit, like an ovary embedded in a parametric[1] deposit, are entirely unproven. The adhesions are usually very delicate and loose, permit movements of the kidney to continue, and by no means prevent it from being replaced in its normal position. It is true that the kidney returns to the place to which it adheres, which is generally the lower surface of the liver (untere Leberlappen), gall-bladder or transverse colon, so that its return is a test for the diagnosis of the adhesions. No further symptoms are caused by the adhesions, as would be expected from their usual non-inflammatory origin.

The recognition of the *symptoms of incarceration* so-called is only specially difficult when the existence of a moveable kidney is unknown or overlooked. Under these circumstances the extreme sensitiveness of the abdomen and the fixation of the kidney in a high state of œdematous swelling and infiltration forbids careful palpation, so that this condition is frequently mistaken for perityphlitis or perimetritis, on account of the resemblance between the symptoms of these diseases. The sudden appearance of violent symptoms, the conditions of the secretion and excretion of the urine, the suddenly commencing and rapidly proceeding disappearance of the apparently large deposit, however, establish the diagnosis even in these cases.

The *hydronephrosis* of moveable kidneys affords only a *single* sign which distinguishes it from hydronephroses due

[1] ? Perimetric.—Translator.

to other causes, namely, its frequently intermitting appearance attracting even clinical attention. These intermissions are of course observed under other circumstances, as for instance, in cancer of the bladder in the region of the trigone, or in the case of the repeated passage of stones from the kidney, but other symptoms characteristic of these disorders render it almost impossible to mistake them for hydronephrosis in a moveable kidney. It should not be expected that, after the disappearance of the tumour, remains of kidney tissue in the hydronephrotic sac will be palpable through the abdominal walls; the sac is far too thin for accurate palpation and when empty sinks back against the vertebral column. Even after complete evacuation of unilocular ovarian tumours, the most searching bimanual palpation sometimes fails to make out their walls, as I have repeatedly ascertained.

Besides this, hydronephroses of moveable kidney have nothing at all special about them; indeed, as their origin implies, when they become larger they completely resemble the hydronephroses of fixed kidneys. Both grow in the direction of least resistance, the fixed variety downwards, the moveable upwards, so that it is only when they are of a certain moderate size, *i.e.* repletion, that their relations to neighbouring organs, especially the colon, are different. The assertions of *Simon* (177) that in the case of the hydronephrotic sacs of moveable kidneys, the ascending or descending colon remains in its normal situation; and of *Ahlfeld* (178)—that hydronephrosis in a moveable kidney developes in front of the intestines like an ovarian tumour, cannot therefore be regarded as correct. Again, the hydronephroses of moveable kidneys gradually grow into the lumbar region, so that when of a certain size they cannot be distinguished from the hydronephroses of fixed kidneys. I will not here dwell further upon the remaining diagnostic points, especially upon the value of exploratory tapping and the differential diagnosis between hydronephroses and other tumours.

X. Prognosis.

The prognosis of uncomplicated moveable kidney is favourable as far as life is concerned. No case has been hitherto recorded in the literature of the subject in which death could be even probably attributed to it. The fatal case quoted by *Keppler* (179) can hardly be regarded as a proof that an uncomplicated moveable kidney can cause death. Still more must the assertion of *Keppler* (180)—that "Moveable kidney, even without any complications at all, gives rise to slow but continually developing disturbances of nutrition, which, as time goes on, infallibly undermine the health and even life"—be rejected as quite unproven; and we can only agree with *Trousseau* (181) who—referring to the treatment of moveable kidney by leeches, venesection, and still more questionable measures—expressed his opinion as follows: "Le pronostic du rein déplacé n'a vraiment pas de gravité; il ne devient grave que par les erreurs aux quelles il peut donner naissance, et le traitement erroné qui en découle est ordinairement d'autant plus actif que le médecin est moins convaincu."

The prognosis of uncomplicated moveable kidney has however grown decidedly worse since men have begun to remove healthy moveable kidneys, basing their practice on the view set forth by *Keppler*. Of course some of the patients (who otherwise would not have been killed by the moveable kidney which gave occasion to the operation) now pay for this with their lives.

Spontaneous cures of moveable kidneys are commoner, especially in cases of acute traumatic dislocation, and they have been observed in cases in which emaciation following acute febrile affections occasioned the mobility of the kidney.

Rollet (182) gave a correct explanation of this when he said that when the kidney is put back into its normal position and kept there by suitable means such as quiet decubitus, the stretched subperitoneal cellular tissue around the kidney and against the posterior wall of the abdomen, as well as the peritoneum itself, gains time to contract more firmly by

virtue of its elasticity, and thus to afford a powerful obstacle to the displacement of the kidney. *Oppolzer*, *Rayer*, and *Hare* have proved the ocurrence of cures after the supervention of pregnancy. In like manner many observers have seen considerable relief to all the troubles (amounting to cure) follow the menopause and the proper treatment of symptoms by drugs, diet, and mechanical appliances. A cure is sometimes effected by nothing more than a correct diagnosis, and an explanation to the patient of the benign character of her complaint; after which her hypochondriacal frame of mind and numerous imaginary maladies disappear suddenly.

Even with regard to the complications of moveable kidney a good prognosis as far as concerns life is on the whole to be given. Thus, even the menacing symptoms of incarceration have never yet been described as the cause of a fatal termination.

The prognosis after the establishment of hydronephrosis and perinephritic and paranephritic abscesses is less favourable, but even then only in case of improper treatment.

XI. Treatment.

Moveable kidney does not require treatment corresponding to the Indicatio morbi, unless it is the ruling cause of the morbid symptoms. To direct one's treatment against a moveable kidney in a case of general disease due to consumption or cancer, would be just as much beside the mark as to treat a moveable sarcomatous kidney on account of its mobility. Attention must before all things be paid to the causes of the moveable kidney. If they are of such a nature as to act continually on the mobility of the kidney (such as pendulous belly, or prolapse of the generative organs), it will be useless to treat the moveable kidney alone. Fortunately the Indicatio morbi often coincides with the Indicatio causalis; but in debating different methods it is important to be clear from the first about the object to be aimed at in each individual case, and about the end which it is possible as well as desirable to attain by one method or

another; the more so as moveable kidney gives rise to different symptoms in individual cases, even under the same ætiological and anatomical conditions.

As in displacements of the uterus and herniæ, it must here be our task to restore the kidney to its normal situation and keep it there.[1]

Reposition is very easily affected in the horizontal decubitus by moderate pressure after removing all tight articles of clothing. In recent cases, especially such as have arisen after acute injuries, horizontal decubitus persisted in for some time, during which the kidney remains in its normal situation, may be sufficient to produce a cure.

The *maintenance* of a moveable kidney in its normal position is however extremely difficult. To effect this object a number of the most complicated orthopædic appliances, bandages, renal trusses, &c., have been proposed and tried. The most inconvenient are those which depend for their support on the soft abdominal walls alone, for they are constantly shifting, and, to start with, do not admit of a mode of fixation similar to the ordinary trusses for keeping up the bowels. Better than these are the bandages to fasten on the thorax and provided with a pad, which exert leverage for the most part on the spot occupied by the kidney in the erect position. *Guéneau de Mussy* has described an angular pad for this purpose, the lower and horizontal arm of which is intended to support the displaced kidney, and the perpendicular arm to prevent its moving in an inward direction. All these pains however to maintain the replaced kidney in its position must remain fruitless, for the kidney is covered by the bowels, and is exposed to no pressure from the pad when the pressure is moderately strong, while an increase of pressure will drive it away from the pad. These appliances therefore, being useless and much more apt to cause pain and discomfort than their relief, are soon discarded by the patients of their own accord.

On account of the incompetence of mechanical appliances to fix a moveable kidney in its position, it has been quite recently proposed to fasten the kidney *by operation*, namely

[1] This association between moveable kidney, hernia, and descent of the uterus will be mentioned further on, see p. 357.—TRANSLATOR.

by sewing it to the abdominal walls (183). Favourable results however from this proceeding, based as it is on incorrect anatomical and pathological ideas, is not to be expected. If an attempt were made (as by *Hahn*) to make an incision in the lumbar region and get the kidney to heal in it, this proceeding would displace the kidney, whose normal situation is higher and more lateral than the lumbar incision. But if an attempt is made to sew the kidney into its normal situation, it would be necessary to resect one or two ribs, or insert sutures at random into the last intercostal space through the muscles and the kidney. In either case one would expect to open the pleural cavity and to puncture important vessels and nerves. But even after the sewing has been safely accomplished, a cure of the moveable kidney is not to be expected, for we have not two serous surfaces, capable of mutual adhesion, to deal with, and the causes of the mobility of the kidney are of course not removed by sewing.

All these experiments, unpromising and dangerous as they would seem *a priori*, can however be abandoned, as it is not necessary, in order to remove the discomforts caused by moveable kidney, to fix it in its normal situation; but just as in the case of retroflexion of the uterus it is generally sufficient to keep it as immoveable as possible. With this view we must act indirectly on the kidney by fixation of the abdominal walls and viscera, since it is impossible to act directly upon it. This is best accomplished by a comfortable and firm binder encircling the whole abdomen. If the kidneys are only prevented from constant movements downwards and inwards, and from rotation on their axes, the discomforts caused by dragging of the nerves and vessels as well as by pressure, cease. It is of course difficult to produce general immobility of the abdominal contents and walls by a binder, because the size and shape of the abdomen vary not only in different individuals, but even in the same person, during breathing, and with different postures, movements, &c. Even binders accurately made to the measures of the abdomen do not therefore fulfil the desired end, for they ride up as the patient walks, and leave the lower half of the abdomen free for the descent of the abdominal contents, or they exert

an unbearable pressure over the region of the stomach by their upper circuit.

The material and shape of the binder must therefore be varied in the most manifold ways according to the condition and occupation of the individual. Elastic binders of india-rubber are least of all to be commended, as they produce the greatest pressure and also retain the perspiration. Ticking is better in summer, flannel in winter, with lengths of some elastic material let in. Further, it may be convenient to prevent the abdominal binder from riding up by means of a T bandage or perineal bands (Schenkelbänder), and to fix it by braces to the shoulders. I have never seen any special good from padding the binder over the situation of the displaced kidney. It is preferable to let in a thin concave tin shield over this spot, as I have often tried with success.

All these binders are however uncomfortable for many reasons, and I have therefore tried to find a material which is more or less capable of forming an *artificial abdominal wall* by itself. My experiments with poroplastic felt, thin tin and whalebones let into the front surface of the binder have not however been very successful, but I have been led by these experiments to an appliance, which, modified to suit our purpose, is capable of almost completely fixing the abdominal walls and with them the moveable kidney—namely, the much abused stays, the use of which is at once shown by the support it gives to the breasts and the defence it affords to the abdomen from the pressure of the strings of the garments. I have obviated the disadvantage of the usual short stays reaching about to the navel, which compress the upper part of the abdomen and allow the lower part to bulge, by having the longer stays lately come into fashion lengthened as far as the pubes and both Poupart's ligaments. Shaped thus, they cover the whole abdomen, they exercise an equable pressure on the abdominal contents, without being tightly laced, and (unlike all forms of abdominal binders) taking their support from the bony thorax, they are not displaceable.

