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AND ITS RELATIONS TO DISEASES OF THE LIVER AND KIDNEYS.

BY

ROBSON ROOSE, M.D

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With the Author's Compts.

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AND ITS RELATIONS TO DISEASES OF THE LIVER AND KIDNEYS.

BY

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LONDON:

H. K. LEWIS, 136, GOWER STREET.

1885.

TO

SIR WILLIAM W. GULL, BART., M.D. LOND., F.R.S.,

D.C.L. Oxon., LL.D. Cantab. and Edin.,

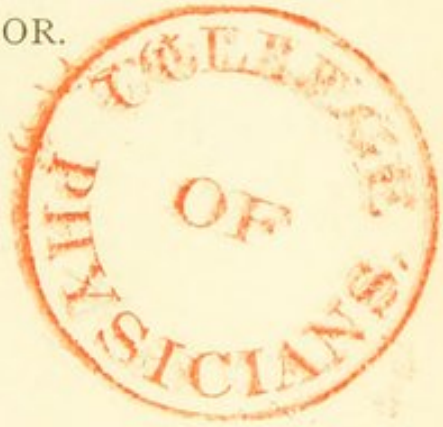
THIS WORK IS BY PERMISSION

Dedicated,

AS A TOKEN OF GRATITUDE, ESTEEM, AND ADMIRATION,

BY

THE AUTHOR.



PREFACE.

THE following pages contain the results at which I have arrived after a somewhat extensive clinical experience of the gouty dyscrasia in its various forms. While far from denying the influence of heredity and other causes, I am fully convinced that functional disorder of the liver underlies the majority of gouty manifestations, and that the kidneys are only secondarily implicated. If this view be correct, it would seem to follow that the object of treatment should be not merely the neutralisation of the *materies morbi* by means of alkalies, but, in an especial manner, the restoration of the hepatic functions. How this is to be effected, I have described at some length in the chapter on treatment. All statements quoted from other writers have been carefully criticised, and I have endeavoured to make my book in every way a record of my own observation and experience.

9, REGENCY SQUARE, BRIGHTON,
May, 1885.

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CHAPTER I.

GENERAL CONSIDERATIONS WITH REGARD TO GOUT; THEORIES AS TO NATURE, AND MORBID APPEARANCES.

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GOUT is undoubtedly one of the most remarkable and perplexing disorders with which the physician has to deal. Wayward and capricious in its onset and course, and astonishingly varied in the forms which it assumes, its manifestations may at first sight appear to be subject to no intelligible law. An acute attack indeed is marked by a perfectly definite series of phenomena, as regular in their development as

those of uncomplicated scarlatina. On the other hand, the disease may be almost entirely latent, its presence being revealed by obscure symptoms, slight in degree, and evanescent in character, and often referable to their true origin only when a decided family proclivity, or the subsequent progress of the case, serves to guide the diagnosis aright. Midway between these forms are others in which the malady makes itself felt either in the internal organs alone, or in these alternately with the superficial parts. Thus, whilst in certain instances the symptoms may be indicative solely of functional disturbance or of structural mischief in the kidney, liver, or stomach, in others they alternate with external phenomena, such as pain and swelling in one or more of the joints. The study of an affection so complex, and, if I may be allowed the expression, kaleidoscopic in its character, is full of interest, but it obviously presents considerable difficulty.

It is not my intention to discuss in detail the ordinary symptoms of gout, for they are too well known to need description. I shall only refer to such of its features as are more immediately connected with that theory of its pathology which I am anxious to establish. My object in this little work is to place

before the profession my views as to the nature of gout, and more especially as to its relations to those disorders of the liver and kidneys with which, according to my experience, it is very often connected. I shall also add a few words on those affections of the skin which I believe to be due to the presence of the gouty diathesis. I use the word "diathesis" to express "a general tendency, in virtue of which an individual becomes the subject of several local affections, similar in their nature." I believe that gout is a disease of a *specific* character, but capable of assuming widely different forms, and of causing a vast number of symptoms, disturbances, and complications, many of which often remain inexplicable until an acute attack furnishes the key to the diagnosis.

There can be no objection to the use of the word "gout" to designate the disease. Its meaning is well understood, and it is much to be preferred to "podagra," "chiragra," &c., which serve only to describe some of the local manifestations. The term "gout," on the other hand, owed its origin to the "humoral" view of the pathology of the disease, the idea being that some morbid humour existed in the blood, and was thrown out or distilled into the joints "drop by drop." We shall presently see that this

idea is the basis of that theory of the origin of the disease which is most in accordance with well-ascertained chemical and pathological facts.

If we wish to define the word "gout," and to classify the various forms of the affection, we can scarcely do better than adopt Cullen's language and arrangement. Cullen tells us that gout is hereditary, coming on without apparent external causes, but generally preceded by an affection of the stomach; that it is characterised by pyrexia, by pain at some one of the joints, generally at that of the great toe; certainly attacking by preference the articulations of the feet and hands; returning at intervals, and often alternating with affections of the stomach or other internal parts. *Regular* gout, Cullen defines as characterised by considerable and violent inflammation of the joints, continuing for several days, and receding gradually with swelling, itching, and desquamation of the affected part. His second variety is *atonic* gout, in which there is debility of the stomach or of some other internal part, either without the usual inflammation of the joints, or with slight and transient pain in them, often alternating with dyspepsia or other symptoms of debility. In the third form, *retrocedent* gout, the inflammation of the joints sud-

denly disappears, but is soon followed by atony of the stomach, or of some other internal part. In *wandering* gout, there is inflammation of some internal part, sometimes preceded by inflammation of the joints, which quickly disappears. Cullen adds to these descriptions the significant remark that gout is sometimes accompanied by other diseases. This old nomenclature is really based on clinical observation, and does not involve any theory as to the nature of the disease. Cullen's opinion on this point, however, was that gout is an affection of the nervous system.

Whatever form the attacks of gout may assume, other symptoms are pretty certain to become developed during the course of the disorder, and especially in the intervals between the attacks: of these, hepatic derangements, dyspepsia, and gravel are by far the most common. If, moreover, careful inquiry be made into the history of a case of acute gout, we almost always find that the attack has been preceded by more or less distinct symptoms of disorder of the liver or stomach, or of both of these organs; and this fact is of importance as regards the etiology of the disease. There is another well-marked characteristic of gout, viz., that in most cases the attacks become more frequent year by year, without

diminishing in length ; the disease in many cases being really continuous, but presenting irregular exacerbations and remissions. This chronic stage generally owes its origin to serious implication of the kidneys.

Three principal theories are still in vogue with regard to the nature and origin of gout. According to the first of these, gout is due to digestive anomalies, causing the blood to become loaded with certain morbid elements produced mainly in the stomach and duodenum, and uniting in the blood with some element of the bile which has been suffered to accumulate through defective secretory action of the liver. This view was supported by the late Dr. Todd,* who admitted, however, that the condition is usually associated with the lithic acid diathesis. The second theory likewise assumes an impure state of blood, but refers this condition principally to disturbance of the renal function ; Dr. Garrod is the chief supporter of this theory. The third view, that of Cullen, already referred to, has lately been resuscitated. It assumes that the nervous system is primarily at fault in cases of gout.

Putting theories on one side for the present, let us

* "Practical Remarks on Gout, Rheumatic Fever, &c.," p. 74.

see what *facts* we possess which may serve to explain the nature of gout. The most important *fact* in connection with the pathogeny of gout is that uric acid is found in the products of gouty inflammation, and that the blood contains an increased amount of this substance, which in both cases is combined with soda. In health, only the most minute traces of uric acid can be detected in the blood ; in gout, more than one-sixth of a grain has been obtained from 1000 grains of serum. The process for its detection, as devised by Dr. Garrod, is simple enough. One or two fluid drachms of serum are put into a flattened glass dish or capsule, three inches in diameter, and about one-third of an inch in depth ; six minims of strong acetic acid are added to each drachm of serum. The fluids are well mixed, and a few fibres of linen are introduced by means of a glass rod. The glass is then set aside in a cool place until the serum is nearly dry, and if uric acid be present it will crystallise in a rhombic form on the threads, and will be easily recognised under the microscope. The serum must be fresh, for otherwise the uric acid will undergo decomposition and become converted into oxalic acid and urea. When blood cannot be obtained, the experiment may be performed with the serum collected

from a blister, provided that this latter has not been applied to a point attacked by gouty inflammation, inasmuch as inflammatory action causes the uric acid to disappear from the affected part and its immediate neighbourhood. In cases of chronic gout uric acid can always be detected in the serum ; in acute gout it may be absent between the attacks, but can always be discovered shortly before these take place. It must be borne in mind, however, that a similar excess of uric acid in the blood is often found in cases of chronic lead-poisoning and of certain diseases of the kidney. Besides being found in the blood, uric acid often exists in various secretions and fluids in gouty cases. Thus it has been found in the cerebro-spinal fluid ; in the effusion of pleurisy and pericarditis ; in intestinal secretions ; in the discharges from cutaneous eruptions, and in the form of dust (sodium urate) on the skin.

The blood presents other, though far less easily discoverable differences in its chemical composition. These are summed up by Charcot* as follows :—

1. In acute gout the proportion of the corpuscles is not diminished (in this the disease differs

* "Lectures on Senile Diseases," New Syd. Soc. Trans. p. 55.

remarkably from rheumatism); in chronic gout there is considerable diminution and more or less anæmia.

2. In acute gout the fibrin is increased; the clots are buffy.
3. In chronic gout the albumen of the blood is diminished, if there be any disease of the kidneys, and in such cases the blood contains an increased amount of urea.
4. The alkalinity of the blood is diminished, a condition which seems to favour the production of chalky deposits.
5. The blood sometimes contains traces of oxalic acid.

It is evident from the foregoing statements, that while excess of uric acid is the most prominent and characteristic change in the blood in gout, other alterations, more or less important, are frequently present.

The condition of the urine in gouty subjects exhibits several points of interest which may be thus briefly enumerated. During acute attacks the secretion is scanty and high-coloured, but it contains less than the normal quantity of uric acid; perhaps only two grains, instead of seven or eight, are excreted in the

twenty-four hours. The blood-serum can easily be shown to contain excess of uric acid. In the intervals the condition of the urine varies, but there are often deposits of urates and of crystalline uric acid. In chronic gout the excretion of uric acid is for the most part diminished, but from time to time there are crystalline and amorphous deposits containing this substance. Oxalate of lime is often found, tube-casts and albumen are by no means rare, and sugar is occasionally present.

Having thus briefly referred to the state of the blood and urine, the morbid changes in gouty subjects constitute the next point of importance. They are well marked in severe and chronic cases, and are for the most part the direct consequences of the excess of uric acid in the blood. Certain tissues and organs are the seat of deposits consisting of sodium urate, and a careful study of these changes throws much light upon the nature of the disease. The existence of the so-called "chalk-stones" has been recognised from a very early period, but they were regarded as associated only with very severe cases of gout. A closer investigation has shown that even a very slight attack leaves behind it marked changes in the structure of the joints, and that these changes generally remain,

even in favourable cases, throughout life. The cartilages of the joints are the parts primarily involved; deposits of sodium urate form in them, and, as pointed out by Charcot, occupy the superficial part, and are situated either between the cells, or actually within them. The first changes generally take place as far as possible from the insertion of the synovial membrane—a fact which Charcot explains by assuming that the so-called non-vascular tissues are especially liable to be the seat of these deposits. It may be that urates are less soluble in lymph than in blood. On the other hand, the articular surfaces of the bones and the synovial membrane, being highly vascular, are less liable to be thus invaded.

At a later period of the disease, a similar deposit is found in the synovial membrane, and here the same rule is observed, inasmuch as the less vascular portions at the circumference are the first to be affected. As the disease progresses, the ligaments, the tendons, and the connective tissue surrounding the joint are successively attacked. It very rarely happens that deposits of this kind are found in the substance of the bone itself, owing, it may be presumed, to the marked vascularity of the osseous tissue. Even where the surfaces of the cartilage are completely

encrusted, the neighbouring portion of bone is usually free from any trace of uric acid. The bones of gouty persons, however, frequently contain an increased amount of fat and a diminished quantity of earthy matter. The most important point in connection with these deposits is, that they appear to avoid the neighbourhood of the blood-vessels. The cartilage of the ear is often thus affected, and in a few rare cases which have been reported the nodules were visible in this part alone, while on post-mortem examination the ligaments and cartilages of many articulations were found to be more or less thickly covered. In still rarer cases no external deposits are discoverable during life.