There are few women, mostly those whose bellies are very pendulous, or who suffer from violent epigastric pains, who cannot bear these stays. Women again who have sedentary

occupations cannot wear them unless the lower end of the middle piece, the so-called busk (Blanchette) is well padded, or, as I have tried, arranged to fold up. But in most cases these long stays act extremely well. The favourable effect of this *artificial abdominal wall* consists not only in rendering the abdominal contents immoveable, but at the same time in the temporary removal of one of the fundamental causes of moveable kidney, pendulous belly, for the radical treatment of which we possess no method.[1]

The abdominal muscles, which are generally loose in cases of moveable kidney and pendulous belly, must however be also directly strengthened. To this end the use of cold in the form of cold compresses and douches, or sensible hydropathic treatment or sea bathing, or lastly electrical treatment of the abdominal muscles by the induced current, are to be recommended.

Methodical shampooing of the abdomen again appears beneficial in many ways in the treatment of moveable kidney ; in the first place it strengthens the abdominal muscles, in the second place it is capable of removing constipation with its peculiarly evil consequences, by exciting the peristaltic movements of the intestines, and in certain cases by direct mechanical depression of the fæces (184).

By the combined use of the above methods remarkably good results are often obtained. A stay of several weeks at

[1] The above considerations on the very important subject of stays lead us back to the question of the intra-abdominal pressure. Moveable kidney seems to be a member of the Hernial Group (including Hernia, Pendulous Belly, and Descent of the Pelvic Organs, including, probably backward displacements of the womb even where descent is not well-marked, Prolapsus Ani, &c.), one of the characteristics of which is the relative *increase* of the intra-abdominal pressure (relative that is to the resistance afforded by the supports). We need not again discuss the question of increased buoyancy of the kidney by increase of the intra-abdominal pressure, which seems not to be the main question. But increase of pressure by stays will *increase* the intra-abdominal pressure, and will almost certainly tend to increase the descent of a hernia, of the pelvic organs, or of the rectum, all of which must be considered together. It is quite likely that the stays will tend to fix the viscera, but they are at best only palliative appliances, and it must not be forgotten that by increasing the intra-abdominal pressure they come under the class of palliatives which increase the original morbid state.—TRANSLATOR.

sea-bathing places or hydropathic establishments, in which shampooing and electricity can be applied by skilled hands is therefore advisable for patients afflicted with moveable kidney and pendulous belly.

It is obvious that associated maladies, such as chlorosis, anæmia, chronic catarrh of the stomach and intestines, as well as descent and flexions of the uterus require special treatment, and that in the case of serious general diseases treatment which gives exclusive attention to the moveable kidney is inadmissible.

An entirely different method has been lately recommended for the treatment of moveable kidney ; this will be discussed in the following section.

On the removal of Uncomplicated Moveable Kidney.

After *Keppler's* (185) assertion, that "Moveable kidney as such, even without any complications at all, should be removed by excision through the abdomen, as soon as it causes any disturbances in the system," and in accordance with this advice, —up to the end of 1880 six healthy moveable kidneys have been removed, four of them by *Martin* (186), with two deaths, once by *Smyth* (187), and once by *Merkel*, in whose case the result was fatal. Thus of six persons three died.

The excision of a healthy moveable kidney, however, is in every sense unjustifiable. We possess milder mechanical and therapeutical means of alleviating the discomforts of a patient with moveable kidney, nay even of removing them entirely, as is shown by numerous observations of the most trustworthy writers. Reasons again for the extirpation of an organ so important for the human economy as the healthy kidney have neither been produced, nor indeed do they exist. There neither exists an accepted case in which death has been caused by moveable kidney, nor are the cases brought forward by *Keppler* as proof of its injurious influence unequivocal or conclusive. The fact that patients whose healthy moveable kidneys have been removed felt well immediately after the operation, and suffered no inconvenience from the moveable kidney, can only astonish and enlist on

the side of the operation a man who would think it remarkable that a woman, whose healthy ovaries and healthy uterus have been removed, ceases to suffer from uterine hæmorrhages.

It requires no demonstration that an operation, however brilliant, does not seem to be indicated for its own sake, but only when it restores the patient to health. From this point of view the amputation of a normal cervix uteri or the removal of normal ovaries, which perhaps give rise to dysmenorrhœa or other slight ailments, is not to be endorsed; how much more blameworthy does the extirpation of an organ, so important to life as the kidney seem to be!

What should we say of the recommendation to amputate a leg for sciatica or varicose ulcers, a recommendation which stands on the same footing as that here given—to remove the kidney?

Besides it is certain that if the operation is undertaken on the above indications, some of the patients will die from the operation itself (septicæmia, hæmorrhage, &c.). Moreover, putting these accidents aside, a fatal issue is certain if (as in *Merker's* case), after the moveable kidney has been removed, the other, in a state of fatty degeneration, is no longer capable of vicariously supplying the function of the one removed; or if (as in a case of *Lange's*) the other kidney is hydronephrotic and so the occurrence of uræmia is inevitable. It ought also not to be forgotten, in considering the recommendation to remove healthy moveable kidneys, that cases in which there is *only one* kidney are by no means rare. Even *Willis* (188) collected thirty-six such cases out of the literature of the subject, *Beumer* (189) forty-eight. There is however no means of diagnosing the absence of one kidney without opening the abdomen. Thus in many cases the operation in question will be the direct cause of death.

But the life even of those patients who survive the operation will be shortened if the other kidney, although present, is attacked by certain diseases, such as stone, hydronephrosis, Bright's disease,—complaints which, in the presence of a second kidney to supply vicariously the function of the first, are by no means fatal. It is no accident that calculous affections are especially frequent in cases in which only one

kidney is functionally active. Lastly, it is not a matter of indifference—even in the case of other diseases which (apart from the weakness of old age) finally befall every human being—whether one or two kidneys provide for the secretion and excretion of the urine. Although under physiological conditions one kidney takes up the function of the other, hypertrophies, and so on, it appears very doubtful whether in case of affections of the heart, lungs and liver, *one* kidney is enough to discharge the renal functions in the presence of the disorders of elimination present under these conditions and to replace the other. Certainly the chances of the maintenance of life are less in persons with *one* kidney than with two; how much more when the second kidney also is or becomes moveable.

All these dangers must be faced by the physician who advises so serious an operation, even although the chances, as far as the operation is concerned, are of the most favourable kind in the present state of surgery. The rage for operation is far more dangerous in its consequences when it concerns the kidneys than when it concerns the cervix uteri and ovaries which are not necessary for the maintenance of life. The hypochondriacal frame of mind and the wish of the patients to get thoroughly rid of a malady which they regard as malignant, ought not to decide the physician to undertake, for the sake of an exploit which imposes on the laity, an operation which endangers and shortens life. Unfortunately, nephrectomy which frequently saves life in cases of abscess, stone in the kidney, &c., is only discredited by such proceedings.

Treatment of the Complications.

Special consideration is required with regard to the treatment of the symptoms of incarceration so-called. The importance of a correct estimation of the pathogenesis of this train of symptoms is here very plain. If these symptoms were due to the actual incarceration of the kidney, it would be our duty to try and replace the

kidney by every possible means, at any stage of the complaint. As in the case of strangulated hernia, the inflammatory and other concomitant symptoms would disappear after reduction. But experience shows us that forcible attempts at reduction are useless, and that the complaint usually ends favourably without them. We should therefore lay well to heart the advice of *Rollet* (190), which is contrary to the ordinary views of the nature of this affection. *Rollet* says we should put off the reduction of the kidney "until the inflammatory symptoms have yielded to antiphlogistic measures, which, as experience shows us, generally happens in a few days." Indeed reduction is usually contraindicated by the condition of the kidney itself, which swells in a very short time, and in some cases in a few hours, to double its size, and therefore is too big for its old situation. But when, some days later, the inflammatory symptoms have subsided, all attempts to replace the kidney become at once superfluous. For the cure of the kink, twist and subsequent thrombosis of the renal vein proceeds spontaneously, either by the establishment of a collateral circulation, or by organisation of the clot, or by the undoing of the kink or twist in the vein, without any interference, if the patient is kept on her back.

If it is however decided to attempt reduction, the attempt should be made in the first few hours of the attack, with the greatest precautions, if possible, in a warm bath and under chloroform, especially as we can never know whether the renal vein is already thrombosed, and, if so, how far. Finally, a purely expectative treatment and the avoidance of further injurious influences are advisable. It is proper to assuage pain by opium and warm baths according to the symptoms; if inflammatory symptoms become prominent, it is sufficient to apply an ice-bag over the prolapsed kidney. It is also advisable—not so much with a view to mitigating the inflammatory symptoms as to relieving the engorged kidney—if the swelling becomes intense, to apply several (six to ten) leeches to the renal region.

This expectative treatment must however be at once abandoned, as soon as symptoms of a *perinephritic or paranephritic abscess* manifest themselves; but this will be seldom.

Free incisions in the region of the abscess, and even the removal of the kidney are in this case, and this case alone indicated. This event is happily so rare, that I have not succeeded in finding a single case in point in recent literature.

Treatment of Hydronephrosis in a Moveable Kidney.

Although Hydronephrosis in a moveable kidney shows its characteristic intermissions from a clinical point of view, we can wait quietly till it has become stationary. But even then nothing but the general treatment of hydronephrosis is appropriate; and indeed only two methods deserve consideration, namely, *total extirpation of the sac*, and *establishment of an abdominal fistula from the pelvis of the kidney.*

Kroner's (191) list shows that the *whole sac has been extirpated* eight times up to the present date; of these operations four were performed unintentionally by mistake for an ovarian tumour; four were fatal. I will not discuss the performance of the operation, nor the special question whether the entire sac should be removed through an abdominal or lumbar incision, especially as the last interesting question has been exhaustively discussed by *Barker* (192) and *Czerny* (193). We should be particularly careful in dealing with the pedicle, on account of the frequent abnormalities of the renal vessels, especially the vein, and on account of an eventual thrombosis of the renal vein. As in the case of tying the navel-string in new-born children a clot may be pressed through the ductus venosus (Arantii) (194), so here the same cause may force a clot in the renal vein also into the vena cava. Thus, in a case of *Barker's*, embolism of the pulmonary artery was found after the unfortunate removal of a sarcomatous moveable kidney. The renal vein was found cut across close by the vena cava; it was not observed either on dissection or during the operation; there had been hæmaturia during life, so that it does not seem to me impossible that death was caused in this case by a clot in the renal vein, as described above.

The prognosis of the second method of cure now under

consideration, namely, the *establishment of an abdominal fistula from the pelvis of the kidney,* as recommended by *Simon,* is far more favourable. All the cases hitherto treated in this way, by *Ahlfeld, Pernice,* and *Landau* ended favourably. The evacuation of the urine through the fistula in no way interfered with the occupations of the patients, and the site of the fistula, as my case (see Fig. 9) shows, was situated most favourably for the escape of the urine.

With regard to the mode of operating, it is advisable to *operate at one sitting,* as has already been often done after a wrong diagnosis has been made, and as I have done intentionally. In this case, as in that of abdominal hydatids (195) and of extra-uterine pregnancy (196), I consider that my plan of stitching the sac of the hydronephrosis (to the abdominal wall) before opening it (as against *Kroner's* plan) offers decided advantages ; for the contents of the sac, which may be mixed with pus, the products of decomposition, and colloid masses, are not harmless to the peritoneal cavity. It appears however highly disadvantageous to operate at two or more sittings ; of such methods, *Simon's* method by puncture is certainly the best.

The question whether, after an abdominal fistula has been successfully established in connection with the pelvis of the kidney, *Simon's* proposal of passing a probe along the ureter from the bladder or from the wound, in order to make it pervious again, and then closing the abdominal wound should be adopted, has still to be discussed. But attempts to effect this offer little prospect of success, as dissections of hydronephroses show ; and even if they were to succeed, the reappearance of the old hydronephrotic condition might be anticipated, so that it seems better to put this operation entirely aside.

An attempt might also be made to destroy the remains of the kidney which still secreted urine, by caustics, the actual cautery, &c ; but this is painful, and (as *Simon's* attempts, in such a case which lasted for months, show) it is hardly possible. In this case we should still have to perform extirpation of the remains of the kidney by a subsequent operation, which could have been done in the course of the first operation. But this also seems to me unjustifiable, for

extirpation is a dangerous operation, and the presence of kidney tissue which is still healthy, able to discharge its functions, and cannot be replaced, is here of the greater value, since the risk of the affection of the other kidney (as autopsies on cases of moveable kidney show) is increased.

In debating the question whether one should decide once for all to extirpate the whole sac, or to establish a fistula, apart from the above considerations, individual circumstances must be considered, such as the age and occupation of the patient, the presence of still active kidney tissue, and the state of the other kidney. Other things being equal, I would not myself decide on extirpation, even if it were no more dangerous than the establishment of a fistula. Experience shows that after the establishment of a fistula, the flattened renal papillæ, now freed from pressure, recover themselves and secrete urine nearly normal in quantity and quality, so that by this operation the patient regains at once the use of a kidney which had been rendered useless.

XII. Original Observations.

(1) *Moveable Kidney on the right side, Pendulous Belly, urinary troubles.*

Cl. St—, 44 years of age, has had 11 children in 12 years, the last seven years previously. She complains of violent pain and a swelling in the right side, the removal of which had been advised on the ground of its malignant nature. She suffers from frequent calls to pass water, pains in the loin, &c. After using a long pair of stays the patient feels quite well, for the tumour is nothing but a moveable kidney.

(2) *Moveable Kidney on the right side, Pendulous Belly.*

D. Kr—, 32 years of age, has had 7 children and 2 miscarriages. She is to undergo an operation for an abdominal tumour. She is a strong buxom woman with very little dis-

comfort, and nothing amiss except a moveable kidney on the right side and a large pendulous belly. She gets on very well with an abdominal belt and without an operation.

(3) *Moveable Kidney on the right side, Pendulous Belly after ovariotomy.*

N. S—, 47 years of age, no children, observed an abdominal tumour when only 19 years old; this has constantly increased for the following 24 years. When taken in to my clinique the circumference of the abdomen was 108 centimetres. After the removal of a multilocular ovarian tumour the patient at first felt very well, but a ventral hernia formed, and, together with lax shrivelled abdominal walls, contributed to form a pendulous belly. Since then she has complained of feeling as if she had something alive in her abdomen. Examination shows a moveable kidney on the right side.

(4) *Moveable Kidney on the right side. Sensation of a foreign body in the abdomen, increased inconvenience during menstruation.*

Frau A. H—, 34 years of age, has had several children in a short time. Since her last confinement (two years ago) she says she has worked very hard and in doing so has strained herself. She has never worn stays. Whenever she stands she suffers violent dragging pain in the abdomen. Her feelings, according to her own account, are "as if something had been unhooked in her abdomen, or as if all her inside were falling out. Now and then as if a heavy body turned round in her belly." If she lifts her right arm up she has a peculiarly unpleasant sensation, "as if all her side was falling away." All her troubles are worse during her menstruation (which is regular) and during work. Also she has frequent calls to pass water, and has not been able to lie on the side for two years.

I found an unusually moveable kidney on the right side. On moving this to and fro the same unpleasant sensations

were produced as those which the woman complained of. The generative organs were normal except that the uterus was low down.

The account of this woman, who was considered hysterical, is all the more valuable from the fact that neither she herself nor the doctors who had treated her had detected an abdominal tumour.

(5) *Moveable Kidney on the right side, symptoms of incarceration, adhesions to the liver, jaundice.*

J. W—, 55 years of age, has had 4 children, last menstruation nine years ago. After the last confinement, sixteen years ago, the patient observed a lump in the right hypochondrium, which was at first very moveable. For some years she has suffered from a peculiar boring and pinching pain deep down in the abdomen. Two days ago she was suddenly seized with retching and a darting pain in the abdomen especially on the right side, and became much collapsed. The abdomen is much distended and painful even on the slightest touch. Percussion is dull from just below the liver to the level of the iliac crest. Palpation is impossible on account of abdominal tenderness. Improvement soon followed the use of opiates and ice poultices. It was some weeks before the kidney could be felt, easily displaceable into the lumbar region. On pressing this kidney the patient complains of a circumscribed pain in the[1] left side of the epigastrium and the left loin. The dulness below the liver has quite disappeared. A year later, examination showed the right kidney no longer so moveable as before ; on pressure it can be replaced into the lumbar region, but on relaxing the pressure it soon reappears, even in the horizontal posture, below the right lobe of the liver.

Two years after the first acute attack Frau W— had a fresh attack of violent pain in the abdomen, which again became very tender and distended, the tumour below the liver

[1] The fact of pain on the *opposite* side to the kidney affected (see Cases 5 and 7) must be remarked. A similar transference of sensation is not very rare in the case of the ovaries.—TRANSLATOR.

considerably increased, but little moveable, the kidney shape recognisable. After some days distinct jaundice appeared, and lasted some three weeks, accompanied by symptoms of high fever which showed a distinctly intermittent type; the spleen was very large. The fever only yielded to large doses of quinine, so that I do not hesitate to consider this an intercurrent attack of ague. How far the adhesion of the kidney to the right lobe of the liver, which had meanwhile taken place, contributed to the jaundice, I will not decide. At the present time the patient is quite well.

(6) *Moveable Kidney on both sides, especially the left.*

Cl. V. S—, 30 years of age, has had no children, she has menstruated regularly, and has lately lost much flesh, which circumstance alone induces her to seek medical aid. On examination on April 23, 1881, I discovered both kidneys very moveable, especially the left, the uterus retroflexed; nothing else. There was no discoverable cause for the moveable kidney except the extreme and apparently rapid emaciation.

(7) *Moveable Kidney on both sides, right more than left.*

M. St—, 25 years of age, had a child five years ago. Complains of violent pain, especially in the left side of the abdomen, in the last left intercostal space, and in both loins. The urine is now and again scanty and dark, during which time the patient feels worse. She also complains of an increase in all her symptoms at every monthly period. Both kidneys moveable, the right more than the left. Pressure on the right kidney causes pain in the left side. Moderate pendulous belly, moderate amount of subcutaneous fat. No other cause than the habit of carrying a heavy coal-box on the hips, as the patient has done for years, can be found.

(8) *Intermittent Hydronephrosis in a Moveable Kidney on the right side. Perinephritic abscess, establishment of an abdominal fistula from the pelvis of the kidney at one sitting. Cure.*

C. M—, 60 years of age, has had 2 children, born respectively forty and thirty-six years ago. After the last confinement the generative organs became prolapsed. For about the last ten years she has suffered from pain in the sacrum, constant abdominal discomfort, epigastric pain, gastric troubles, dragging and pain in the loins and pressure in the region of the bladder. Eight years ago she observed for the first time a moveable tumour in her abdomen, which however disappeared from time to time, and was variously regarded by the different physicians under whose care she was, as a hydatid of the omentum, an ovarian tumour, and as a tumour of the liver. On September 14, 1879, the patient came to my out-patient room.

Present condition.—A pale emaciated woman with prolapse of the anterior vaginal wall and uterus. In the right hypochondriac and epigastric regions is a tensely elastic globular tumour, passing almost imperceptibly into the liver above, and reaching internally almost to the linea alba, externally almost to the axillary line, and inferiorly some three centimetres below the level of the navel. The tumour is freely moveable, especially inwards and downwards. On depressing the tumour downwards, a sulcus is remarked between it and the liver. The percussion note over it is dully tympanitic, and quite dull only on the summit of the convexity; in the axillary line it is impaired.

Aspiration with *Dieulafoy's* finest needle (which withdrew about three cubic centimetres) proved the contents of the tumour to be a limpid fluid, white even to transmitted light, clear, destitute of formed elements, non-albuminous, and containing abundance of chlorides.[1]

It could therefore be only a hydatid of the liver or kidney, or a hydronephrosis in a moveable kidney. After repeated examination in different postures on September 20th, 1879,

[1] Specific gravity?—TRANSLATOR.

and after withdrawing about three cubic centimetres of fluid by a second aspiration, the operation was fixed for the following day. On the next day however the tumour had disappeared, and percussion showed the presence of intestine in its place. Deep palpation failed to detect any firm body, even on pressing deeply beneath the right arch of the ribs. On inquiry, the patient—who felt comparatively well—declared that after the examination of the previous day she had passed an unusually large quantity of limpid and clear urine, an occurrence by no means unusual with her, for the quantity of urine was constantly varying. The urine itself was normal.

It was still doubtful whether the fluid from the cyst had escaped by the fine puncture of the sac into the peritoneum or had been passed *per vias naturales*. In the former case, the diagnosis would remain doubtful, in the latter it would certainly be hydronephrosis. It was possible that the kinked or twisted ureter might have untwisted itself by rotation of the sac in consequence of examinations in the very various postures which had been adopted; or it was possible that the diminution of pressure in the sac in consequence of puncture might have removed a valvular impediment.

For the first few weeks however the diagnosis remained doubtful, for the urticaria characteristic of hydatid did not appear, and the tumour did not return. It was not until February, 1880, that the patient came back complaining of unbearable pressure in the abdomen, and presented much the same appearances as on the first examination. The diagnosis of hydronephrosis was now sure, and was moreover confirmed by a careful chemical examination of the fluid which was now withdrawn in somewhat greater quantity. The report on the result of this examination kindly made by Priv.-Doc. *Dr. Lewin* is as follows:—"The fluid was slightly turbid and its reaction, as far as could be determined by gaslight, faintly alkaline. The turbidity disappeared on the addition of nitric acid. This circumstance shows the turbidity to be due to the presence of phosphates. No uric acid crystals can be found, and the proof of the presence of uric acid by the murexide test remains doubtful on account of its being conducted by (artificial) light. Urea, on the other

hand, is proved to be present; for after eliminating the phosphates, the addition of nitrate of mercury to the fluid gave a white precipitate of hydrargyrate of urea. The proof of the presence of urea by converting it into nitrate of urea succeeds even better; for on leaving the fluid treated with nitric acid to spontaneous evaporation, characteristic rhombic crystals of nitrate of urea lying over one another like tiles on a roof, separated out."

I was able to observe the refilling and emptying of the cyst four times more, each time with the above described symptoms connected with excretion of the urine. At last however the sac did not disappear, the general health visibly declined, a course of Carlsbad failed to improve the gastric symptoms, continued pyrexia ensued, the aspirated fluid, which was formerly clear and destitute of formed elements, contained many pus corpuscles, so that I could no longer delay the operation. Meanwhile the tumour had increased, and had become very tense, and very tender to the slightest touch.