As is well known, the metatarso-phalangeal joint of the great toe is the one most commonly affected. Next to it in order of frequency come the fingers, and then the knees and elbows; other joints are liable to suffer, but the morbid changes are very seldom so severe as in those just enumerated. It not unfrequently happens that the great toe is the only joint attacked. Several reasons have been adduced to account for this preference; it has been supposed that this joint, bearing, as it does, much of the weight of the body, and often cramped by ill-fitting boots, is

peculiarly liable to injury, and is therefore a *locus minoris resistentiæ.* This theory is supported by those instances in which injuries to joints in gouty subjects have been followed by the characteristic deposits. A case has been reported to me by a friend, of an extremely gouty subject who took very little exercise save on horseback. In his case, during a long series of years, the knees alone were affected. Similar results of injuries to bones and joints often occur in the course of syphilis.

This deposit of sodium urate is peculiar to gout, and does not occur in any form of rheumatism. It is, as Dr. Garrod points out, the *cause* and not the *effect* of the inflammatory symptoms, for when these are severe the urate undergoes alterations and is removed from the neighbourhood of the affected part. The deposit, examined with the naked eye, is found to resemble plaster of Paris; examined under the microscope, it is seen to consist of minute crystalline needles of sodium urate, often collected into stellate tufts and bundles. As already stated, the deposit is both intra-cellular and interstitial, that is, it occupies the interior of the cells, and the interstices between them. When layers of cartilage thus affected are soaked for some hours in warm water, the deposit

becomes completely dissolved, and the cartilage is almost or quite normal in structure. Wherever the deposit is found, the microscopic appearances are similar to those presented by the affected cartilage. If acetic acid be added, rhomboidal crystals of uric acid will appear. The *tophi*, or chalk-stones, often seen in the neighbourhood of the joints, consist of sodium urate, with a little urate and phosphate of calcium. They differ entirely from the bony nodosities of chronic articular rheumatism. Their most favourite seats are the joints of the hands and feet and external ear. After some time, the skin covering them often becomes thin and brittle, and particles of chalk-like matter are found to cover the surface. In some cases the chalk-stones set up considerable irritation, and abscesses form, which, on bursting, leave ulcers. The discharges from such abscesses are milky and paste-like, and consist of sodium urate, pus, and *débris*. The ulcers are indolent; the granulations are spongy, scanty, and yellowish; and the bases and edges are studded with particles of mortar-like material. Such ulcers often remain without change for long periods, and are apt to re-open after cicatrization.

In order to complete this sketch of the morbid anatomy of gout, allusion must be made to the

various lesions of internal organs, due to the existence of the gouty diathesis. Atheroma of the aorta is not unfrequent in chronic cases, and sodium urate has been found in the coats of the vessel. Fatty degeneration of the cardiac walls, due to atheroma of the coronary vessels, has also been noticed. It is doubtful whether the valvular deposits frequently found in the hearts of gouty subjects are really due to gouty inflammation, inasmuch as they do not contain sodium urate, but consist, as usual, of phosphate and carbonate of lime. With regard to the lungs, sodium urate has been found in the bronchial walls, and uric acid has been detected in the expectoration in a case of gouty bronchitis.

It is, however, in the kidneys of gouty subjects that the most prominent and characteristic lesions are often discoverable, and a close study of these is especially important, as regards not only the etiology, but also the prognosis and treatment of the disease. Gouty nephritis, the so-called *gouty kidney*, is often found in severe and chronic cases, but a less serious affection not unfrequently complicates the slighter manifestations and earlier stages of the disease, and the lesions found in the kidneys may therefore be divided into two classes.

In the first class, the kidneys present deposits of uric acid and sodium urate scattered throughout their substance. These deposits are found in the cortical portion, in the pyramids, and in the papillæ, and likewise in the calyces and pelvis. The substance of the kidney is hyperæmic, and otherwise resembles in appearance that of the first stage of chronic interstitial nephritis. The changes may be regarded as the results of gouty inflammation, affecting not only the uriniferous tubules, but also the fibrous structures of the organ, and accompanied by the deposits of urates. Dr. Garrod's view, that the fibrous structures of the kidney are sometimes involved before the joints, is supported by those instances in which severe pain in the back precedes and is relieved by the appearance of swelling in the great toe. The occurrence of temporary albuminuria is doubtless sometimes due to imperfect metamorphosis of albuminous substances by the liver, and perhaps to transient gouty inflammation of the kidneys. If, however, the albumen is permanently present, changes of a serious nature must have set in.

This question of albuminuria in connection with gout will be especially discussed in a subsequent chapter.

In the second class of cases, the kidneys present not only the infarctions above described, but in addition marked alterations of structure corresponding to those of chronic desquamative and interstitial nephritis. The infarctions appear as whitish streaks in the medullary substance, and the papillæ present little whitish points. The deposits are formed in and obstruct the uriniferous tubules, and are also found in the interstitial tissue. Under the microscope these infarctions are seen to consist of crystalline needles of sodium urate, and they are chemically identical with the substance found in chalk-stones, and in the incrustations covering the articular cartilages. The structure of the kidney, moreover, presents marked alterations. According to Charcot, there is at first a tubular nephritis, of two different degrees. In the first, the size of the kidney is unchanged, but the cortical substance is thickened and yellowish in colour. The Malpighian bodies are injected, and the uriniferous tubules are filled with epithelium cells, distended, opaque, and full of fatty or albuminoid granules. In the second stage there is atrophy of the cortical substance, and a granular condition of the kidney generally.

The alterations above described affect principally

the uriniferous tubules, but in the gouty kidney, properly so called, there are likewise interstitial changes of a cirrhotic character. Thus there is thickening of the intertubular connective tissue and proliferation of the nuclei; the kidney becomes harder and diminished in weight and volume. The capsule is thickened and opaque, the surface is wrinkled and nodulated, and the cortical substance much reduced in quantity; in some cases the pyramids almost reach the surface of the organ. The infarctions of sodium urate appear as whitish streaks in the pyramidal portion. In some cases of this character the kidney has been found to weigh only two-and-a-half ounces.

Of the renal lesions just described the deposits of sodium urate may be designated as altogether peculiar to gout; for they are seldom, if ever, found in connection with any other disease. The other appearances are often found in that form of Bright's disease which is termed "cirrhosis" of the kidney. The co-existence of the urate infarctions with the contracted kidney frequently occurs in advanced cases of gout, and indicates a very close relation between the two diseases. There is also strong evidence in favour of the view that the kidneys

are often affected at a very early period, even after a few moderate attacks have occurred. A granular condition of the epithelium, with excess of oil globules, and white streaks in the pyramids, constitute the earliest changes, while crystals of sodium urate occupy the intertubular fibrous structure and the tubules. The changes may co-exist with a perfectly natural size and weight of the kidneys, and with such a condition of their structure as appears normal to the naked eye. This fact has an important bearing upon the etiology of gout, and upon the part which renal disease takes in intensifying and perpetuating its manifestations.

Whether the kidneys simply excrete or really secrete uric acid, disease of their tubular structure must affect the amount of this substance eliminated from the body, just as occurs in the case of urea.

In advanced stages the tubes become denuded of epithelium and are shrivelled and wasted. The Malpighian bodies are brought closer to each other and appear to have increased in number. The coats of the vessels, especially of the afferent arteries of the tufts, are thickened or hypertrophied, and the vessels themselves become tortuous. All these changes still further impede the excretion of the urinary constituents.

Sodium urate, having been thus shown to be the *materies morbi* of gout, I shall, in the next chapter, discuss the existing theories with regard to the source and place of origin of uric acid.

CHAPTER II.

SOURCES AND PLACES OF ORIGIN OF URIC ACID IN THE SYSTEM.

VARIATIONS IN QUANTITY OF URIC ACID UNDER VARIOUS CIRCUMSTANCES—IN DISEASE—INFLUENCE OF ACIDITY—MODE AND PLACES OF ORIGIN OF URIC ACID IN THE ECONOMY—TWO THEORIES: (1) FORMATION IN THE SYSTEM, ESPECIALLY IN THE LIVER; (2) FORMATION IN THE KIDNEYS ALONE—EXPERIMENTS ON BIRDS AND SNAKES—CONCLUSIONS TO BE ARRIVED AT—DR. PARKES' VIEWS—DR. LATHAM'S THEORY OF GOUT — RELATION OF URIC ACID TO GOUT — TWO THEORIES: (1) RETENTION OF URIC ACID IN THE BLOOD AS A RESULT OF DEFECTIVE ELIMINATION BY THE KIDNEYS; (2) URIC ACID INCREASED AS A RESULT OF SUB-OXIDATION OR OTHERWISE—CONSIDERATION OF FIRST THEORY—OBJECTIONS TO IT.

THE account given in the previous chapter of the pathological appearances observed in gout clearly shows that the deposition of sodium urate in the cartilaginous structures of the joints is the fundamental *fact* upon which all theories as to the nature of the disease must be founded. The demonstration of an excess of uric acid in the blood constituted an important step towards the discovery of the cause of the

disease ; but the sources and origin of uric acid in the system, even under normal circumstances, are still to be reckoned among the obscure points of physiology. It is therefore difficult to construct a complete and satisfactory theory which will explain the excessive production of this substance in disease. Before, however, entering upon the question of excessive production, it seems desirable first to discuss the various opinions now in vogue concerning the sources and places of origin of uric acid in the human body.

Under ordinary circumstances a healthy man excretes only 8 grains of uric acid ($C_5H_4N_4O_3$) *per diem*. It exists in the urine partly free and partly combined with the alkaline phosphates. The quantity rises and falls with that of the urea ; the average proportion between the two substances being 1 to 50. Under conditions of abstinence from food, and when only non-nitrogenous substances are taken, the quantity excreted may fall as low as two or three grains ; on the other hand, with a full animal diet it may rise as high as 30 grains. It is, however, a matter of fact that an excessive amount of uric acid is sometimes regularly excreted by pale and impoverished persons living on a diet poor in quality and not over-abundant. Under such circumstances the excess may be sup-

posed to be due to imperfect metabolism. As a general rule, the variations in quantity are independent of differences in age, sex, height, weight, or temperature. Moderate exercise appears to cause a slight diminution in the quantity; prolonged or violent exertion, on the other hand, produces an opposite effect. A decided diminution has been observed after copious draughts of water, and the quantity is said to be diminished after the use of alcohol, and after the administration of quinine in full doses. Iodide of potassium, chloride of sodium and caffein cause more or less diminution, though the statements with regard to salt are somewhat conflicting. The inhalation of oxygen is followed by rapid diminution of the uric acid, or even by its total disappearance from the urine. The administration of the carbonates of soda and lithia has been thought to exercise a very decided influence upon the excretion of uric acid, inasmuch as urinary deposits composed of this substance rapidly disappear during a course of these drugs. It is, however, doubtful whether the uric acid is really *diminished in quantity*, and it would seem more probable that the disappearance of the sediment when alkaline carbonates are taken is due to the conversion of the acid phosphate of soda into a

neutral salt, and a consequently *diminished precipitation* of the uric acid. Besides this, some of the uric acid would unite with the alkalies, and form urates which are far more soluble than uric acid. The use of mineral waters, containing the sulphate and carbonate of soda, with a little chloride of sodium, is followed by a decided diminution of uric acid in the urine.

The known variations in the quantity of uric acid excreted in cases of disease are as follows :—

In febrile states of the system, *e.g.*, in traumatic fever, typhoid, small-pox, and acute rheumatism, the quantity of uric acid rises and falls with that of the urea. It is absolutely increased in those cases in which the elevation of temperature is associated with impediments to respiration, as occurs when such complications as pneumonia, capillary bronchitis, pericarditis, or pleuritic exudations are present. In cases of disease in which the action of the diaphragm is impeded from any cause, as in abdominal tumours, ascites, &c., there is an increased amount of uric acid in the urine. It would therefore appear that an increased excretion of uric acid generally occurs whenever there is *decreased energy* of the processes of *oxidation*.