On June 20th, 1880, I operated, making my incision from the angle of the right costal arch, more or less parallel to the fibres of the internal[1] oblique muscle, towards the navel, and about twelve centimetres long; a fleshy-looking cyst-wall appeared. As the excessive tension of the tumour prevented its being grasped, it was punctured with a fine needle and some 300 cubic centimetres of very purulent fluid drawn off, the incision was then prolonged for some three centimetres towards the axillary line, after which it became possible to get round the tumour in all directions. It was found to be bounded above by the liver, internally and in part anteriorly by the intestines, externally by a second tumour reaching to the vertebral column, which proved on puncture to be a perinephritic abscess with thicker, more tenacious and more purulent contents than those of the hydronephrotic sac. The idea of the entire removal of the sac was therefore given up on account of this perinephritic abscess, and the cyst-wall was stitched to the abdominal wall on both sides of the

[1] In the above sentence the word "internal" appears to be a mistake for "external." The fibres of the external oblique run downwards and inwards those of the internal oblique in this region downwards and *outwards*.—TRANSLATOR.

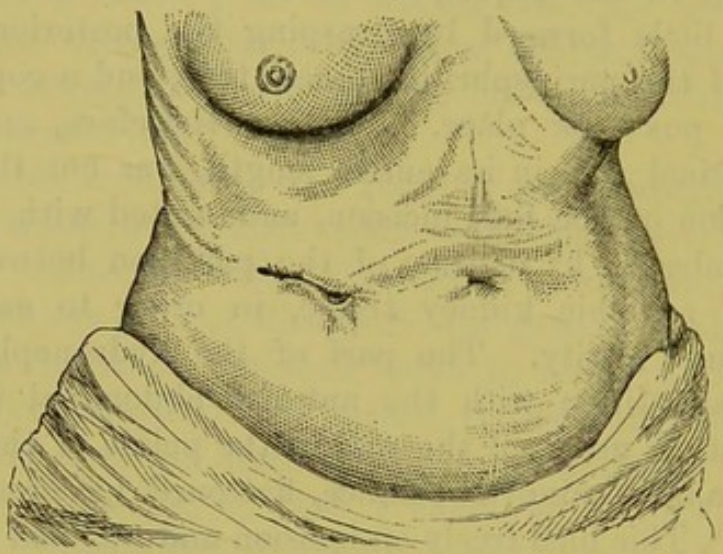
wound, beginning from the inner angle of the wound, the sutures thus running at right angles to the direction of the incision. The peritoneal cavity was thus protected from the escape into it of purulent fluid, a circumstance which proved very useful in the sequel, for in an endeavour to lift the tumour a little forward by grasping the posterior surface, the wall of the perinephritic abscess tore, and a copious discharge of pus took place. The sac therefore, stitched to the abdominal wall in its entire length, was cut through in the direction of the first incision, and united with the perinephritic abscess by incision of the partition between them formed of atrophic kidney tissue, in order to establish a single wound cavity. The part of the hydronephrotic sac lying in apposition with the anterior abdominal wall does not consist, as usual, of the wall of the pelvis of the kidney, but of genuine kidney substance, flattened by pressure and atrophied. This bled freely on incision and was tied *en masse*. The fluid evacuated was highly purulent, its hydronephrotic receptacle larger than a child's head, everywhere studded with flattened and atrophied portions of kidney. The opening of the ureter could neither be seen nor found by a probe.

The sac was now further secured by several (12) sutures and the cavity drained. The subsequent course of the case presented nothing remarkable. On July 19th the whole wound was closed with the exception of an opening at its inner angle, as big as a finger. In this an india-rubber tube was left, passing easily some fourteen centimetres into the cavity, and allowing an easy escape to the urine which was at first scanty, but gradually became more and more copious, and also permitting the cavity to be washed out. The fluid, which was at first purulent, became more and more clear and urinous, the sac contracted towards its centre, the renal papillæ which were flat and atrophic became more and more compact and hypertrophied. The ligatures on the renal substance did not come away for three and a half months. A renal papilla was seen to be slightly everted through the inner angle of the wound.

A portable urinal was not well borne, and was moreover superfluous, for the patient was by no means impeded from leading her ordinary life and pursuing her ordinary occupa-

tions with a simple linen bandage, and a simple drainage-tube in the inner angle of the wound. The appended drawing, taken after the expiration of a year, shows the scar

Fig. 9.



Fistulous opening in the abdominal wall from the pelvis of the kidney.

to have contracted, but otherwise to have undergone but little change in relation to the wound. The fistulous opening which remains, situated at the inner angle of the wound, is only about one centimetre in diameter.

(9) *Peculiar connection between Moveable Kidney and Hydronephrosis (Eger)*[1].

Frau M—, 29 years of age, had been delivered of a son ten years previously. Nine months later she began to feel a slight gnawing pain in the left lumbar region, which disappeared after a short period of recumbency, and during the next few years returned but seldom and then always after mental disturbance. Four years back she became pregnant for the second time; during the first two months the gnawing pain returned, and remained till sixteen weeks after her confinement. At this time she weaned her child on account of

[1] I had the opportunity at Breslau of examining this case, which was under *Eger's* treatment, and was described in the 'Berl. klin. Wochenschr.,' 1876, No. 28.—Author.

anæmia. Shortly afterwards the pain in the left lumbar region increased and violent attacks developed, which did not leave the patient for years, defied all treatment, and persisted until *Eger* saw her for the first time on January 13, 1874.

She is a slender but muscular woman, with normal circulatory and respiratory organs, normal spleen and liver dulness. The only striking thing about her is the absence of subcutaneous fat, anæmia and the remarkable laxity of the abdominal walls. The attacks alluded to were much alike; their mode of occurrence was that, independently of physical exertion, imprudent conduct, the monthly period, or psychical disturbance, the patient is attacked while at work by a pain in the left lumbar region, radiating to the inguinal and umbilical regions, which is so violent as at once to render her incapable of work. A rigor then occurs, followed by violent vomiting of bile-stained mucus, with coldness and pallor of the extremities, fainting fits, and deep depression of spirits. The pulse is small, infrequent, no rise of temperature. Immediately after the commencement of the pain, a smooth, bean-shaped tumour, with its convexity directed outwards and downwards quickly makes its appearance beneath the left arch of the ribs in the umbilical and hypogastric region, which at once feels like the kidney. Usually it is closely apposed to the abdominal wall, but is nevertheless overlaid by coils of intestine; it is always separated from the hepatic and splenic dulness by wide areas containing intestines. The tumour maintains the same size throughout the attack, and is immoveable. Any attempt to displace it is extremely painful and always unsuccessful. On one solitary occasion the attempt to displace the tumour upwards succeeded, though with enormous suffering, but the attack ceased some hours earlier than usual.

Simultaneously with the appearance of the tumour, frequent and very annoying desire to pass water set in, during which only a few drops of clear and *always non-albuminous* urine were voided.

A whole day usually, and often a day and night or longer, elapsed before the attack ended. The troubles were as severe during the whole night as by day, every morsel of

food and every drop of water swallowed were vomited. Then the pain ceased, usually all at once, and the tumour disappeared as rapidly as it came, with eructations, vomiting, and the passage of an increased though not considerable quantity of urine, which was bright yellow, clear, and always non-albuminous, and the health became perfect with the exception of an easily intelligible weakness.

In this manner the attacks returned at intervals of fourteen to twenty days, once they ceased for six weeks. An attempt to prevent the descent of the kidney (which was the only imaginable condition) with a padded binder was unsuccessful. Morphia alone afforded the patient constant, though only slight relief.

Then, in July, 1874, the patient became pregnant again, for the first time for three years. Instead of the relief hoped for from the chance of the mechanical prevention of the prolapse of the kidney, a considerable aggravation of the symptoms ensued during the next few months. The attacks became more violent and the intervals were often hardly as long as a week. When at last the attacks seriously threatened the general condition of the patient, they suddenly ceased between the fourth and fifth months of pregnancy. The patient became free from pain for the first time for three years, an occasional gnawing in the left inguinal region (which did not amount to real pain) was the only reminiscence of the terrors of the past. Pregnancy, and labour, which ensued on March 12, 1875, ran their course without disturbance. The uterus was however hardly quit of the child and after-birth, before a tumour of the size of a small child's head pushed itself forward without any pain, in the left side of the hypogastrium, beneath the left arch of the ribs. It was tense, elastic, irregularly nodular, not tender on pressure, and absolutely immoveable from its place. Entirely unlike a kidney, it remained hardly half a centimetre from the linea alba and usually had coils of intestine in front of it. The percussion note over it was dull, or more or less tympanitic, according to the position of the coils of intestine over it. It was separated from the hepatic and splenic dulness by broad bands of intestinal resonance. The uterus and its appendages proved, on subsequent examination, to be entirely unconnected

with it, and normal. The tumour, which gave the abdomen a peculiar, irregular appearance, underwent during the time of its persistence not the least diminution in size or consistence. There was always an evacuation of a normal quantity of non-albuminous and clear urine.

With the exception of an uncomfortable feeling of repletion, the tumour gave the patient no trouble. On the 15th of May—eight weeks after her confinement—while the patient was going to bed, she felt the size of the tumour diminish, without any disturbance of her general comfort. Next morning it had disappeared. The abdomen now remained quite free from the tumour till May 28, on which day the monthly period reappeared for the first time, with some slight indisposition, and a feeling of weight in the lower abdomen, but sleep was not especially disturbed by it. Next morning a small tumour appeared in the old place, and within a few hours reached the size of that which had been there before the confinement. It behaved in all respects like the former tumour, and remained till June 20, without any disturbance of the secretion and excretion of the urine. On that day the patient washed the room and staircase with bare feet. The next night she experienced frequent desire to pass water, and passed an enormous quantity of limpid urine. Next morning the tumour had vanished, and has remained invisible till now (August). The patient, who resumed her work in the manufactory soon after her confinement, has never felt so well as now, her appearance is incomparably more healthy. Her urine, which has been frequently examined, is normal in quantity, always bright yellow, clear, free from albumen and morphological constituents.

In the annexed table I have given a short *resumé* of the cases which I have observed up to April, 1881, and most of which I have demonstrated in my courses for doctors in practice and in my Lectures.

I. *Moveable Kidney on the right side.*

No.	Age.	Number of confinements.	Other ailments.
1. W.	36	9	Retroflexion of the uterus.
2. R.	32	7	Pendulous belly.
3. F.	35	6	Pendulous belly.
4. R.	33	?	—
5. B.	49	7 in 14 years	Prolapse of the anterior vaginal wall.
6. H.	47	12	Cancer of the womb.
7. R.	48	5 in 6 years	Retroflexion of the uterus.
8. D.	32	3	Pendulous belly.
9. E.	44	7 in 9 years	Right inguinal hernia.
10. H.	34	4	Extremely pendulous belly.
11. M.	28	4	Pendulous belly; prolapsus of the uterus.
12. T.	32	6 in 7½ years	Pendulous belly; descent of the uterus.
13. St.	44	11 in 12 years	Pendulous belly.
14. C.	24	0	Acute injury.
15. S.	47	0	Ovariotomy.
16. R.	30	8 in 7 years	Retroflexion of the uterus; jaundice.
17. J.	35	6 in 12 years	Pregnancy; œdema of right leg.
18. W.	32	2 in 1 year	Pendulous belly; symptoms of incarceration.
19. St.	34	4 in 5 years	Pendulous belly; jaundice; gall-stones.
20. S.	30	7	Retroflexion of the uterus.
21. L.	58	8 in 14 years	Commencing cancer.
22. H.	34	8	Morphia habit.
23. H.	39	10	
24. Z.	41	5	Right femoral hernia.
25. G.	38	3	—
26. G.	34	9	
27. F.	41	2	
28. C.	38	?	
29. L.	51	?	Retroflexion of the uterus.
30. L.	49	?	—
31. L.	52	?	—
32. B.	60	?	Cancer of the womb.
33. P.	44	?	—
34. G.	40	?	—
35. R.	29	2	
36. N.	68	?	
37. R.	38	3	Descent of the uterus.
38. N.	34	2	—
39. W.	55	4	Adhesions to the lower border of the liver; symptoms of incarceration; jaundice.
40. M.	59	2	Prolapsus of the uterus; hydronephrosis.

II. *Moveable Kidney on the left side.*

No.	Age.	Number of confinements.	Other ailments.
41. S.	40	1	Pendulous belly.
42. W.	36	?	——

III. *Moveable Kidney on both sides.*

43. St.	25	1	Repeated injury.
44. Sch.	36	0	Consumption.
45. M.	60	?	Cancer of both kidneys.

Note.—In the above tables, under the head "other ailments," the author sometimes uses a dash (—), and sometimes a blank. It has been concluded that their meaning is different, and that the dash (—) is equivalent to "ditto."—TRANSLATOR.

Note.—These figures are capable of various summaries; but one is too important to omit. It will be seen that the following conditions occurred as below.

Pendulous belly . .	occurred in	11,	or about	25	per cent.
"Retroflection" of the uterus	„	7	„	15	„
Descent of uterus or vagina	„	6	„	13	„
Hernia	„	3	„	7	„

Now, "retroflexion" is the only term here used for backward displacements, and no note is taken whether it was accompanied or not by some descent. It becomes increasingly probable that backward displacements are indications of descent, which is therefore the main condition. If it were true that these 7 cases of "retroflexion" were of this nature, we should have the remarkable result that descent of the uterus or vagina, with or without backward displacement, occurred thirteen times in 45 cases. or in nearly 30 per cent.

Family history does not find place in this memoir, but in any subsequent investigation inquiry should be made for rupture, prolapsus ani, descent of the uterus, &c., varicose veins, and other "hernial" conditions. Though not always found, family histories are sometimes unexpectedly comprehensive, as the following family history of a woman who came to me at St. George's Hospital a few days ago will show:

Emma R—, æt. 52, laundress, complains of womb coming

down outside for twenty-five years. (It is in fact procident.) Father had a rupture, mother a procidentia. Patient's eldest daughter (æt. 30) has a procidentia ever since æt. 21. All patient's sons (three) had prolapsus ani as children.

Similar histories could no doubt be multiplied by observers with such material as lies at the disposal, for instance, of the Truss Society.—Translator.

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[1] Ancient authors include the condition which we now call Hydronephrosis (*Rayer*) under the name Ischuria. Even *Galen* must have known this condition, as appears from a passage in lib. 3, de symptom. caus., p. 246: "Cum urina omnino in vesicam non venit, vocat etiam vulgus medicorum *ischuriam*, quamquam ischuria non sit, et permittendi sunt profecto ita nominare, cum propriam appellationem non habeant."

[The original is as follows: (Ed. Chart., vii [98]. Ed. Bas., iii [244]. κεφ' ή [98].) "καλοῦσι δὲ οὐδὲν ἧττον οἱ πολλοὶ τῶν ἰατρῶν ἰσχουρίαν κἀκεῖνο τὸ σύμπτωμα, καίτοιγε οὐκ ἦν ἰσχουρία, ἐπειδὴ μηδόλως εἰς τὴν κύστιν ἀφικνεῖται τὸ οὖρον, ἀπολλυμένης τῶν νεφρῶν τῆς ἐνεργείας. καὶ συγχωρητέον γε αὐτοῖς ὀνομάζειν οὕτως ἀποροῦσι προσηγορίας οἰκείας."—TRANSLATOR.]

(165) l. c., S. 550.
(166) *Lauenstein*, Archiv für Chirurgie, Bd. xxvi.
(167) l. c., No. 18.
(168) s. *Jenner*, British Medical Journal, Jan. 2, 18; Feb. 6, 13; March 6, 1869; and *Freund*, Hufeisen-Niere, Beiträge zur Geburtshülfe u. Gynäkologie, iv, 2.
(169) l. c., S. 759,
(170) l. c., S. 176.
(171) l. c., S. 757.
(172) l. c., S. 50.
(173) s. *le Ray*, S. 45.
(174) *Spiegelberg*, Die Diagnose der Eierstockstumoren, besonders der Cysten, in *Volkmann's* Sammlung Klin. Vortr., Bd. ii, S. 445.
(175) *Spencer Wells*, Lancet, 1865, und Eierstocksgeschwülste (? "Diseases of the Ovaries").
(176) *Landau*, Ueber den Werth der Rectaluntersuchung mit der vollen Hand., Arch. für. Gyn., Bd. vii, S. 541, 1875.
(177) l. c., S. 217.
(178) l. c., S. 120.
(179) l. c., S. 527.
(180) l. c., S. 521.
(181) l. c., S. 763.
(182) l. c., S. 28.
(183) s. Deutsche medicinische Wochenschr., 1881, 4 Juni; Anm. Referat über den Chirurgen Congress, 1881.—In the meantime *Hahn* has described (in the Centralblatt für Chirurgie, Juli, 1881) two cases treated by him by the operation of sewing. The above considerations, however, are not in the least weakened by *Hahn's* statement that one case was cured, since the period during which they were observed is far too short. Moreover (with a view to estimate such isolated cases of alleged cure properly) it should be remembered that *King* saw improvement follow an *abortive attempt* to remove a moveable kidney.

[The name "*Nephrorrhaphy*" has been lately given to this operation. For an account of its first performance in England by Dr. David Newman, see Lancet, April 28, 1883, p. 749. Relief is said to have resulted.—TRANSLATOR.]

(184) *Buch*, Ueber die Behandlung des Ileus mit Massage, Berl. klin. Woch., 1880, No. 41, S. 584.
(185) l. c., S. 522.
(186) Ibid. and *Barker*, Med.-Chir. Transact., lxiii, 1880, p. 588.
(187) *Schmidt's* Jahrb., 1881, i Heft.
(188) *Willis*, Die Krankheiten des Harnsystems und ihre Behandlung. Uebersetzt von *Heusinger*, Eisenach, 1841, S. 462 ff.
(189) *Virchow's* Archiv, Bd. xxii, 1878; *Falk*, ibid., 1881, Bd. lxxx.
(190) l. c., S. 31.
(191) *Kroner*, Rechtsseitige Hydronephrose, &c., Archiv für Gynäkol., Bd. xvii, S. 102, 1881.

(192) *Barker*, Nephrectomy by Abdominal Section, Med.-Chir. Transact., vol. lxiii, S. 182 ff.

(193) *Czerny*, Archiv für klin. Chir., Bd. xxv, Heft 4, und xxvi, Heft 4.

(194) *Landau*, Ueber Melaena Neugeborener, nebst Bemerkungen über die Obliteration der fötalen Wege, Breslau, 1874.

(195) *Landau*, Zur operation der Echinococcen in der Bauchhöhle, Berl. klin. Woch., 1885, Nos. 7 and 8.

(196) *Landau*, Zur Lehre der Eierstocks-schwangerschaft, Archiv für Gynäk., 1880, Bd. xvi, S. 436.

[See also D. Newman, Glasgow Med. Jour., Aug., 1883.—TRANSLATOR.]

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REPORT

PRESENTED TO THE

TWENTY-FIFTH ANNUAL MEETING

OF THE

NEW SYDENHAM SOCIETY

HELD AT LIVERPOOL,

AUGUST 8TH, 1883.

WITH

Classified List of Published Works

AND OTHER INFORMATION.

LONDON:

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1883.

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Those marked with an Asterisk were not in office last year.

REPORT

PRESENTED TO THE TWENTY-FIFTH ANNUAL MEETING OF THE NEW SYDENHAM SOCIETY.

IN presenting their Report for the past year the Council has little to state beyond the record of the works published and the announcement of those in preparation. The production of the Lexicon of Medical Terms has been continued with as much speed as circumstances have permitted, and two numbers have as usual been issued during the year.

The issue for the current year will probably consist of—

A Fasciculus of the Atlas of Pathology.

Selections from the Works of Duchenne (of Boulogne), edited by Dr. Vivian Poore.

Two or more Parts of the Lexicon of Medical Terms.

The first Volume of Hirsch's work on Historico-Geographical Pathology.

A Volume of Selected Monographs, of which Senator on Albuminuria and Landau on Movable Kidney will form part.

The works which have been issued during the past year are the following:—

Dr. Stokes on Diseases of the Chest, with a Portrait of the Author, and a Memoir by Dr. Acland.

The Collected Writings of the late Dr. Warburton Begbie, edited by Dr. Dyce Duckworth, with a Memoir and Portrait.

A Translation, by Dr. Hadden, of Charcot's Treatise on the Localisation of Cerebral and Spinal Disease.

The Sixteenth Fasciculus of the Society's Atlas of Skin Diseases.

The Seventh and Eighth Parts of the Society's Lexicon of Medical Terms.

Amongst those which are in preparation are the following:—

A Fasciculus of the Atlas of Portraits of Skin Diseases.

The Collected Works of Dr. Peacock, with Memoir and Portrait.

One or more Volumes of Selected Clinical Lectures, from German sources.

The Society's accounts for the year have been audited as usual, and a balance sheet prepared.

Receipts.

Balance in hand, Dec. 1881 (see preceding Balance Sheet)				1074	6	8½
Subscriptions, 1 for 1859			1 1 0			
,, 1 ,, 1860			1 1 0			
,, 2 ,, 1861			2 2 0			
,, 1 ,, 1862			1 1 0			
,, 1 ,, 1863			1 1 0			
,, 1 ,, 1864			1 1 0			
,, 1 ,, 1865			1 1 0			
,, 2 ,, 1866			2 2 0			
,, 3 ,, 1867			3 3 0			
,, 3 ,, 1868			3 3 0			
,, 3 ,, 1869			3 3 0			
,, 3 ,, 1870			3 3 0			
,, 3 ,, 1871			3 3 0			
,, 4 ,, 1872			4 4 0			
,, 3 ,, 1873			3 3 0			
,, 3 ,, 1874			3 3 0			
,, 4 ,, 1875			4 4 0			
,, 35 ,, 1876			36 15 0			
,, 39 ,, 1877			40 19 0			
,, 34 ,, 1878			35 14 0			
,, 62 ,, 1879			65 2 0			
,, 144 ,, 1880			151 4 0			
,, 574 ,, 1881			602 14 0			
,, 1409 ,, 1882			1479 9 0			
,, 22 ,, 1883			23 2 0			
Volumes of Trousseau			116 4 0			
Fasciculi of Atlases			19 10 0			
Volumes of Hebra			30 0 6			
Other volumes			183 13 0			
			2825 5 6			
Less deductions by Local Secretaries			11 11 0			
				2813	14	6
				£3888	1	2½

Expenditure.

Folio I. Artists, editors, and translators			74	4	0
,, II. Printers			2010	4	7
,, III. Paper			366	7	0
,, IV. Bookbinders			145	18	6
,, V. Expenses of Management:—					
Secretary's Expenses	53 11 0				
Treasurer's Expenses	5 12 6				
Fire Insurance	12 0 0				
Agent's Salary and Percentage	277 3 5				
Disbursements, &c. (chiefly carriage)	139 15 7				
Advertisements	97 11 0				
			585	13	6
Sum Total of Expenditure			3182	7	7
Balance in hand, December 31st, 1882			705	13	7½
			£3888	1	2½

Examined, compared with the vouchers, and found correct, the balance on 31st Dec. 1882, being £705 13*s.* 7½*d.*, at an audit held this 1st day of August, 1883.