In chronic gout, with deposits of urates in and about the joints, there is, as we have seen, a much diminished excretion of uric acid by the kidneys. In some forms of chronic splenic tumour, a similar condition is observed; but in others, and notably in cases of leucocythæmia, the quantity is often enormously increased. In one reported case, the daily excretion amounted to sixty grains. It may be that this increase is due to increased production in the diseased spleen, or to diminished oxidation in the system.

In anæmia and chlorosis, the uric acid is reduced in quantity, unless there be dyspnœa, in which case an increase is observed. In chronic affections of the respiratory and circulatory organs, accompanied by difficulty of breathing, and a consequent diminution in the supply of oxygen, the uric acid is increased in quantity. In carbonic oxide poisoning the proportion of uric acid to urea has been observed to be as high as 1 to 27.

In chronic affections of the kidney, diabetes, and polyuria, the uric acid is generally diminished in quantity. In diseases of the liver there are considerable variations; in cirrhosis the quantity of sodium urate excreted is sometimes very high. Also in acute congestive hepatic affections the quantity is much

increased, but according to Dr. G. Harley,* it is diminished in fatal permanent jaundice. In various forms of indigestion, and in cases of diminished cutaneous activity, sediments of urates are of common occurrence in the urine on cooling, but their precipitation must not be regarded as necessarily indicative of the presence of an unusual amount of uric acid. There is, moreover, as pointed out by Dr. Bence Jones, no relation "between the *acidity* of the urine and the *absolute amount* of uric acid which it may contain; for in the urine which is most acid, and which deposits the largest uric acid sediment, very little uric acid may really exist; whilst that which contains most uric acid may hold it in perfect solution, and may have but a feeble acid reaction." The presence of some other acid is doubtless the main cause of the deposit of uric acid sediments, for the addition of any acid to the healthy secretion passed soon after food is always sufficient to produce it. The temperature of the fluid also influences the precipitation; if this be high, a larger amount of uric acid is held in solution than under opposite conditions. Concentration of the liquid, on the other hand, favours the deposition of the uric acid sediment, for it augments the proportion

* "Diseases of the Liver," p. 761.

of the urate to the water, and intensifies the acid reaction. "Thus the uric acid sediment may be regarded as dependent upon three concurrent conditions, (1) decrease of temperature, (2) increased proportion of uric acid compound to the water, positively or relatively, (3) increased acidity of the urine."* It is therefore evident that a copious precipitate either of uric acid or urates is no real indication of the quantity of uric acid actually present, which can be determined only by analysis.

Having thus described the principal variations occurring in the quantity of uric acid eliminated, the next subjects for consideration are the mode and place of origin in the human body of this important constituent of the urine.

Uric acid is one of the results of the metabolic processes which are constantly going on during life. Of the four principal elements, carbon, hydrogen, oxygen, and nitrogen, of which the body is composed, and which constitute the bulk of the excrementitious materials, the nitrogen is eliminated almost exclusively by the kidneys, in the forms of urea and uric acid. These two substances differ in one very

* See Carpenter's "Principles of Human Physiology," ninth edition, p. 476.

important respect, viz., as regards their solubility, urea being very soluble in water and in the fluids of the body; uric acid, on the other hand, requiring for its solution about eight thousand times its weight of distilled water at the temperature of the blood. Uric acid unites with various bases, and the salts thus formed are more soluble than the acid itself. They are, however, much less soluble than many other substances, and they and uric acid readily crystallise out from their solutions. It is owing to their insolubility and their proneness to reassume the solid form, that uric acid and its salts, though existing in such small amounts in the urinary excretion, so frequently give rise to disease. The fact that the urine of birds and of reptiles is composed almost entirely of uric acid is one of considerable interest as regards the relations of this substance to urea.

Two principal theories have been advanced with regard to the *place of origin* of uric acid in the animal economy. According to the first of these, uric acid is formed in the system in general, during the changes which are constantly going on. Some authorities believe that it is produced mainly in the spleen, liver, and lymphatic glands; others think that the connective tissue is the principal seat of origin.

It is certainly found in considerable quantities in the liver and spleen, but only slight traces are discoverable in the muscles. After its formation it passes into the blood, and is rapidly eliminated by the kidneys. According to this view, the kidneys merely serve as a filter through which the uric acid passes.*

According to the second theory, uric acid is produced exclusively in the kidneys, and does not exist preformed in the blood. The epithelial cells of the uriniferous tubules are credited with the power of selecting from the blood the necessary materials, and of converting them into uric acid. The fact that uric acid has been repeatedly discovered in the blood appears to militate against this theory; but its presence is explained by assuming that it is absorbed from the cells of the kidney.

In reference to this important point, it may be useful to refer to some experiments in which the ureters of certain birds were occluded by means of ligatures. As is well known, the urine of birds consists to a very great extent of uric acid, but this

* See Dr. Garrod's Lumleian Lectures on "Uric Acid and its relation to Renal Calculi and Gravel," *Medical Times and Gazette*, 1883, vol. i.

substance has not hitherto been detected in the blood of these animals. After the ligature of the ureters, incrustations of urates were found throughout the kidney (the Malpighian corpuscles alone excepted), on all the serous membranes, on the mucous membrane of the tongue, œsophagus, and bowels, in the spleen, liver, gall-bladder, lungs, joints, muscles, bones, and lymphatic vessels; only the brain and the blood remained free. The same results were obtained after ligature of the ureters in snakes; but after removal of the kidneys in these creatures, deposits of urates were found only upon the edges of the wound, and in the places which the kidneys originally occupied. Inasmuch as when the kidneys were suffered to remain, and the ureters tied, copious deposits were found, but when these organs were removed, the deposits were almost absent, it might be inferred that uric acid is produced, in these animals at least, exclusively by the kidneys.

Several objections may, however, be raised against this interpretation of these experiments. If uric acid were formed in the kidneys alone we should not expect to find it in the cicatrices left after the removal of these organs. Moreover, in the experiments in which the ureters were tied, with a copious

deposit of urates in many organs and tissues as the result, we should expect to find uric acid in the blood, if this substance were formed in the kidneys, and carried by the circulation to the parts in which the deposits occur. It is well known that the tubuli uriniferi of serpents generally contain large deposits of urates, and it is quite possible that the incrustations found in various organs after ligature of the ureters may have resulted from the absorption of the deposits originally contained in the kidneys. Moreover, the discovery of urates in so many organs while the blood was free from them would seem to prove that the uric acid was actually formed in the organ in which it was found.

Other arguments might be adduced to show that the formation of uric acid takes place throughout the body. It may be that a minute quantity is produced in the kidneys as a result of the active changes going on in these organs; but the single fact that uric acid can be discovered in the connective tissue and in various organs in a state of health, is conclusive evidence that its production takes place throughout the body generally. A similar source of origin may be claimed for urea, which is most probably formed in all the glandular cells of the body, but especially in

the liver. According to Dr. Parkes,* in cases of hepatitis and hepatic abscess, when suppuration was excessive, the urea was found to be lessened in a degree proportionate to the extent to which the secreting structure was destroyed by the abscess. When the liver was not suppurating, but actively congested and enlarged, the amount of urea and uric acid seemed to be increased. With regard to the relations between these two substances, Dr. Parkes' opinion was that uric acid is not an anterior stage of urea, but has an independent origin of its own in some cells which are especially endowed with the power to form it. From his observations of several cases of enlarged spleen, attended by a nearly four-fold excretion of uric acid, he was led to suggest that the spleen may perhaps produce more uric acid than urea. Some physiologists think that urea is produced in the liver, and results from the disintegration of the hæmoglobin of the effete red blood-corpuscles. Dr. G. Harley† thinks that urea is not a special product of the liver, but the ultimate product of all tissue disintegration. He admits, however, that in acute yellow atrophy of

* *Lancet*, 1871, vol. i, p. 467.

† "Diseases of the Liver," p. 761.

the liver, the amount of urea eliminated diminishes in proportion to the destruction of the liver-tissue.

A theory recently advanced by Dr. P. W. Latham* with regard to the formation of uric acid, is thought by him to meet several difficulties connected with other views. Dr. Latham suggests that urea is formed from the glycocine (glycine) of the bile. After this fluid has served its purpose in digestion, the glycocine and taurine are returned into the blood, and are carried by the portal vein into the liver. In this organ these substances, together with leucine and tyrosine, are converted into urea; but if from any cause the metabolism of glycocine is interrupted while taurine and leucine, &c., still undergo the normal changes, the liver would contain both urea and glycocine. Dr. Latham further assumes that the conjugation of these substances by the gland would result in the production of certain compounds, one of which on arriving at the kidneys is, in these organs, conjugated with urea and is excreted as ammonium urate.

This substance is very slightly soluble, one part requiring as much as 2,400 parts of water at 100° Fahr. for solution. In consequence of this property a

* "On the Formation of Uric Acid in Animals and its relation to Gout and Gravel."

certain portion may escape excretion and remain in the blood—"overflow, as it were, and so pass on into the circulation." In this fluid the ammonium salt meeting with the soda would be converted into sodium urate, the form in which it is deposited in and around gouty joints. Dr. Latham concludes that the appearance of uric acid in the secretion, is the result of the imperfect metabolism of glycocine into urea, whether that glycocine be derived from the bile poured out into the duodenum or formed elsewhere in the body. That it is from the bile is made somewhat probable from the fact that in the carnivora whose urine contains little or no uric acid, the bile contains no glycocholic, but only taurocholic acid, and therefore no glycocine.

According to this view the imperfect metabolism of glycocine is the primary and essential defect in gout and gravel, and therefore functional disorder of the liver is considered to take a prominent share in the production of these disorders. Dr. Latham, however, thinks that some change in the nervous system is the most important factor in their etiology, such change being either hereditary or acquired, and being situated in the medulla oblongata, spinal cord, or both. I shall have occasion to refer again to this theory in a sub-

sequent chapter. The adoption of Dr. Latham's view as to the production of urea involves the acceptance of another and, as I think, an untenable theory, viz., that the formation of uric acid takes place in the kidneys. Moreover, our knowledge of the metabolic processes which go on in the organism is far too imperfect to allow us to assume that they are always identical with changes which can be produced in the laboratory.

The question as to the relation of uric acid to gout will now be discussed, and I proceed to inquire whether the disease is due mainly to retention of uric acid in the system, or whether its primary cause is an increased formation of this substance, as a result of suboxidation of the tissues or otherwise.

The theory of the retention of the uric acid has of late years been upheld by several observers, and notably by Dr. Garrod.* Whichever theory be adopted with regard to the origin of the uric acid, it is assumed that in gout the excretory power of the kidneys is defective as regards this constituent, though not as regards

* "A Treatise on Gout and Rheumatic Gout," third edition, p. 280. Dr. Garrod says: "The imperfection in the eliminating power of the kidneys sometimes appears to be the chief, if not the only cause of the impurity of the blood." Dr. Garrod of course allows that increased formation of uric acid plays a certain part in the production of gout.

the urea and other substances. As a result of this defective capacity of the kidneys, the uric acid accumulates in the blood, and the prodromal symptoms of gout are likely to become manifest. If the patient's habits of life are such as to cause an increased production of uric acid, an attack of gout will sooner or later take place, and the disease will then pursue its course with more or less regularity. According to this theory, a functional affection of the kidney is the true pathogenetic cause of gout. The experiments in which the ureters of fowls and geese were tied, and the operation was followed by deposits of sodium urate in the joints, kidneys, and other viscera, have been thought to support this view.

There are, however, many objections to this theory. Gouty deposits do not necessarily take place in connection with those diseases of the urinary organs which are attended with a much diminished secretion of urine. Moreover, gout often occurs in individuals whose kidneys are to all appearances healthy, but whose blood can be shown to contain an excess of uric acid. If the urine be examined in the early stages of gout, there is, as a general rule, no evidence of any renal affection. It is quite true that on post-mortem examination, the kidneys of gouty subjects

are often found to contain uric acid infarctions and to present other morbid appearances, but these are the consequences and not the causes of the gouty dyscrasia. It may readily be conceived that the irritation, often repeated or almost continuous, which deposits of urates in the uriniferous tubules must sooner or later set up, will give rise to serious changes in the renal tissue, and the deposits which occur in the interstitial tissue of the kidneys may be regarded as the result of an attack of gout localised in these organs. Persons who have never had any attacks of gout, but whose urine often contains a deposit of uric acid, are especially prone to suffer from inflammation of the kidneys, and the relationship between gout and uric acid deposits is based upon the fact that the uric acid dyscrasia is common to both conditions.