EDWARD CLAPTON.
SAMUEL FENWICK. } *Auditors*
F. M. CORNER.

CLASSIFIED LIST

OF THE

SOCIETY'S PUBLICATIONS.

Medicine.

ON THE TEMPERATURE IN DISEASE: A MANUAL OF MEDICAL THERMOMETRY. By Dr. C. A. Wunderlich. (Leipzig). Translated by Dr. Bathurst Woodman. With forty Woodcuts and seven Lithographs.

"It is well to recollect that this contains not only observations on the temperature in disease, but also in health, and is a complete epitome as to the history of the subject up to date. It is a work of reference absolutely necessary for all who would keep themselves abreast of the day in relation to so important a matter as corporeal temperature."—*Edin. Med. Journ.*, May, 1872.

"In short, without pledging ourselves to Wunderlich as infallible, we may say, emphatically, that his is a masterwork, in which every part of his subject is considered with that thoroughness which comes of ripe knowledge and reflection. Let us add that Dr. Bathurst Woodman, following one or two laudable examples that have been set by other translators for the Sydenham Society, has enriched the work with notes of his own observations and those of other English writers, which are of no small value, and unquestionably do much to make the volume complete and full."—*Lancet*, April 20, 1872.

"The translator has rendered into readable English, and enriched with practical notes, a book which, even in its original form, has started into active work many physicians in England, France, and America, and which now, in its popular form, must render the diagnosis of disease infinitely more accurate."—*Medical Times and Gazette*, June 3, 1871.

"The publication of this volume marks an epoch in the history of medical thermometry. The very possibility of such a book—full not only of exact knowledge, but of important generalisations—is an indication that the great problems relating to the alterations in the human temperature—the problems of fever and collapse—are now being studied in a manner calculated to throw light on the hidden processes of disease. . . . . The value of this great work of Professor Wunderlich is that it lays open his vast clinical experience of the thermometer, and that it sketches in general terms the course of the temperature in various forms of disease."—*Glasgow Medical Journal*, August, 1871.

"This treatise displays so much perseverance and thoroughness, such admirable caution and insight, and such wide and minute learning, that it may be said not only to establish this branch of investigation for the first time upon a deep and lasting basis, but also to build up a very great part of the edifice, and to point out with clearness the directions in which future labour must be applied."—Dr. Allbutt in *Brit. and For. Med. Chir. Rev.*, April, 1870.

LECTURES ON CLINICAL MEDICINE, delivered at the Hotel Dieu, Paris. By Professor Trousseau. Five Volumes. Vol. 1, translated, with notes and appendices, by the late

Dr. Bazire. Vols. 2 to 5, translated from the third edition, revised and enlarged, by Sir John Rose Cormack.

"We are indebted to the New Sydenham Society for this rich contribution to our medical literature. . . . . Trousseau is an author to be read rather than reviewed. He can only be criticised worthily at the bedside. . . . . We commend this great physician's work to the study of every reader."—*Lancet*, October 15, 1870.

"The above-mentioned works constitute the nineteenth annual issue to its subscribers of the New Sydenham Society; and, though relating to different subjects, we have classed them together, because it seems of more importance to the profession that they should know the very valuable practical information they can secure for one guinea, than at this time of day they should be treated to an elaborate critique on Trousseau's Clinical Medicine, or on Wunderlich's Treatise on Thermometry; the worth of these volumes being well known to all but the merest tyro in medicine."—*Edinburgh Medical Journal*, May, 1872.

"We should think any medical library absurdly incomplete now which did not have, alongside of Watson and Graves and Tanner, the clinical medicine of Trousseau. The work is full of the results of the richest natural observation, and is the production of one who was enlightened enough to combine with new methods of investigation the vigorous and independent ideas of the old physicians, whom he so eloquently magnifies. The volume is an extremely rich and valuable addition to the library of physicians and practitioners generally."—*Lancet*, December 4, 1869.

LATHAM'S COLLECTED WORKS. 2 vols. Edited by Dr. Robert Martin. With Memoir of Latham by Sir Thomas Watson.

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FIVE FASCICULI have been published, and it is proposed to issue one every year.

The following subjects have been illustrated :—

FIRST FASCICULUS.

Scrofula; Syphilis; and Lymph-Adenoma.—Plate I.

Fig. 1. Scrofulous Disease of the Kidney and Ureter. Fig. 2. Scrofulous Disease of the Kidney. Fig. 3. Scrofulous Disease of the Kidney. Fig. 4. A Mass of Syphilitic Deposit in the Cortical Substance of the Kidney. Fig. 5. Lymph-Adenoma of Kidney.

Nephritis after Diphtheria; Scarlet Fever; and Burns.—Plate II.

Fig. 1. Nephritis after Diphtheria.—Section of Kidney. Fig. 2. Subacute Nephritis after Scarlet Fever.—Outer surface of kidney. Fig. 3. Subacute Nephritis after Scarlet Fever. Fig. 4. Acute Nephritis after Scarlet Fever. Fig. 5. Subacute Nephritis after Scarlet Fever. Fig. 6. Acute Nephritis after a Burn.—Outer surface of the kidney of a child who died after a very extensive burn. Fig. 7. Acute Nephritis after a Burn.—Section of the same kidney.

The Granular Kidney in different stages.—Plate III.

Fig. 1. Extremely Granular Kidney. Fig. 2. Extremely Granular Kidney.—Section of the same kidney. Fig. 3. Less Granular (contracted) Kidney.—Outer surface of the right kidney taken from the same subject as the left kidney shown in Figs. 1 and 2. Fig. 4. Granular Kidney of Bright. Fig. 5. Contracted Granular Kidney, in section. Fig. 6. Contracted Granular Kidney; exterior. Fig. 7. Large Granular Kidney. Fig. 8. Large Granular Kidney with cysts.

Embolism; Infarction Processes from Pyæmia; Jaundice and Purpura; Scrofula.—Plate IV.

Fig. 1. Embolic Changes in Pyæmia. Fig. 2. Embolic Changes in Pyæmia. Fig. 3. Pyæmic Deposits in Kidney. Fig. 4. Pyæmic Deposits in the Kidney. Fig. 5. Results of Jaundice and Purpura. Fig. 6. A variety of the Scrofulous Kidney.—The substance of the kidney is wholly destroyed and replaced by cavities containing a white mortar-like substance.

SECOND FASCICULUS.

Diseases of the Kidney.—Plate V.

Fig. 1. Amyloid Disease of Kidney in advanced stage. Fig. 2. A section of the same Kidney. Fig. 3. The pale flabby Kidney. Fig. 4. The same organ seen in section. Fig. 5. Medullary Cancer of the Kidney.

Various Diseased Conditions of the Spleen.—Plate VI.

Fig. 1. Hodgkin's Disease of Spleen (Lympho-sarcoma). Fig. 2. Acute Splenic enlargement in Diphtheria. Fig. 3. Suppurating infarction of Spleen from a case of Ulcerative Endocarditis. Fig. 4. Embolic changes in Pyæmia. Fig. 5. Rupture of the Spleen.

Diseases of the Supra Renal Capsules and Spleen.—Plate VII.

Fig. 1. Cancer of the Supra Renal Capsule. Figs. 2, 3, 4. Adenoma of the Supra Renal Capsule. Fig. 6. Addison's Disease of the Supra Renal Capsule (in section). Fig. 5. Addison's Disease of the Supra Renal Capsule.—"Fibro-calcareous or strumous disease." Fig. 7. Tubercle of the Spleen (external surface). Fig. 8. Tubercle of the Spleen (in section). Fig. 9. Lardaceous Spleen.

Microscopic Pathology of Kidneys.—Plate VIII.

Fig. 1. Lardaceous Degeneration of the Kidney.—Section of cortex. Fig. 2. Lardaceous Degeneration.—*g*. A glomerulus from the same kidney, as in Fig. 1, which has undergone lardaceous degeneration and is becoming fatty. Fig. 3. Part of the same seen with a higher power, showing contents of one of the tubules. Fig. 4. Lardaceous Degeneration in earlier stage combined with interstitial fibrous change. Figs. 5 & 6. Lardaceous Degeneration (after Cornil). Fig. 5. Section showing the hyaline membranous wall of the tubules *a a* much swollen, stained violet-red, showing waxy degeneration. Fig. 6. Transverse section of one of the pyramids, near summit of cone. Fig. 7. Granular Contracted Kidney. Fig. 8. From the same.—A thickened arteriole surrounded by fibroid growth. Fig. 9. Partial Fibrous Degeneration of Malpighian body in slight chronic intertubular nephritis. Fig. 10. From the same kidney; showing early changes around Malpighian body. Fig. 11. Multiplication of Nuclei on glomerulus with adhesion of capillary tuft to wall of capsule. Fig. 12. Subacute Interstitial Nephritis with large white kidney. Fig. 13. Scarlatinal Nephritis.—Intertubular exudation in a case fatal on 7th day of fever. Fig. 14. Subacute Interstitial Nephritis. Fig. 15. Acute Catarrhal Nephritis, showing swelling and granular degeneration of epithelium. (100 diam.) Fig. 16. Part of the same seen with a higher power. Fig. 17. Section of cortex from a case of parenchymatous (catarrhal) nephritis at a later stage (so-called "fatty" kidney). Fig. 18. From nearly transverse section near base of pyramid in similar case. Fig. 19. Casts in tubes in interstitial nephritis (post scarlatinal). Fig. 20. Colloid cast, *b*, in tubule; *a*, unaltered epithelium.

Microscopic Pathology of the Kidney.—Plate IX.

Fig. 1. Scarlatinal Nephritis. Fig. 2. Shows two of the glomeruli from same section as Fig. 1. Fig. 3. Section from the same.—Part of the wall of a Malpighian body from which the capillary tuft has fallen out. Fig. 4. Scarlatinal Nephritis.—(From a case fatal about 12 weeks from attack of fever). Fig. 5. Scarlatinal Nephritis.—(From a case fatal 15 months after attack of scarlet fever). Fig. 6. From same kidney as Fig. 5, but in a deeper part of cortex, close to medulla. Similar growth of interstitial connective tissue. Fig. 7. Subacute Interstitial Nephritis, probably Scarlatinal, under low power; showing diffuse infiltration and cluster of dilated tubules. Fig. 8. Chronic Parenchymatous Nephritis (large white kidney) with little or no interstitial change.—Section of cortex, showing changes in epithelium of convoluted tubules. Fig. 9. Kidney in leucocythæmia—to show localisation of changes around glomeruli and vessels. Fig. 10. Swelling of inner coat of small artery in granular contracted kidney. Fig. 11. Tuberculous Pyelonephritis. Fig. 12. Fatty Degeneration from Alcoholic Poisoning (after Lancereaux). Fig. 13. Fatty Degeneration in Cancer. Fig. 14. Individual epithelial cells from the preceding section; in various stages of fatty degeneration. Fig. 15. Cystic Degeneration of Kidney (after Lancereaux.) Fig. 16. From a cyst in kidney near base of pyramid. Fig. 17. Colloid Degeneration of Kidney. Figs. 18, 19, 20, and 21, illustrate the hyaline changes found in the splenic arteries in certain febrile conditions. Fig. 18. From a section through the spleen of a case of early scarlatina, showing hyaline degeneration of the coat of an artery, transversely cut. Fig. 19. Artery in longitudinal section. Fig. 20. Malpighian corpuscle from the spleen of a case of early scarlatina. Fig. 21. Part of the central and intermediate zone of the same Malpighian corpuscle as in Fig. 20, only more highly magnified (180 diam.) Fig. 22. Hodgkin's Disease. — Section of a spleen to show the overgrowth of the lymphatic sheath in Hodgkin's disease. (1 inch.) Fig. 23. Adenoma of the Supra Renal Capsule, showing the columns stuffed with fatty granules.

Microscopic Pathology of Spleen and SupraR enals.—Plate X.

Fig. 1. Capsulitis of the Spleen.—Vertical section of fibrous nodule in the capsule of the spleen, showing that the thickening of the capsule takes place by cellular growth in its deeper layers. Fig. 2. Fibrosis of the Spleen.—From the enlarged spleen of a ricketty child. Fig. 3. Fibrosis of the Spleen.—Showing a more advanced or fibrous condition spreading round some dilated veins. Fig. 4. Muscular Hypertrophy.—Over-growth of muscular trabeculæ in the spleen. Fig. 5. Muscular Hypertrophy. — Extreme stage of fibro-muscular growth in the spleen. Fig. 6. The Leucocythæmic Spleen.—Section of the edge of a Malpighian corpuscle, showing the compressed fibrous tissue between it and the splenic pulp. Fig. 7. The Leucocythæmic Spleen. — The pulp and stroma are normal. Fig. 8. Hodgkin's Disease.—The texture of a lymphoid nodule in the spleen of Hodgkin's disease. Fig. 9. Tubercular Spleen. (37 diam.) Fig. 10. Tubercular Spleen. Fig. 11. Induration and Atrophy. — A section of the spleen from a case of heart disease. Fig. 12. Lardaceous Spleen.—The sago spleen, showing the Malpighian corpuscles and small arteries mapped out by structureless hyaline lardaceous matter. Fig. 13. Lardaceous Spleen.—Transverse section of a Malpighian corpuscle, or small artery, with its surrounding lymphoid sheath. Fig. 14. Addison's Disease.—Vertical section of a supra renal capsule from the exterior inwards, to show the early changes in Morbus Addisonii. (250 diam.) Fig. 15. Addison's Disease.—Section of a supra renal capsule, to show the late, or fibro-calcareous, stage of Morbus Addisonii.

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THIRD FASCICULUS.

Diseases of the Liver.—Plate XI.

Lymphadenoma of Liver.

Diseases of the Liver.—Plate XII.

Fig. 1. Dilatation of the Bile Ducts in the Liver from pressure of a gall stone in cystic duct.

Fig. 2. Cancer of the Liver, with dilatation of the ducts and staining of the hepatic tissue.

Diseases of the Liver.—Plate XIII.

Syphilitic Cirrhosis of the Liver.

Diseases of the Liver.—Plate XIV.

Fig. 1. Red Atrophy, with acute Yellow Atrophy of the Liver.

Fig. 2. Microscopical appearances of the yellow swollen parts of the Liver (Acute Yellow Atrophy).

Fig. 3. Microscopical appearances of Red Atrophy of the Liver.

Diseases of the Liver.—Plate XV.

Fig. 1. Lardaceous Liver.
Fig. 2. Lardaceous Liver, showing the iodine reaction.

Diseases of the Liver.—Plate XVI.

Fig. 1. Cancer of the Liver.
Fig. 2. Nutmeg Liver, Chronic Congestion, and Atrophy of the Liver from mitral disease.

FOURTH FASCICULUS.

Diseases of the Liver, including one Figure of Spleen.—Plates XVII. to XXII.

Diseases of the Liver and Spleen.—Plate XVII.

Fig. 1. Cirrhosis of the Liver resembling the Nutmeg Liver.
Fig. 2. Brown Atrophy of the Liver.
Fig. 3. Cirrhosis of the Liver.
Fig. 4. Lymphadenoma of the Spleen (Hodgkin's Disease).

Diseases of the Liver.—Plate XVIII.

Fig. 1. Fatty Liver from Poisoning by Phosphorus.
Fig. 2. Cirrhosis of the Liver.
Fig. 3. Tubercular Liver.
Fig. 4. Cirrhosis of the Liver.

Diseases of the Liver.—Plate XIX.

Cystic Disease of the Liver.

Diseases of the Liver.—Plate XX.

Fig. 1. Lardaceous Disease of the Liver. Fig. 2. Fatty Liver. Fig. 3. Early Cirrhosis. Figs. 4 & 5. Cirrhosis of the Liver (after Hamilton). Fig. 6. Cirrhosis of the Liver. Fig. 7. A Vegetation from the surface of the Liver. Fig. 8. Spindle-cell Sarcoma of the Liver. Fig. 9. Disseminated Growths of Fibrous Nature in the Liver. Fig. 10. Lardaceous Disease of the Liver. Fig. 11. Cavernous Tumour in the Liver. Fig. 12. Acute Yellow Atrophy of the Liver. Fig. 13. Cavernous Tumour in the Liver. Fig. 14. Early Cirrhosis. Fig. 15. Columnar Epithelioma of the Liver.

Diseases of the Liver.—Plate XXI.

Fig. 1. Cirrhosis of the Liver. Fig. 2. Cirrhosis of the Liver. Fig. 3. Monolobular Cirrhosis. Fig. 4. The Nutmeg Liver (Romose Atrophy of Moxon). Fig. 5. Tubercular Liver. Fig. 6. The Nutmeg Liver. Fig. 7. Miliary Gummata. Fig. 8. Idiopathic Anæmia. Figs. 9 & 10. Cancer of the Bile Ducts. Fig. 11. Cancer spreading from the Biliary Ducts. Fig. 12. Early Gummatous Infiltration of the Liver. Fig. 13. "Common" Cirrhosis. Fig. 14. Tubercular Liver. Fig. 15. Idiopathic Anæmia.

Diseases of the Liver.—Plate XXII.

Fig. 1. "Pericellular" Cirrhosis. Fig. 2. Cirrhosis of the Liver. Fig. 3. Nutmeg Liver. Fig. 4. Cystic Liver. Fig. 5. Cystic Liver. Fig. 6. Early Cancer of the Liver. Fig. 7. Extreme Tubercular Disease of the Liver. Fig. 8. Brown Atrophy of the Liver. Fig. 9. Extreme Tubercular Disease. Fig. 10. Myxœdematous Liver. Figs. 11, 12 & 13. "Contracting Scirrhus of the Liver simulating Cirrhosis." Figs. 14, 15 & 16. Varieties of Cell Vacuolation and Proliferation. Fig. 17. Primary Adenoma of the Liver. Fig. 18. Leukæmic Liver. Fig. 19. Primary Adenoma of the Liver.

FIFTH FASCICULUS.

Diseases of the Liver (chiefly of the Gall-Bladder and Larger Bile Ducts).—Plate XXIII.

Syphilitic and Lardaceous Disease of the Liver.

Diseases of the Liver.—Plate XXIV.

Fig. 1. Abscesses in the Liver.

Fig. 2. Papilloma of the Gall-Bladder.

Diseases of the Liver.—Plate XXV.

Cancer of Gall-Bladder and Liver.

Gall-stones, with Obstruction and Dilatation of the Cystic Duct.

Diseases of the Liver.—Plate XXVI.

Cancer of the Stomach extending to the Cystic Duct.

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Diseases of the Eye and Ear.

ON THE ANOMALIES OF ACCOMMODATION AND REFRACTION OF THE EYE, with a PRELIMINARY ESSAY ON PHYSIOLOGICAL DIOPTRICS. By F. C. Donders, M.D., Professor of Physiology and Ophthalmology in the University of Utrecht. Written expressly for the Society. Translated from the Author's Manuscript by W. D. Moore, M.D.

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Diseases of the Nervous System.

SCHRŒDER VAN DER KOLK ON A CASE OF ATROPHY OF THE LEFT HEMISPHERE OF THE BRAIN. Translated by Dr. W. Moore, of Dublin. Four Lithographs.

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A MANUAL OF MENTAL PATHOLOGY AND THERAPEUTICS. By Professor Griesinger. Translated by Dr. Lockhart Robertson and Dr. James Rutherford.

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CHARCOT'S TREATISE ON THE LOCALISATION OF CEREBRAL AND SPINAL DISEASE. Translated by Dr. HADDEN.

"It will give to its reader a clear understanding of what is known of the subject it professes to treat of."—*Edinburgh Medical Journal*, Dec., 1883.

"Dr. Hadden is to be congratulated upon having produced a translation of these valuable Lectures, which, whilst faithful to the text, is not marred by being too literal."—*Medical Times and Gazette*, October 27, 1883.

Anatomy, Physiology, and General Pathology.

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EXPERIMENTAL RESEARCHES ON THE EFFECTS OF LOSS OF BLOOD IN PRODUCING CONVULSIONS, By Drs. KUSSMAUL and TENNER. Translated by Dr. BRONNER, of Bradford.

A MANUAL OF PATHOLOGICAL HISTOLOGY, intended to serve as an introduction to the study of Morbid Anatomy. By Professor RINDFLEISCH. (Bonn.) 2 vols. Translated by Dr. BAXTER.

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A LEXICON OF MEDICAL TERMS. Edited by Mr. Power and Dr. Sedgwick. Parts 1 to 8. This Lexicon is based upon the well-known work of Dr. Mayne, the copyright of which was purchased by the Society. It is, however, entirely rewritten by the present Editors, and very much enlarged.

"The work is carefully and elaborately done, and comprehends every reference which the medical or scientific inquirer could possibly require."—*Medical Press and Circular*, June 22nd, 1881.

Diseases of the Skin and Syphilis.

ON SYPHILIS IN INFANTS. By Paul Diday. Translated by Dr. Whitley.

"The work of M. Diday is of great merit; it contains all that has been written on infantile syphilis, and he puts the whole subject in a well-arranged form for further investigation as well as present use."—*Brit. and For. Med. Chir. Rev.*

ON DISEASES OF THE SKIN, INCLUDING THE EXANTHEMATA. By Professor Hebra. 5 vols. Translated and Edited by Dr. Hilton Fagge, Dr. Pye-Smith, and Mr. Waren Tay.

"Had we space we should have been glad to enter into a lengthened critique of the second volume of Hebra's work. We are relieved from any misgiving, however, by the fact that the work will be very largely circulated amongst our readers by the Sydenham Society, and that they, with others who

aspire to any real knowledge of skin diseases, would not, under any circumstances, be satisfied without studying the work for themselves. . . . . This second volume contains information relative to the most important diseases of the skin; and it will, we are confident, do good service in helping on the cause of cutaneous medicine in England."—*Lancet*, November 7, 1868.

"Of all the works produced by the New Sydenham Society this is one of the most valuable and most welcome. . . . . It is to be remarked that this book is not a mere translation of the German work; it is a new and revised edition, undertaken by the author for his English brethren."—*Medical Times and Gazette*, April 27, 1867.

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Vol. 4.—"The entire work is admirable for its lucidity of arrangement, its simplification of confused and intricate subjects, and not least for the avoidance of those pedantic and repelling terms which a celebrated dermatologist has grandiloquently styled the 'terminological innovations of modern nomenclators.'"—*Dublin Journal of Medical Science*, May, 1875.

LANCEREAUX'S TREATISE ON SYPHILIS. 2 vols. Translated by Dr. Whitley.

"The work is the most exhaustive book which has been published on the subject, and has been quoted by all the recent writers in this country, America, and the Continent. It is a perfect mine of information. The translation is well done, and the New Syd. Soc. may be congratulated on having added such an important treatise to its list of works."—*Lancet*, March, 1869.

The Society's Atlas of Diseases of the Skin.