Uric acid deposits and gout are frequently associated in the same subjects, but cases sometimes occur in which there are severe paroxysms of gout, but no marked deposits of uric acid in the urine. Under such circumstances neither albumen nor casts are likely to occur. Such patients are, however, very liable to renal inflammation, and when this occurs there will be an increase of uric acid in the blood and a diminished amount in the urine. If the mere

occlusion of the tubules could give rise to gout, or if the excess of uric acid in the tissues of gouty persons were the consequence of its retention in the kidneys, the disease would be of far more frequent occurrence, and even infants would be liable to gouty deposits, inasmuch as Virchow has shown that the kidneys of new-born children frequently contain uric acid infarctions. In addition to this, it is well known that many persons suffer more or less from gravel during their whole life, but are quite free from attacks of gout. Some members of a gouty family exhibit all the symptoms of the disease; others suffer only from gravel. It can easily be understood that an affection of the kidneys which is accompanied by a lessened excretion of urinary constituents will tend to increase the frequency and severity of the gouty attacks. So long as the kidneys remain sound, a rapid excretion prevents undue accumulation.

The experiments above referred to in which ligature of the ureters in geese and chickens was followed by deposits of urates in and upon various organs will scarcely help us to explain the phenomena of gout. If the human organism produced as much uric acid as that of birds, and if the flow of urine through the ureters were absolutely prevented so that no more

could possibly be removed by the kidneys, it is quite conceivable that the uric acid would be deposited in and upon all organs of the body adapted for its retention. There is, however, a very decided difference between such deposits, and those of gout occurring in an individual whose urine is free to escape.

The theory that in gout the uric acid excreting function of the kidney is defective, rests upon a pure hypothesis, and could be accepted only if no more suitable theory could be discovered. Even those who support it admit that it accounts only for the prodromal symptoms of gout, and they allow that an increased production of uric acid is necessary for the full development of the disease. Such increased production will form the next point for consideration.

CHAPTER III.

THEORIES AS TO THE NATURE OF GOUT.

Excessive Production of Uric Acid in Gout—Circumstances influencing the Excess—Formation of Urea and Uric Acid by the Liver—Functions of the Liver—Temperature of the Liver—Lithæmia due to Hepatic Derangements—Symptoms of the Uric Acid Diathesis—Frequent occurrence of temporary Albuminuria in Gouty Dyspepsia — Eczema as a Symptom of the Gouty Diathesis—Neurotic Theories of Gout—Views held by Dr. Dyce Duckworth, Dr. Meldon, Dr. E. Liveing, Sir J. Paget, Dr. Ord, and Dr. Latham — The Author's Views.

Towards the close of the preceding chapter I endeavoured to show that defective diminution of uric acid is not sufficient to account for its undue accumulation in the blood in cases of gout. I now proceed to discuss another theory, viz., that of excessive production of uric acid in the system, a condition which is liable to occur under the following circumstances :—

In the first place, albuminous constituents may be supplied in excess, and the surplus fails to become properly oxidized. In the second, the oxidizing pro-

cesses are imperfectly performed; though the supply of those constituents may be no more than is fairly proportioned to the normal wants of the organism. There is also a third class, in which not only are the oxidizing processes far below the normal standard, but the supply is manifestly in excess of the requirements. Under all these conditions the blood is liable to become surcharged with imperfectly oxidized constituents, of which uric acid is, for our present purposes, the most important.

In the healthy organism by far the larger part of the nitrogenous excreta appears as urea, which is more highly oxidized than uric acid. Whether the entire amount of urea which is eliminated from the system results from the oxidation of this latter substance—that is, whether uric acid forms a necessary stage in the production of the urea—is a question which must be answered in the negative. It is possible that uric acid may be only one of several substances, out of which urea is formed; and in a healthy organism its production may be the result of changes slightly divergent from those concerned in the formation of urea. It has been already stated that in all probability several tissues of the body participate in the production of these substances, but there is strong

evidence in favour of the view that the liver is the principal seat of their origin.

Up to within quite recent times the secretion of bile was thought to constitute the principal, if not the only, function of the liver; though it would appear that the earliest writers on medicine had far more extended views as to the work performed by this organ. It now seems clear that these old views deserve to be revived, and that the liver must be credited with the discharge of at least *three* functions, viz.: (1) the secretion of bile, (2) the formation of glycogen, and (3) the destruction of albuminous matters derived from the food and textures, and the formation of urea and uric acid. It is with this third function that an endeavour to trace the origin of gout is closely concerned, and it is to the writings of the late Dr. Murchison that the profession is especially indebted for a clear exposition of the present state of knowledge on this subject,* and for the inferences to be deduced therefrom. His observations show that the liver is largely concerned in the formation of the nitrogenous matters which are eliminated by the kidneys. Some evidence in favour of this theory has been adduced in

* Murchison, "Functional Derangements of the Liver." Second edition, 1879.

preceding pages, but there are other facts which deserve attention. Deposits of uric acid and urates, and an imperfect formation of urea are frequent signs of functional as well as of organic affections of the liver; while there is experimental evidence of the existence and formation of urea in this organ.

Another fact, referred to by Dr. Murchison, has an important bearing on the subject of the hepatic functions. It appears from Bernard's experiments that the temperature of the healthy liver reaches 104°, or even 106°. In dogs, the temperature of the blood in the hepatic veins is much higher than that of the portal vein, and the temperature of the upper part of the vena cava is higher than that of any other part of the body. The active chemical changes going on in the liver are the probable cause of this elevation of temperature, and this view is further supported by the fact that in diseases of the liver the temperature is often lower than normal, and that after the common duct has been tied in animals there is no longer any difference in temperature between the blood of the portal and that of the hepatic vein.

If this view of the function of the liver be correct, it is easy to understand how the condition termed *lithæmia*, in which there is an excessive amount of

uric acid in the blood, may often depend upon derangement of the liver, and therefore that the gouty dyscrasia may have a similar causation. If there be an imperfect transformation of albuminous matters, with the production of uric acid instead of urea as a consequence, we have at least *one* of the factors necessary for the development of gout. The most common cause of such imperfect transformation is an excess of supply, combined (as it very often is) with deficient action of the assimilating organs. It is well known that an occasional deposit of urates in the urine is a common result of over-indulgence at the table, and is of no serious significance. When, however, such a deposit is constantly or even frequently noticed, it is of more or less grave import. Patients are apt to think that the kidneys are "out of order," but in point of fact it is not these organs, but the liver, which is generally in fault; and this distinction is one of paramount importance as regards the treatment, whether curative or prophylactic, of such cases.

When these deposits are of constant occurrence, the uric acid diathesis may be said to have become established; and in the majority of cases other symptoms, perhaps noticed occasionally before, soon become troublesome. There is more or less dyspepsia,

as evidenced by flatulence, distension, and feelings of uneasiness, or even severe pain, in the stomach and duodenum. There is often nausea and either pyrosis or acid eructations, in many cases there is a bitter taste in the mouth, the tongue is dry and furred, the bowels are irregular and generally constipated, the liver is somewhat enlarged and tender on palpation, the skin sometimes has a slightly jaundiced hue. Palpitation of the heart and shortness of breath, aggravated by exertion, are often complained of, and a short dry cough is sometimes very troublesome.* Symptoms of derangement of the nervous system are almost always superadded. The temper becomes irritable and the spirits are depressed. The patient feels uneasy and restless, or else there is lassitude, drowsiness (especially after food), headache, and inability for mental exertion. The sleep is broken and unrefreshing. Noises in the ears and vertigo are sometimes very troublesome and alarming. Hypochondriasis is not unfrequent, and in women hysterical symptoms may occur. In some cases frequent attacks

* Dr. Woakes (on "Post-Nasal Catarrh") has described many of the symptoms of the uric acid diathesis as those of "the premonitory stage of catarrh," and has explained in a very lucid manner the decided predisposition towards taking cold evinced by many subjects of this diathesis.

of migraine, or of other forms of neuralgia, are prominent symptoms; in others, the small joints are painful from time to time, but there is no decided attack of gout. Pains in the heels and painful sensations of burning and tingling in the feet are sometimes present and cause great annoyance.

It is obvious that all these symptoms are connected with some peculiar condition of the system at large. They are liable to periodical exacerbations, and they are invariably aggravated by errors of diet. A little beer or a glass or two of champagne will often cause twinges in the knuckles, burning sensations in the palms and soles, or even more decided manifestations in these subjects.

Cases presenting many of the above symptoms, variously combined, often come before me in practice, and I never fail to examine the urine, which, in not a few instances, I find to contain a distinct quantity of albumen, without tube-casts. In a succeeding chapter I shall endeavour to explain this appearance; for the present it will be sufficient to say that I do not regard the albuminuria in these cases as necessarily indicative of pathological changes within the kidneys. The urine is likewise scanty, generally high-coloured, and abnormally acid; after standing, it deposits an abun-

dance of urates and uric acid. Oxalate of lime is often present. Precipitation of urates sometimes takes place within the bladder, and in that case the urine is more or less turbid when passed.

In a majority of cases the attack of articular inflammation is preceded by many of these symptoms, and appears to follow them directly; while in some decidedly gouty persons no other signs of gout than those which characterise the uric acid diathesis occur at any period of life. It may be said that in these latter cases the gouty phase is never reached. It is worthy of remark that some patients who suffer from aggravated dyspepsia, and from gout, find themselves very much better than usual, as regards the dyspeptic symptoms, for some little time before the acute attack.

There is one other symptom which I must not fail to mention. In many of my patients presenting the symptoms of gouty dyspepsia, I have noticed the occurrence of eczema, which appeared to alternate with the gouty symptoms, inasmuch as it increased in severity as the latter were relieved by the treatment. The dyspeptic symptoms can be removed by very careful attention to diet and appropriate medicines, and, if due care be taken, they will not necessarily

recur. The patient, however, is still liable to attacks of eczema, especially in the spring of the year, and the complaint, in the absence of further treatment, tends to become almost continuous. I shall have more to say on the connection between eczema and gout in a subsequent chapter.

An attempt to explain the nature of gout would obviously be imperfect without taking into consideration the evidence in favour of the *neurotic* origin of the disease. By some authorities gout is regarded as a disorder of the nervous system, that is, as a tropho-neurosis—a view which has recently been advocated by Dr. Dyce Duckworth.* This would appear to be a convenient place for an account of the arguments brought forward by him and other writers in support of this theory.

Dr. Duckworth admits that it is impossible to resist the evidence in favour of the direct connection between the excess of uric acid in the blood and the manifestations of gout, and that whatever views be entertained as to the *whole* of the pathogeny of this disease, the *facts* discovered by Dr. Garrod cannot be set aside. The question, however, is whether the theory of the excess of uric acid being the cause

* *British Medical Journal*, March 26, 1881.

of gout is *sufficient* to account for all the phenomena. Cullen, as already mentioned, regarded gout as manifestly an affection of the nervous system, and asserted that gout is more indicative of nervous disorder than any other pyrexia. Stahl, however, was the first to advocate the neurotic theory of gout, and Cullen adopted his views.

Diseases belonging to the neurotic type may be either primary or central, or may be secondary or induced, and it is alleged that gout presents not a few of the characteristics peculiar to the neuroses, *e.g.*, heredity, the periodicity of the attacks, and subjection of the disease to the law of alternation. Primary gout is regarded as a *diathetic* neurosis, but a difficulty arises in accounting for cases of gout occurring in the absence of any neurotic taint. It is assumed that, at least in some of these, a condition of lithæmia and hyperinosis is set up as a result of high living, &c., and that the consequent dyscrasia of the blood reacts upon the nervous centres; in other words, that a secondary affection of some nerve-centre occurs in consequence of the altered state of the blood, and thus the order and special phenomena of the gouty attack become developed. The fact that joint-affections are occasionally witnessed in the course of

diseases of the cord and brain is also adduced as a strong argument in favour of this theory; and the gouty arthritis is supposed to be connected with these affections. A trophic centre for joints is assumed to exist near the roots of the vagus in the medulla oblongata.