In sixteen Fasciculi comprising the following subjects. Unless otherwise indicated, the Plates are original.

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"This Fasciculus supplies life size portraits of pityriasis rubra, papular syphilis, with indurated chancres, and pruriginous impetigo following varicella, which are extremely beautiful, and look life-like."—*Edin. Medical Journal*, May, 1872.

"They are better, to our mind, than any other plates in use amongst us; and there cannot be a question as to the Society's issue being as popular as it is useful."—*Lancet.*

"We have received the thirteenth fasciculus of this splendid collection of drawings, of which no further praise is needed than to say that they are executed with the same artistic skill and fidelity to nature which have characterised the whole series."—*Dublin Journal of Medical Science*, May, 1874.

A CATALOGUE OF THE PORTRAITS COMPRISED IN THE SOCIETY'S ATLAS OF SKIN DISEASES. Prepared, at the request of the Council, by Mr. Hutchinson. Parts 1 and 2.

"The descriptions, cases, and plates are well given. . . . . There is one good feature in some of the cases described. Take that of Addison's Keloid, p. 160. In it we have notes, &c., of a rare skin disease, which has been accurately described by the observers under whose care the patient had been at various stages of the case. This is, therefore, a valuable contribution to medicine."—*Edinburgh Medical Journal*, February, 1877.

LIST OF PUBLISHED WORKS

Arranged according to the Year of Issue.

Vol. 1859. (*First Year.*)

1. Diday on Infantile Syphilis.
2. Gooch on Diseases of Women.
3. Memoirs on Diphtheria.
4. Van der Kolk on the Spinal Cord, &c.
5. Monographs (Kussmaul and Tenner, Graefe, Wagner, &c.)

1860. (*Second Year.*)

6. Dr. Bright on Abdominal Tumours.
7. Frerichs on Diseases of the Liver. Vol. I.
8. A Yearbook for 1859.
9. Atlas of Portraits of Skin Diseases. (1st Fasciculus.)

1861. (*Third Year.*)

10. A Yearbook for 1860.
11. Monographs (Czermak, Dusch, Radicke, &c.)
12. Casper's Forensic Medicine. Vol. I.
14. Atlas of Portraits of Skin Diseases. (2nd Fasciculus.)

1862. (*Fourth Year.*)

13. Frerichs on Diseases of the Liver. Vol. II.
15. A Yearbook for 1861.
16. Casper's Forensic Medicine. Vol. II.
17. Atlas of Portraits of Skin Diseases (3rd Fasciculus.)

1863. (*Fifth Year.*)

18. Kramer on Diseases of the Ear.
19. A Yearbook for 1862.
20. Neubauer and Vogel on the Urine.

VOL. 1864. (*Sixth Year.*)

21. CASPER'S Forensic Medicine. Vol. III.
22. DONDERS on the Accommodation and Refraction of the Eye.
23. A YEARBOOK for 1863.
24. ATLAS of Portraits of Skin Diseases. (4th Fasciculus).

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25. A YEARBOOK for 1864.
26. CASPER'S Forensic Medicine. Vol. IV.
27. ATLAS of Portraits of Skin Diseases. (5th Fasciculus).

1866. (*Eighth Year.*)

28. BERNUTZ and GOUPIL on the Diseases of Women. Vol. I.
29. ATLAS of Portraits of Skin Diseases. (6th Fasciculus.)
30. HEBRA on Diseases of the Skin. Vol. I.
31. BERNUTZ and GOUPIL on Diseases of Women. Vol. II.

1867. (*Ninth Year.*)

32. BIENNIAL Retrospect of Medicine and Surgery.
33. GRIESINGER on Mental Pathology and Therapeutics.
34. ATLAS of Portraits of Skin Diseases. (7th Fasciculus).
35. TROUSSEAU'S Clinical Medicine. Vol. I.

1868. (*Tenth Year.*)

36. THE Collected Works of Dr. Addison.
37. HEBRA on Skin Diseases. Vol. II.
38. LANCEREAUX'S Treatise on Syphilis. Vol. I.
39. ATLAS of Portraits of Skin Diseases. (8th Fasciculus).
40. CATALOGUE of Atlas of Skin Diseases. (First Part.)

1869. (*Eleventh Year.*)

41. LANCEREAUX'S Treatise on Syphilis. Vol. II.
42. TROUSSEAU'S Clinical Medicine. Vol. II.
43. BIENNIAL Retrospect of Medicine and Surgery.
44. ATLAS of Portraits of Skin Diseases. (9th Fasciculus.)

1870. (*Twelfth Year.*)

45. TROUSSEAU'S Lectures on Clinical Medicine. Vol. III.
46. NIEMEYER'S Lectures on Pulmonary Consumption.
47. STRICKER'S Manual of Histology. Vol. I.
48. ATLAS of Portraits of Skin Diseases. (10th Fasciculus).

VOL. 1871. (*Thirteenth Year.*)

49. WUNDERLICH'S Medical Thermometry.
50. BIENNIAL Retrospect of Medicine and Surgery.
51. TROUSSEAU'S Clinical Medicine. Vol. IV.
52. ATLAS of Portraits of Skin Diseases. (11th Fasciculus.)

1872. (*Fourteenth Year.*)

53. STRICKER'S Manual of Histology. Vol. II.
54. RINDFLEISCH'S Pathological Histology. Vol. I.
55. TROUSSEAU'S Clinical Medicine. Vol. V.
56. ATLAS of Portraits of Skin Diseases. (12th Fasciculus.)

1873. (*Fifteenth Year.*)

57. STRICKER'S Manual of Histology. Vol. III.
58. RINDFLEISCH'S Pathological Histology. Vol. II.
59. BIENNIAL Retrospect of Medicine and Surgery.
60. ATLAS of Portraits of Skin Diseases. (13th Fasciculus.)

1874. (*Sixteenth Year.*)

61. HEBRA on Skin Diseases. Vol. III.
62. Von TROELTSCH on Diseases of the Ear.
    HELMHOLTZ on Membrana Tympani, &c. (In one Vol.)
63. ATLAS of Portraits of Skin Diseases. (14th Fasciculus.)
64. HEBRA on Skin Diseases. Vol. IV.

1875. (*Seventeenth Year.*)

65. BIENNIAL Retrospect of Medicine and Surgery.
66. CATALOGUE of Atlas of Skin Diseases. (Second Part.)
67. ATLAS of Portraits of Skin Diseases. (15th Fasciculus.)
68. CLINICAL Lectures by various German Professors. Vol. I.
69. LATHAM'S Works. Vol. I.

1876. (*Eighteenth Year.*)

70. SMELLIE'S Midwifery, by McClintock. Vol. I.
71. CLINICAL Lectures by various German Professors. Vol. II.
72. CHARCOT'S Clinical Lectures on Diseases of the Nervous System. Vol. I.
73. BILLROTH'S Lectures on Surgical Pathology. Vol. I.

1877. (*Nineteenth Year.*)

74. SMELLIE'S Midwifery, by McClintock. Vol. II.
75. THE Medical Digest, by Dr. Neale.
76. BILLROTH'S Lectures on Surgical Pathology. Vol. II.
77. ATLAS of Illustrations of Pathology. (Fasciculus I.)

Vol.

1878. (*Twentieth Year.*)

78. Bibliotheca Therapeutica, by Dr. Waring. Vol. I.
79. Smellie's Midwifery, by McClintock. Vol. III.
80. Latham's Works. Vol. II.
81. Lexicon of Medical Terms. (First Part.) *Issued with Part II. only, as Vol. 83.*

1879. (*Twenty-first Year.*)

82. Bibliotheca Therapeutica, by Dr. Waring. Vol. II.
83. Lexicon of Medical Terms. (Second Part.) *Including re-issue of First Part.*
84. Manual of Physical Diagnosis, by Dr. Guttmann.
85. Atlas of Illustrations of Pathology. (Fasciculus II.)

1880. (*Twenty-second Year.*)

86. Hebra on Diseases of the Skin. Vol. V.
87. Lexicon of Medical Terms. (Third Part.)
88. Koch's Researches on Wound Infection.
89. Lexicon of Medical Terms. (Fourth Part.)
90. Charcot's Clinical Lectures on Diseases of the Nervous System. Vol. II.
91. Atlas of Illustrations of Pathology. (Fasciculus III.)

1881. (*Twenty-third Year.*)

92. Selections from the Works of Abraham Colles.
93. Lexicon of Medical Terms. (Fifth Part.)
94. Billroth's Clinical Surgery.
95. Charcot on Diseases of Old Age.
96. Lexicon of Medical Terms. (Sixth Part.)
97. Atlas of Illustrations of Pathology. (Fasciculus IV.)

1882. (*Twenty-fourth Year.*)

98. Stokes on Diseases of the Chest.
99. Atlas of Portraits of Skin Diseases. (16th Fasciculus.)
100. The Collected Works of Dr. Warburton Begbie.
101. Lexicon of Medical Terms. (Seventh Part.)
102. Charcot on Localisation of Cerebral and Spinal Disease.
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V.—Five Members of the Council shall form a quorum.

VI.—The Officers of the Society shall be elected by ballot at the General Anniversary Meeting of the Society. Balloting lists of Officers proposed by the Council, with blank places for such alterations as any Member may wish to make, shall be laid on the Society's table for the use of Members.

VII.—The President, Vice-Presidents, and Council, shall be eligible for re-election, except that of the Vice-Presidents four, and of the Council eight, shall retire every year.

VIII.—The Council shall appoint local Honorary Secretaries wherever they shall see fit.

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XIV.—The annual Accounts of the Receipts and Expenditure of the Society shall be audited by a Committee of three Members, selected at the preceding Anniversary Meeting from among the Members at large.

XV.—The Secretary shall have the management of the general Correspondence of the Society, and of such other business as may arise in carrying out its objects.

XVI.—The local Secretaries shall further the objects of the Society in their respective districts, and shall be in communication with the metropolitan Secretary.

XVII.—The Anniversary Meeting shall be held in the same town as, and at the time of, the Annual Meeting of the British Medical Association, notice of it having been given to all Members at least a week before the day fixed on.

XVIII.—The Members generally shall be invited and encouraged to propose Works, &c., and to make any suggestions to the Council they may think likely to be useful.

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XX.—No alteration in the Laws of the Society shall be made, except at a General Meeting. Notice of the alteration to be proposed must also have been laid before the Council at least a month previously.

XXI.—The Council shall have power to call a General Meeting of the Members at any time, and shall also be required to do so within three weeks, upon receiving a requisition in writing to that effect from not less than twenty Members of the Society.

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XXIII.—The Council shall meet at least once in two months, unless by special resolution to the contrary.

GENERAL INFORMATION.

A Third Edition of the Volumes for 1859 was printed, and a second of that for 1860. For subsequent years the First Edition was much larger; and it is not likely that any of the Volumes will be reprinted.

Most of the stones for plates, &c., both those for the Atlas of Skin Diseases and those for printed Volumes, have been destroyed, and will not be reproduced.

The Society is now in its Twenty-fifth year. Arrangements have been made by which new Members can obtain single Volumes, or sets of Volumes, from the Society's stock in hand. Some of the Volumes, of which a larger surplus exists than of others, can be purchased at fixed prices (for which see list). The Society's Agent is empowered to make special arrangements with new Members who may wish to obtain any of the past Volumes.

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PS.—The Society's Agent is prepared to supply, at fixed prices, CASES for binding the Lexicon, and PORTFOLIOS for the reception of the Plates of Skin Diseases, and for the Pathological Atlas.

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