In addition to such features as heredity and periodicity, gout is considered to present many other analogies with disorders of the nervous system. The sudden supervention of the attacks; the preceding sensations of ease and comfort so often experienced; the time of the occurrence of the attacks and their paroxysmal character; the connection of the disease with other well-recognised neuroses; and the fact that the same causes are liable to excite attacks, have all been cited as evidences of the close relation which exists between gout and the nervous system. The remarkable connection between gout and diabetes is also adduced; it being a well-recognised fact that the two diseases sometimes alternate, and that members of gouty families are prone to suffer from diabetes. It is also claimed that this theory of the origin of gout serves to explain the effect of colchicum—a drug which has a powerful action upon the nervous system.

Dr. Meldon,* of Dublin, has long advocated what may be termed a neuro-humoral theory. He admits that uric acid and soda must exist in the blood before the disease can be produced, but is convinced that the presence of these substances is not the sole cause of gout. His view is that there must be *depression of the nervous system* to cause an attack of gout—such depression bringing about the union between uric acid and soda so as to form the urate of soda. According to this theory, nervous force when in a normal condition keeps these two substances separate and in a fluid state, and in a condition fit for elimination by the skin, kidneys, and bowels. When the nervous influence is lessened, the uric acid and soda unite in the tissues most removed from the blood and circulation. The irritation and inflammation excite the nervous system to increased energy, and the disease for the time is arrested. One objection to this theory is constituted by the fact that urate of sodium is far more soluble than uric acid, and therefore more easily eliminable by the kidneys.

Dr. E. Liveing,† in his classical work on Megrim,

* "Gout, Rheumatism, and Rheumatic Gout," 1872; and *British Medical Journal*, vol. i, 1881, p. 466.

† "Megrim, Sick-headache, and some Allied Disorders: A Contribution to the Pathology of Nerve-Storms," 1873, p. 404.

is much inclined to assent to the view that gout in its various forms is the manifestation of a disorder which has its primary seat in the nervous system itself; and he lays great stress on the connection which may often be traced between gout and such disorders as "megrim, asthma, angina pectoris, gastralgic paroxysms, and certain forms of transient mental derangement." Sir James Paget expresses a somewhat more guarded opinion with regard to the part played by the nervous system in the causation of gout.* He says: "Disturbance in the nervous system in some form and part may be regarded as a factor in every case of gout. There are reasons enough for thinking that changes in the nervous centres determine the locality of each gouty process, while changes in the relations of the blood and tissues determine its method and effects; and that thus we may explain the symmetries of disease in gout, sometimes bilateral, sometimes antero-posterior, and thus its metastases. But these changes are a part of the pathology of gout which is not yet clinical."

According to another theory, proposed by Dr. Ord,† the part played by the nervous system in this disorder

* "Clinical Lectures and Essays," 1879, p. 382.

† "St. Thomas's Hospital Reports," 1872.

is held to be the propagation of gouty inflammation from part to part. It is also considered that there are evidences of the direct action of the nervous system in the production of the attacks. When the gouty diathesis exists, any sudden excitement of the nervous system is capable of producing gouty inflammation in a violent form, and in several parts at once. As to the nature of gout, Dr. Ord's views are that the disease is a mode of decay of the whole system ; that the deposit of urates is the result of local or general disintegration, the local inflammation not necessarily depending upon such deposit, but often set up by local exciting causes ; that the local inflammation and degeneration tend to infect the rest of the system through the blood, and to set up similar actions elsewhere through reflex nervous influence. These views have been adopted in the main by Dr. Bristowe.*

A reference to the recently published views of Dr. P. W. Latham† will conclude this part of the subject. Dr. Latham thinks that some change in the nervous system is the most important factor in the etiology of gout ; and that the change is localised in

* "The Theory and Practice of Medicine," fourth edition, p. 874.

† "On the Formation of Uric Acid in Animals," &c., 1884, p. 30.

the medulla oblongata or spinal cord, or both, and that it may be either hereditary or acquired. He considers (1) that derangement of the hepatic functions leading to excessive formation of uric acid may be connected with some "weak spot" in a portion of the medulla oblongata involving some of the roots of the vagus; (2) that uric acid in the blood resulting from hepatic derangement might act upon any weak spot near the roots of the vagus and cause gastric troubles, asthma, cardiac irregularities, &c.; (3) that if in addition to some change in the medulla oblongata, those portions of the spinal cord which control the nutrition of the joints are affected, articular inflammation with deposition of sodium urate will become developed.

Such then are the theories now in vogue with regard to the pathology of gout. The fact that the blood in this disorder contains urate of sodium is common to them all, but it is evident that very different views are held as to the part played by this substance in the causation of the phenomena. I have given a somewhat lengthy account of the *nervous* theory of the disease, because it is advocated by many distinguished authorities at the present day, and bids fair to gain increased acceptance. It appears, however, to be open to several objections, and cannot, I

think, be adopted in its entirety, especially in the form given to it by Dr. Latham.

It must be admitted that disorder of the nervous system is a prominent feature in many cases of gout; but it is surely going too far to assume that this disorder is the *primary cause* of the phenomena, the *fons et origo mali.* Disturbances of the nervous system are common enough in many disorders universally believed to be due to the presence of some morbific matter in the blood, and it is generally thought that the poison exerts some peculiar action upon the nervous centres. Now, in gout we have a poisonous material circulating with the blood, and that the nervous system should be affected by it is at the least highly probable, judging from our experience of fevers, pyæmia, &c. Dr. Latham's ingenious hypothesis of a "weak spot" in the medulla oblongata is surely unnecessary for the explanation of lithæmia, and we know too little about a "trophic centre for the joints" to be able to trace gouty attacks to primary disorder of that portion of the nervous system. In the condition known as pyæmia we often meet with suppurative inflammation of joints far removed from the original wound. It is highly probable that the nervous system is deeply concerned in these diffused

"metastatic" inflammations; but it is clearly impossible to assume that *antecedent* mischief or "weak spots" in trophic centres determine the incidents and course of the morbid phenomena.

My theory with regard to gout recognises the cause of the disorder in the presence of abnormal quantities of sodium urate in the blood. I am willing to admit that many of the symptoms are indicative of nerve disturbances; but these latter are due to the poisonous action on the nervous centres, of the materials formed in the body, and resulting from imperfect metabolism. The various neuroses which often affect gouty subjects probably depend upon the operation of the same cause. The hypothesis of antecedent nervous lesions is at least unwarranted.

A consideration of the statements contained in the foregoing paragraphs will, I think, justify the following propositions:—

1. Uric acid, in the form of sodium urate, is the *materies morbi* of gout.
2. The deposit of sodium urate in the joints is the cause of the gouty inflammation.
3. This substance is produced in excess, as a result of the imperfect transformation of albuminous substances.

4. This imperfect transformation is for the most part due to functional disorder of the liver, or to excessive supply of nutritive materials, or, as often happens, to a combination of these causes.
5. So long as the excess of uric acid is eliminated by the kidneys, decided attacks of gout may be absent; but the symptoms above described as pertaining to the uric acid diathesis are liable to be present.
6. The kidneys are apt to become *secondarily* affected, owing to the irritation set up by excess of uric acid and other products of defective metamorphosis, and by deposits of urates. Primary disorder of the kidney is not a necessary factor in the production of gout.
7. In the majority of cases of chronic gout increased production of uric acid is associated with defective elimination by the kidneys.
8. The symptoms of nervous disorder in gout are due to the action of the *materia peccans* on the nerve-centres.

CHAPTER IV.

CAUSES OF GOUT.

PREDISPOSING CAUSES — HEREDITY — THE ACQUIRED FORM — GEOGRAPHICAL DISTRIBUTION—SEX AND AGE—CLIMATE AND SEASON—ERRORS IN DIET—EXCESS OF ALBUMINOUS FOOD—WANT OF EXERCISE—DR. CARPENTER ON DIET AS A CAUSE OF GOUT—OTHER ALIMENTARY SUBSTANCES—STARCH, SUGAR, AND FATS—ACIDITY—INFLUENCE OF FERMENTED LIQUORS—LEAD—MENTAL INFLUENCE—DEPRESSING AGENCIES—EXCITING CAUSES OF GOUTY PAROXYSMS.

HAVING discussed various theories as to the nature and *immediate* cause of gout, and stated my own views on these subjects, I proceed to consider what are usually termed the *predisposing* causes of the malady. While regarding gout as in very many cases the result of functional disorder of the liver, it cannot be denied that other factors of a more or less potent character often aid in the production of the disease. I shall therefore devote a few pages to a consideration of the most important of these causes, and also endeavour to trace the origin of the hepatic derangements.

The hereditary transmissibility of gout has been

proved beyond all question, and in this respect the complaint manifestly resembles other forms of perverted nutrition. Hereditary predisposition can be traced in about 55 per cent. of all cases, though probably even this estimate is somewhat too low. While due weight must therefore be allowed to hereditary influence, it is, on the other hand, perfectly certain that gout can be readily acquired. This latter form generally appears later in life than the hereditary disease, symptoms of which are often developed at a very early period. In families in which a gouty taint exists, the disease is apt to show itself most frequently and most severely in the younger children, inasmuch as the dyscrasia generally becomes intensified in the parents with advancing years. In some instances the gouty tendency skips over a generation; the son of a gouty father perhaps suffers only from indigestion, or exhibits marked obesity, but no definite gouty symptoms, whereas his offspring are severely affected by the disease. As a matter of course, the influence of hereditary predisposition may be greatly promoted by the patient's habits of life. With regard to geographical distribution, gout is said to be most common in England and in the southern provinces of Italy. Professor Cantani, of Naples, states that in his country

the hereditary tendency has been handed down from the period of the Greek colonization and the Roman Empire.

It is not surprising to find that women are much less liable to suffer from this disease than men; the proportion of females to males is somewhat less than 3 per cent. Chronic rheumatism, on the other hand, is more frequent among women, and thus differs from gout. It is also worthy of note that in women the attacks of gout usually come on at a later period than in men, unless the hereditary tendency is very strongly marked. Gout very rarely develops itself before the age of eighteen; first attacks are most common between the ages of thirty and forty. It is doubtful whether any particular temperament or constitution can be regarded as a predisposing cause of gout.

With regard to climate it cannot be said that this has any direct effect on the causation of gout. The complaint is almost entirely confined to the temperate regions of the earth, and natives of hot countries (those of Southern Italy excepted) appear to be quite exempt from any of its manifestations. On the other hand, Europeans living in hot climates and indulging freely in animal food and fermented liquors are

almost as prone to suffer as they would be at home. In the palmy days of the East India Company, when high living and copious potations were almost universal, gout was by no means an uncommon disease among the servants of the Company, but the climate had no real share in its production. On the other hand, Dr. Norman Chevers* thinks that "with all its sanitary faults Calcutta is an earthly paradise for those cautiously-living people, whom gout threatens, but has not crippled." It is worthy of note, as showing the influence of season, that first attacks of gout are most common in spring and autumn, and that recurrences are frequent at these periods.

There can be no doubt that errors in diet are the most potent cause both of functional derangement of the liver and also of gout, and that when, as too often happens, deficient exercise is superadded, the development of the gouty diathesis is in many cases only a question of time. Many people habitually take much more food, especially of the albuminous kind, than the system requires; a portion at least of this excess, after conversion into peptone, is absorbed into the blood, and conveyed to the liver. Moreover, the

* *Medical Times*, August 30, 1884, p. 283.

presence in the stomach of an excessive quantity of peptone interferes with the dissolving action of the gastric juice, and imperfectly digested substances are liable to be absorbed. Congestion and enlargement of the liver, a state of general plethora, excessive formation of uric and other acids, and derangement of the eliminative functions of the kidneys are the ordinary consequences. Even after the moderate use of meat, there is increased excretion of uric acid, as compared with the results of fasting, and when albuminates are taken in excess, the increase is very considerable. Many persons who eat immoderately also take an insufficient amount of exercise, and when these two conditions are associated it is difficult to define the share which each plays in the production of disease. The disproportion, however, between the absorbed albuminates and the absorbed oxygen must result in imperfect oxidation, and its consequences; the most important of which are retention in the system of refuse materials, and irritation of the eliminating organs (*e.g.*, the kidneys and skin) by the passage through them of excreta insufficiently prepared for removal from the system. The digestibility of the food is also a very important point in reference to the causation of gout. Rich, highly-seasoned dishes,

by causing indigestion, aggravate the effects of an excessive quantity of food. It must be admitted that large meat-eaters do not invariably suffer from gout, and that they sometimes exhibit no signs of obesity. If a *large* amount of exercise be regularly taken, the excess of albuminates appears to be consumed without causing any functional derangements; and in some cases, where much food is taken, a large proportion is insufficiently masticated and is not absorbed, but discharged with the fæces. Albuminuria is a not unfrequent result of indulgence in excess of albuminous food; some portion of the excess, it may be presumed, being thus got rid of.

In connection with this subject the following observations of Dr. Carpenter* appear to be very apposite:—"It is worthy of remark that in the times when even the wealthy lived during four or five months of the year almost exclusively upon meat, bread, and flour puddings, and when therefore the diet was far too highly azotized, as well as deficient in fresh vegetables, Arthritic, Calculous, and Scorbutic disorders were much more common than at present. The introduction and universal employ-

* "Principles of Human Physiology," ninth edition, p. 100.

ment of the potato has unquestionably done much to correct these two tendencies: on the one hand, by diluting the azotized constituents of the food, so that with the same bulk a much smaller proportion of these is now introduced; and on the other by supplying to the blood some element which is essential to the maintenance of its healthy condition. But with the diminution of the Arthritic diathesis, which the experience of our older practitioners and the medical writings of the last century indicate as having taken place during that period, there has been an increase in the Rheumatic; a change which seems to have a close relation to this alteration in diet."

Opinions differ considerably as to the influence of the other alimentary substances in the causation of gout. Dr. Garrod's* views upon several of these points are somewhat at variance with those usually held, for he appears to think that sugar, for example, inasmuch as it has no influence upon the production of uric acid, may be allowed in cases of gout. It is, on the other hand, *generally* believed that sugar, unless in the most moderate quantities, is almost poisonous to

* Lumleian Lectures, *Medical Times and Gazette*, 1883, vol. i, p. 553. Free use has been made of these lectures in the preparation of several of the following paragraphs.

a gouty subject. This point has an important bearing upon the question of diet, and it is therefore necessary to discuss it somewhat minutely. There are three kinds of sugar met with in food—cane-sugar, grape-sugar or glucose, and milk-sugar or lactose. Cane-sugar when taken into the stomach is converted into glucose, and this substance is very liable to undergo the alcoholic fermentation. Milk-sugar, on the other hand, is much less liable to this change, but in the presence of almost any nitrogenous body it is very prone to undergo conversion into lactic and butyric acids. The fermentation of milk-sugar is especially apt to occur in the presence of cheese. It is a well-known fact that when starchy articles of food are taken, the starch is converted into glucose by the action of the salivary, pancreatic, and intestinal secretions, and it is therefore obvious that amylaceous materials may supply the system with large quantities of sugar. Dr. Garrod doubts whether sugar causes what is popularly termed acidity, and in advising or prohibiting the use of sugar, it is important to discover whether there is any real foundation for the *popular* belief on this point. By some persons lumps of sugar are taken to prevent heartburn, and, as Dr. Garrod points out, it is scarcely credible that a little

cane-sugar would seriously add to the glucose which is daily produced in the alimentary canal of an individual living on an ordinary mixed diet. It is tolerably certain that the use of a large quantity of sugar does not increase the amount of uric acid in the urine; but, on the other hand, the influence of saccharine and starchy materials upon the production of fat is well known. These materials are easily oxidised, and by virtue of this property they delay or prevent the normal disintegration of the albuminous constituents of the body. It has also been supposed that the taking of sugar causes the uric acid to assume a less soluble form, but it has not been proved that the acidity of the urine is increased after the use of sugar in any form. It would therefore seem that the evil effects of saccharine substances in gouty subjects are due in great measure to the fact that they retard metamorphosis. If sugar or starch be taken in excess, the urine often becomes saccharine.

The remark just made with regard to the influence of saccharine and amylaceous food in retarding metamorphosis, applies also to the fatty and oleaginous group. The free use of these substances promotes obesity and prevents waste of tissue, but does not appear to influence in any degree the elimination of

any constituent of the urine. Fatty substances are, however, very prone to undergo butyric fermentation.

The "acidity" so often complained of by gouty subjects is the result partly of the fermentation of food and partly of the increased secretion of gastric juice. The most obvious symptoms of this condition are a sour taste in the mouth, acid eructations, acid reaction of the saliva, and sour odour of the breath. There is no doubt that in many cases the nature of the food greatly influences the degree and character of the acidity. A diet consisting chiefly of vegetables is, as a general rule, much more productive of this symptom than one in which animal food largely preponderates. A reason for this difference could be easily found if it be assumed that by a process of fermentation, glucose becomes converted into lactic acid, the presence of which is the cause of the acidity. Glucose, however, is generally much more prone to undergo the alcoholic than the lactic fermentation, although it is theoretically easy to show that one part of glucose is equivalent to two parts of lactic acid. This latter, again, is readily converted into butyric acid, which, in cases of heartburn, is often abundantly present in the stomach. It is, however, very probable that sugars and starches often undergo lactic fermentation

in the stomach, the gastric mucus, especially when produced in abnormal quantity, acting as a ferment. The albuminous substances are capable of undergoing butyric fermentation, either with or without previous lactic fermentation, and other acids, such as the acetic, succinic, &c., are often developed at the same time.

The next point for consideration, namely, the influence of alcoholic liquors in the production of gout, is one of great importance. These beverages are usually classified under the heads of spirits, wines, and malt liquors. It would seem that alcohol, taken in the form of brandy, whisky, gin, &c., cannot be regarded as a cause of gout, inasmuch as in spirit-drinking countries the disease is almost unknown. The immoderate use, however, of distilled spirits is a common cause of disease of the liver and kidneys; and where the gouty predisposition exists, any habit which tends to damage the kidneys must augment the constitutional disorder. The effect of wine is more easily demonstrated, but it varies very much in degree. Full-bodied wines containing much unfermented matter are potent for evil in persons of a gouty habit, and will doubtless produce the disease in a large proportion of cases in which there is no

gouty taint. The light, well-fermented wines, on the other hand, are not liable to produce gout. The evidence that the disease is often traceable to the use of malt liquor is extremely strong. The occurrence of gout among the poorer classes is, in the absence of hereditary predisposition, almost always due to excessive consumption of beer.

The case with regard to alcohol as a cause of gout may therefore be summed up by saying that distilled spirits and the lighter wines appear to be innocuous in this direction, while malt liquors of all kinds, and the stronger and imperfectly fermented wines (comprising port, sherry, madeira, and champagne), play a very conspicuous part in the causation of gout. We have no definite knowledge as to the mischievous principle; it may be, as Dr. Garrod believes, a something which is the result of imperfect fermentation, for it is certain that those beverages in which fermentation has been prematurely checked are much more liable to produce gout and gravel than those in which the process has been allowed to attain completion. The natural acidity of wines plays a very small part in the production of the gouty diathesis; but that acid wines often excite a paroxysm in gouty subjects is a matter of common observation.

The influence of lead as a predisposing cause of gout is a subject of considerable interest, for there can be no doubt as to the frequency of the coincidence of lead-poisoning and gouty manifestations. In many of the recorded cases it is probable that other factors assisted in the production of the disease; but cases of gout for which no other causes can be discovered are occasionally noticed. It is somewhat curious that in France, where lead-colic is common, gout is a very rare disease, and this fact lends weight to the supposition that the effect of the lead is often much heightened by indulgence in alcoholic liquors. In lead-poisoning the blood is rich in uric acid, while the urine is deficient in this substance. It is doubtful whether there is increased production of uric acid, or defective elimination, but the latter condition would appear to prevail. There is another curious relation between lead-poisoning and gout, for it is highly probable that persons of a gouty habit are more liable than others to suffer from the effects of the mineral. Even in medicinal doses salts of lead are wont to cause very marked symptoms in gouty subjects.

There is one other predisposing cause of gout which deserves a passing notice, and especially

because it is connected with one of the current theories of the nature of the disease. Sydenham found that, in his own case, an attack of gout could be excited by severe study, and in some gouty persons anxiety of mind, grief or annoyance produce the same effect. It is, however, scarcely credible that mental influences alone should suffice for the *development* of the gouty diathesis. Severe study is often associated with sedentary habits and errors in diet, which, as I have just attempted to show, are potent causes of gout. With regard also to other depressing influences, such as exposure to cold, sexual excesses, and the like, these no doubt often excite a paroxysm, and may even aid in the development of the diathesis; but in this latter respect they are not to be compared with the causes already discussed.

A few words as to the *exciting* causes of the paroxysms will conclude this portion of the subject. Experience teaches us that wherever the predisposition exists, almost anything, whether of an exciting or debilitating character, that disturbs the health, is capable of inducing an attack. The most common *exciting* causes are therefore excesses in eating and drinking, indigestible food, articles of diet which

disagree with the patient's idiosyncrasy, exposure to cold, excessive mental exertion, violent bodily exercise, mental excitement, venereal excesses, debilitating diseases, loss of blood, &c. The action of one at least of these causes is very generally recognised. In some gouty subjects pains in the joints, or even an acute attack, are the ordinary results of taking even two or three glasses of port or champagne. In addition, however, to causes of a more or less general nature, it not unfrequently happens that local injuries, whether of a severe or slight character, are sufficient to induce an attack. Thus operations and fractures of various kinds, bruises, sprains, and even the wearing of a tight boot, may act as exciting causes. In gouty subjects very slight local injuries often give rise to unusually persistent and severe pain which is not relieved by ordinary remedies, but subsides under appropriate treatment. Acute gout, moreover, sometimes attacks a joint which has suffered in some previous injury.

CHAPTER V.

IRREGULAR MANIFESTATIONS OF GOUT—VISCERAL AND CUTANEOUS AFFECTIONS.

Irregular Gout—Suppressed Gout—Peculiarities of the Irregular Manifestations—Detection of Uric Acid in the Blood in these cases—Visceral Gout—Functional and Organic Affections—Gouty Diseases of the Throat and Stomach—Dr. M. Mackenzie's Cases of Gouty Throat—Retrocedent Gout of the Stomach—Two Forms: Spasmodic and Inflammatory—Dr. Buzzard on Probability of mistaking Gastric Crises of Tabes for Gout of the Stomach—Question of Metastasis—Experimental Production of the Phenomena of the Uric Acid Diathesis—Gouty Affections of the Heart—Retrocedent Gout—Condition of Heart in Fatal Cases—Fatty Degeneration—Gouty Phlebitis—Gouty Affections of the Lungs: Asthma and Bronchitis—Dr. Stokes' Views—Dr. Greenhow's Cases—Gouty Affections of the Nervous System—Gout retrocedent to the Brain—Neuralgia, Headache, &c., in Gouty Subjects—Gouty Neuritis—Disturbances of the Sensorial Functions—Gout and Eczema—Dr. Piffard's Views on the Connection between certain Skin Diseases and the Rheumic Diathesis.

There is no difficulty in recognising the paroxysms of *regular* gout. The suddenness of the attack, the seat and character of the pain, and the condition of

the joint are sufficient to determine the diagnosis. There are, however, many other phenomena connected with the disease, but of a far less definite character, though due to the same cause, viz., the presence in the blood of excess of sodium urate. I propose to discuss, without unnecessary detail, the most important of these manifestations, to which the term *irregular* may be conveniently applied.

In former times a vast number of symptoms were referred to gout. At the present day a very different tendency prevails, and we hear more about "neurotic conditions," "trophic nerves and lesions," and the like. Some modern authorities indeed assert that the term "gout" is very often a refuge for ignorance; implying that when no other cause can be discovered the phrase "suppressed gout" is clutched at in desperation, in order to evade a serious difficulty. There is probably some amount of truth in this insinuation, but at the same time it is not unreasonable to assume that the same cause or causes which give rise to *regular—i.e., articular*—gout are capable of producing certain effects or symptoms in parts of the body other than the articular structures. It must, I think, be admitted that a high degree of probability is attached to this supposition, and that it is perfectly justifiable to speak

of *irregular* manifestations of gout. It is a matter of common experience that certain symptoms, such as those already mentioned as connected with the uric acid diathesis, often precede the acute attack, disappear on its occurrence, and again become troublesome when the articular symptoms have subsided. We also find that many patients exhibit symptoms of the diathesis, but never suffer from decidedly acute attacks, and there is no escape from the conclusion that both classes of manifestations are due to the same cause. It is not of course contended that all symptoms which may occur, even in decidedly gouty subjects, are necessarily due to the diathesis; but it is extremely probable that a gouty taint modifies the course and symptoms of any disorder which affects its subject.

In cases at all urgent, before arriving at a conclusion with regard to symptoms suspected to be due to gout, it is well to adopt Dr. Garrod's suggestion and examine the blood-serum obtained from a small blister in the manner already described. If crystals of uric acid are discovered it is highly probable that the suspicion is correct. As a matter of course, the age, sex, previous history, habits of life, and other circumstances must be duly considered. The previous occurrence of an acute attack of gout is clearly

suggestive of the causation of anomalous symptoms, subject to the caution given in the preceding paragraph.

In my first chapter I cited Cullen's classification of the various forms of gout. The terms *atonic* and *wandering* are at the present day almost obsolete; we now speak of visceral gout, which includes *misplaced*, *retrocedent*, and *atonic* forms. This collective term is applied to designate all symptoms having their seat in internal organs, and presumably due to the influence of the gouty diathesis. The visceral affections are again further subdivided into functional and organic. An important addition must, however, be made to this category, for, as I shall presently endeavour to show, certain skin-affections are closely connected with the gouty diathesis, and may, indeed, be reckoned among its most important manifestations. In the present chapter I propose to deal with these affections and with gouty disorders of some of the thoracic and abdominal viscera, leaving, however, gouty diseases of the liver and kidneys for a subsequent chapter.

Symptoms of *visceral* gout may precede the articular inflammation, may co-exist with it, or may suddenly appear on its subsidence. Many of the symptoms of the uric acid diathesis (see page 45) may be regarded as those of visceral gout in an incipient stage. Gouty

disorders of the *digestive organs* are common precursors of an attack of gout, and in my experience often occur alone, that is, without any decided articular affection. The throat is not unfrequently affected in gouty patients, the symptoms of angina sometimes alternating with attacks of inflammation of the joints. Dr. Morell Mackenzie* has recorded several well-marked instances. "In one case, a gentleman who frequently suffered from attacks of angina became subject to gout, and was never again attacked with inflammation of the throat. In another case, the patient was suffering from acute pharyngitis, when the symptoms suddenly disappeared, and an acute attack of gout developed in the great toe of the right foot; after three days the gouty inflammation of the toe disappeared and acute hyperæmia of the pharynx supervened."

Dyspepsia in various forms—acidity, flatulence, irregular action of the bowels and hæmorrhoids, are common symptoms in gouty subjects and in persons in whom the hereditary tendency is well marked. Their connection with gout is rendered probable, first by their subsidence on the occurrence of an acute

* "Diseases of the Throat and Nose," vol. i, p. 48.

attack, and, secondly, by the manner in which they are influenced by regimen and medicines suitable to the gouty diathesis. Charcot records a case which illustrates the first of these characteristics. A patient, who had suffered from articular gout, became a martyr to dyspepsia, and, after vainly invoking the aid of regular science, had recourse to homœopathy. The dyspepsia rapidly subsided and the patient was congratulating himself on his change of advisers when a sharp attack of articular gout served to explain the supposed cure.

More important, in some respects, than this *misplaced gout* of the stomach are those attacks to which the term *retrocedent* is commonly applied. It is impossible to suggest any really satisfactory explanation of the so-called "metastasis," but cases undoubtedly occur in which violent gastralgia, vomiting, prostration, or even death, abruptly supervene upon the subsidence of the symptoms of articular gout. In some cases the metastasis is spontaneous; in others, it appears to result from attempts to relieve the articular inflammation by local remedies, or from exposure to cold or other injurious influences, during the course of an acute attack. In a case which came under my notice, a gentleman, aged forty-four, suffer-

ing from acute gout, applied cold wet cloths to the joint. Symptoms of acute gastritis rapidly supervened, and were followed by syncope and death in seventeen hours.

Cold applications to the affected joint or placing the foot in cold water will often relieve the local symptoms, but at the cost of serious gastric or cardiac disorder, and a full dose of colchicum has also been known to produce similar results.

Gout thus affecting the *stomach* presents at least two forms, which may be termed respectively *spasmodic* and *inflammatory*. In the former, which may be regarded as a functional affection, there is violent spasm or cramp in the epigastric region, accompanied by a feeling of intense oppression and distension, nausea, vomiting, difficulty of breathing, and palpitation of the heart. There is also great prostration; the skin is cold and clammy, and the pulse is feeble, frequent, and irregular. There is, however, no severe burning pain, and the cramp is relieved by pressure and stimulants. In the inflammatory form, the symptoms are those of acute gastritis. There is severe epigastric pain of a burning character and much aggravated by pressure, nausea and vomiting, and more or less febrile disturbance, and the ejected matters sometimes contain blood. The pulse is at first

full and strong, but soon becomes feeble and irregular. Stupor is apt to supervene, and the patient becomes almost insensible; but slight pressure upon the epigastrium calls forth evidences of severe pain. These symptoms, like those of the spasmodic form, may either suddenly subside, or terminate in death. Recurrence of the articular inflammation is sometimes simultaneous with the abatement of the gastric symptoms.

Certain phenomena much resembling those of acute gouty disorder of the stomach are not unfrequent in cases of locomotor ataxy. Dr. Buzzard,* indeed, in his excellent Lectures, suggests "that many cases of so-called 'gout in the stomach' would be found, if examined by the light of our present knowledge, to be examples of tabes dorsalis, with gastric crises. The lightning pains, which would probably be associated with the sickness and epigastric pain, would be likely in the minds of many to make the diagnosis of 'gout in the stomach' complete." Severe gastric attacks, it must be remembered, are sometimes witnessed in persons who, although examples of tabes dorsalis, "show at the time no sign of inco-ordination of movement." Their persistence and frequent recur-

* "Clinical Lectures on Diseases of the Nervous System," p. 272.

rence would suffice to exclude gout as a cause, and if accompanied by absence of patellar tendon-reflex and by other indications of tabes, there ought to be no difficulty in the diagnosis.

When gout retrocedes to the intestines, the symptoms take the form of diarrhœa or colic. These organs, however, are far less subject to gouty influences than the stomach, and very few trustworthy reports of gouty enteritis or colic, due to metastasis, are to be found in medical literature.

This subject of metastasis, or transfer of diseased action, is involved in much obscurity, and not a few authorities assert that symptoms such as those above described are capable of a very different explanation. The old notion of metastasis was to this effect—a local irritation, due to some permanent general diathesis, becomes cured or subsides, but throws itself on some other part in which it excites symptoms of disordered action or of inflammation. That this is the *apparent* course of events there can be no doubt, but as to the manner in which the irritation is propagated we can only speculate. It is very probable that in many instances of supposed metastasis, the occurrence, *e.g.*, of gout in the stomach, was a mere coincidence, and that the symptoms were only those of severe

indigestion, to which gouty subjects are especially liable. Chronic gastritis is a common condition in these patients, and the symptoms of gastric disorder are easily aggravated, and become prominent under the operation of comparatively slight causes. Admitting, however, that in some cases the phenomena of retrocedent gout, or gout of an internal organ, are due to a previously existing affection, it is going too far to assert that all instances of retrocedence can be thus explained. Charcot points out that phenomena analogous to those of the uric acid diathesis can be experimentally produced in animals, and that in such cases the gastric juice and the follicles of the stomach are loaded with sodium urate. He thinks that, without assuming the existence of an identical condition to account for the symptoms of stomachic gout, superficial lesions might be easily caused in the digestive system under the influence of retrocession. However this may be, "it is probable that in the long run, permanent lesions are produced in the cases in which these manifestations occur, which seem so purely functional."*

Of the organs of the *thorax*, the *heart* often becomes

* Charcot, "Lectures on Senile Diseases," New Syd. Soc. Trans., p. 85.

affected in cases of gout, and the disorder may be either functional or organic. Functional disorder is evidenced by palpitation, precordial oppression, more or less dyspnœa, and irregularity of pulse. These symptoms are in many cases merely those of dyspepsia, or of an overloaded stomach, and are in no way characteristic of gout. They, however, frequently accompany the development of the uric acid diathesis, and, in some cases at least, are probably due, not directly to indigestion, but to the action upon the heart of the sodium urate contained in the blood. These cardiac symptoms not unfrequently subside when the acute attack supervenes and subsequently reappear, thus alternating with the joint-affection.

In considering retrocedent gout affecting the heart, the difficult question of metastasis again comes before us. Cases, however, now and then occur in which the morbid action *appears* to be transferred from the joint to the heart. I have already referred to one case in which gastric symptoms were followed by fatal syncope. In one recorded case a patient suffering from severe articular gout applied snow to the affected part. The pain was relieved, but marked symptoms of syncope came on, and the patient was for some time in a very critical state. In another case, under the

care of Dr. Garrod,* the cardiac symptoms followed exposure to cold east winds at a time when the patient was recovering from an acute attack. There was violent pain across the chest and down both arms, with faintness and a sense of alarm. The symptoms, after partial subsidence, recurred several times, and a few days after their final disappearance the foot again became affected. "There was no evidence of structural disease of the heart, and no appreciable febrile disturbance."

Whatever may be the true explanation of these and similar cases, the condition of the heart, when death occurs from retrocedence of gout to that organ, is always sufficient to account for the symptoms and the result. Fatty degeneration or fatty deposit, with or without valvular disease, and dilatation, are invariably present. An increased amount of fat on the surface of the heart, accompanied by atrophy of the muscular tissue, is common in gouty persons presenting external signs of obesity. In all these conditions, shortness of breath, or a sense of choking or suffocation, a tendency to faintness, especially on exertion; a feeble cardiac impulse, and a small, slow, and ir-

* "Gout and Rheumatic Gout," third edition, p. 440.

regular pulse are usually more or less prominent symptoms, but it is well known that these are sometimes absent in cases in which extensive degeneration is discovered after death. In other cases there are symptoms of angina pectoris, which in gouty subjects is almost always accompanied by organic changes.

In these cases of fatty degeneration of the heart, rupture of the organ is the most frequent cause of a suddenly fatal termination. Death may also occur from syncope, without rupture. If it be admitted that retrocedent gout may give rise to symptoms of cardiac disturbance in the absence of structural disease of the organ, it is only reasonable to expect that the same cause would produce much more serious consequences when organic lesions are present. It is not necessary to assume that gout sets up any kind of inflammation of the heart, but those factors which are most potent in the development of gout tend also to produce fatty changes in the organ. Gout, moreover, as I shall presently attempt to show, plays an important part in the causation of Bright's disease, and is thus again connected with cardiac disorders. Gout also appears to be one predisposing cause of atheroma, which, when it affects the coronary arteries, often gives rise to fatty degeneration.

In connection with the circulatory organs, gouty *phlebitis* requires to be briefly noticed. Inflammation of the veins, especially in the lower limbs, is somewhat liable to occur in gouty subjects, and either with or without acute attacks in the joints. The superficial veins are those which are most frequently affected; the inflammation begins very suddenly, and causes severe pain and tenderness. It may spread continuously, and may appear simultaneously in several veins. It sometimes disappears and affects other veins at a distance. Sometimes the deep veins are affected, in which case the limb becomes œdematous and distended. The inflammation is apt to recur, and hence recovery is often tedious. Symptoms of embolism have been known to supervene, with death from obstruction of the pulmonary artery as a result. Sir James Paget* says that gouty phlebitis is often hereditary, and that common phlebitis is often traceable to the gouty diathesis. He thinks that gout should always be suspected whenever phlebitis occurs without evident external cause in an elderly person. Dr. Owen Rees thinks that gout is essentially a *capillary phlebitis*, and that this theory best explains

* "Clinical Lectures and Essays," p. 300. See also Sir P. Hewett's Address in Clin. Soc. Trans., 1873.

the phenomena of the acute paroxysm, the venous inflammation being caused by the circulation of a blood-poison.

If we turn to diseases of the respiratory organs, we find that *asthma* and *bronchitis* are often connected with the gouty diathesis. In some cases of gouty asthma, there is no organic affection of the respiratory apparatus, while in others bronchitis or emphysema co-exists. The connection between the asthma and the diathesis is shown by the fact that the former either subsides or remits on the development of articular inflammation.

There is still stronger evidence in support of the connection between gout and *bronchitis*. When we consider that many specific disorders are frequently accompanied by affections of the bronchial mucous membrane, it is not to be wondered at that a disease like gout should be liable to the same complication. I have witnessed the symptoms of acute articular gout disappear on the supervention of bronchitis. Crystals of uric acid have been detected in the sputa of a gouty patient,* and scarcely any stronger evidence could be offered of the occurrence of gouty bronchitis. There

* See note by the late Dr. Hudson on page 81, New Syd. Soc. edition of Stokes "On Diseases of the Chest."

is every reason to believe that all the excretory organs take a share in the elimination of morbid materials, and that catarrh or inflammation is a frequent result. The proof that the bronchial symptoms are connected with gout is found in the fact that they often precede the attack and rapidly subside on its appearance, again to recur when the articular inflammation abates. In the case of a lady aged seventy, recently under my care, attacks of acute gout had been very frequent for some years; bronchitis supervened and the gouty attacks diminished in number and severity. Dr. Todd* has recorded a case in which obstinate and severe bronchitis at once subsided on the appearance of gout in the foot; and Dr. Stokes alludes to cases in which bronchial symptoms having been removed by appropriate treatment, an attack of gout immediately appeared. He also refers to more complicated cases, such as the succession of epilepsy, gout, and fatal bronchitis; inflammation of the trachea, slight general arthritis, glandular enlargements, and gout. He doubts whether in such cases, the anatomical characters of the lesion are different from those of ordinary bronchitis, and thinks that "its specific character will be

* "On Gout and Rheumatism," p. 38.

found more in its mode of invasion and amenability to certain remedies than in its anatomical nature or seat." Valuable clinical evidence with regard to the relation between a gouty constitution and bronchitis has been adduced by Dr. Headlam Greenhow.* He has given details of many interesting cases showing the frequency of gout among bronchitic patients; the frequent co-existence of gout and bronchitis in the same families; the alternation of the two affections in the same persons, and the frequent association of psoriasis, eczema, and gravel with bronchitis. Pleurisy and pneumonia are not unfrequent in gouty subjects, but it is doubtful how far the diathesis is concerned in their development.

Severe and well-marked gouty affections of the *nervous system* are of less frequent occurrence, but retrocedent gout sometimes attacks the brain and its membranes. The supervention of cerebral symptoms upon the sudden disappearance of gout from an extremity has been observed by Dr. Garrod, and Niemeyer mentions two cases of gouty subjects presenting severe symptoms referable to circumscribed inflammation of the meninges. In one case the

* "On Bronchitis and the Morbid Conditions connected with it," second edition, p. 128, *et seq.*

symptoms disappeared simultaneously with a copious excretion of urates by the kidneys; and in the other, with an attack of gouty inflammation of the joints. Retrocedence of gouty inflammation, giving rise to apoplexy, has been known to follow the application of cold to a gouty joint, but as in the analogous case of the heart, it is probable that in the majority of instances, more or less severe organic disease had previously existed in the part. Epilepsy would appear in some cases to be connected with gout, the convulsions ceasing after the development of acute arthritic attacks.

There is much reason for believing that many slight affections of the nervous system are referable to the presence of the gouty diathesis; the term *nervous* gout has indeed been applied to the entire class of irregular gouty manifestations. Nervous affections of this character occur especially in women, and in individuals generally of a nervous temperament and descended from gouty ancestors. In some families the male members are the victims of acute gout, while the females suffer from neuralgia in various forms, headache, &c. As in other cases, the connection of any obscure symptoms with gout may be suspected when there is a history of the disease in

the family and no other cause can be detected. When the symptoms alternate or co-exist with slight attacks of articular inflammation, there can be little doubt as to their origin. Cases are sometimes met with in which patients, once the subjects of frequent articular attacks, become after a time exempt from them, but suffer instead from neuralgia, headache, sleeplessness, vertigo, dizziness, and other symptoms of nervous disorder. Neuralgia is the most common symptom of nervous gout, and it most frequently appears in the form of hemicrania or megrim. In the case of a lady, aged sixty-seven, whom I saw with Sir William Jenner, gout and diabetes co-existed, and the patient suffered also from occasional attacks of megrim, with distressing sickness and intolerance of light. Dr. Liveing thinks that there can be no question "as to the frequent connection of megrim, whether in its blind, sick, or simply hemicranial forms, with a gouty diathesis, and its occasional replacement by fits of regular gout. Megrim, however, is far from being the only neurosis which is thus associated with gout."* As stated in a previous chapter (see page 52), Dr. Liveing is inclined to think that gout in

* "On Megrim, Sick Headache, and some Allied Disorders," 1873, p. 404.

its various forms is the manifestation of a disorder which has its primary seat in the nervous system itself.

Other forms of neuralgia are not uncommon in gouty subjects; sciatica and facial neuralgia, for example, sometimes alternate with articular gout. We may assume that the pain is due to hyperæmia and œdema of the neurilemma, but why only certain branches of a nerve should be affected as a result of the constitutional disorder is a question which cannot be solved. Dr. Buzzard has pointed out that the "lightning-like" pains of tabes are not unfrequently ascribed to gout or rheumatism. It is probable that gout is for the most part only a predisposing cause of neuralgia, and that the excitement of the pain is due to the operation of other causes. Dr. Graves was of opinion that "gouty inflammation of the nerves and their neurilemma may in process of time extend to the spinal marrow and its investments, and give rise to derangements terminating in *ramollissement* and structural degeneration." He has described several cases of gouty paralysis.*

* For evidence in support of the view that inflammatory processes in nerves may be propagated to the cord, see Leyden's essay on "Reflex Paralysis," German Clinical Lectures, New Syd. Soc., p. 163. Also Nothnagel's essay on Neuritis in the second series of Lectures.

Various disturbances of the sensorial functions often occur in gouty subjects and take the form of vertigo, dizziness, tinnitus aurium, perversions of vision, &c. I am now attending a gouty subject in whom vertigo is very marked and troublesome. There is, of course, nothing peculiarly characteristic in these symptoms of nervous irritation, for they are common indications of dyspepsia. Sometimes, however, they alternate with sub-acute attacks of articular gout, subsiding when the inflammatory symptoms are prominent, and recurring shortly after their disappearance. Under these circumstances their connection with the gouty diathesis would appear to be unquestionable. Other manifestations of nervous disorder in gouty subjects, such as cramps in different parts of the body, hysteria, hypochondriasis and maniacal attacks, may, in some cases at least, be placed in the same category.

I have already referred to the fact (see p. 47) that eczema often alternates with symptoms referable to gout, and I have found the association to be of such frequent occurrence that I regard the skin-affection in these cases as a decided manifestation of the gouty diathesis. This view is advocated by many authorities; among others, by my friend Mr. Wyndham Cottle, who informs me that he considers the eczema, in common

with the other irregular symptoms, as but a local manifestation of the same constitutional state. Dr. Piffard,* Professor of Dermatology in the University of New York, has entered very fully into this subject, and his remarks are so consonant with my own observations that I venture to quote them. In opposition to the Vienna school, he believes in the existence of a certain cutaneous diathesis, which he designates as *rheumic*, this being equivalent to the *dartrous* diathesis of the French, and the *predisposing* cause, he assumes, of eczema, psoriasis, and pityriasis. This diathesis also corresponds with the condition known in England as *lithæmia*, or, in other words, with the uric acid diathesis. The state of the blood in lithæmia has been already fully described, and it is a remarkable fact that uric acid has been detected in the scales and excretions of the skin-affections just mentioned,† and that, as described by Dr. Garrod, the administration of uric acid internally has been followed by an

* A Treatise on the Materia Medica and Therapeutics of the Skin," 1881, p. 126, *et seq.*

† In several cases of patients lying bedridden from rheumatic gout, in whom one or both legs were covered with an eczematous eruption, the late Dr. Golding Bird observed microscopic crystals of sodium urate scattered like fine hoar-frost on the parts on which the surface-exudation had dried.

eczematous eruption.* It would therefore seem that these cutaneous affections are really due to accumulation in the blood of certain excrementitious substances, of which uric acid is the one most easily demonstrable. Dr. Piffard thinks that the category of noxious agents includes also lactic acid, oxalic acid, creatin, and creatinin; all of these representing either steps or side-products of the processes which bring about the metamorphosis of food into tissue, and that again into substances ready for excretion. If we regard the normal process as one of oxidation, the production in excess of these excrementitious materials may be assumed to result from sub-oxidation.

So long as these products are removed by the kidneys, there may be no evidences of any injurious action; but when the excess becomes too great, or when the eliminative functions of the kidneys are imperfectly performed, the work is thrown upon other organs, and especially upon the skin, and the result in many cases is an outbreak of eczema, psoriasis, or pityriasis. These affections obviously present a mutual relationship, and possess many

* Lumleian Lectures, *Medical Times and Gazette*, vol. i, 1883, p. 318.

features in common, and they often alternate not only with affections of the joints, but with symptoms referable to the pulmonary and gastric mucous membranes. Psoriasis and eczema are often hereditarily transmitted, and in some cases they appear to be the only manifestation of the gouty diathesis which is thus handed down. According to Dr. Garrod, prurigo must be added to this list. As additional evidence of the connection between these skin-affections and gout, it should be mentioned that they are almost invariably aggravated by causes which increase the production of uric acid; whereas they are improved by constitutional treatment and regimen, but either not influenced at all, or else made worse by ordinary remedies. They are, moreover, very liable to recur so long as the original diathesis remains unchanged.

CHAPTER VI.

HEPATIC AND RENAL DISORDERS CONNECTED WITH GOUT.

HEPATIC CONGESTION FREQUENT IN GOUTY SUBJECTS—INFLAMMATION OF THE BILIARY DUCTS, JAUNDICE, &C.—GOUTY CIRRHOSIS OF THE LIVER—QUESTION AS TO ITS CAUSATION—INFLUENCE OF OTHER SUBSTANCES BESIDES ALCOHOL—DR. BUDD'S VIEWS — BILIARY CONCRETIONS IN GOUTY SUBJECTS—RENAL DISORDERS DUE TO IMPERFECT ASSIMILATION AND HEPATIC DERANGEMENT — ALBUMINURIA AS A SYMPTOM—QUESTION AS TO PHYSIOLOGICAL ALBUMINURIA—DR. SENATOR'S VIEWS—DIGESTION-ALBUMINURIA—INFLUENCE OF ALBUMINURIA UPON THE KIDNEYS — QUESTION AS TO INDICATION OF RENAL MISCHIEF WHEN "DIGESTION-ALBUMINURIA" IS OBSERVED—THE "GOUTY KIDNEY" PROPERLY SO-CALLED, VARIETIES OF—ASSOCIATION OF INFARCTIONS OF URATES WITH RENAL CIRRHOSIS — SYMPTOMS OF GOUTY KIDNEY — GRAVEL, CALCULUS, BLADDER-AFFECTIONS, AND DIABETES IN GOUTY SUBJECTS.

THE part taken by functional disorders of the liver in the causation of gout has been fully described in preceding chapters. Following out the view, so forcibly advocated by the late Dr. Murchison, that *lithæmia* is very often due to functional hepatic derangement, I have sketched, somewhat minutely, the symptoms and

causes of the uric acid diathesis, of which gout is to be regarded as a further development. I have stated my belief that a normal condition of the structure of the kidneys generally obtains during the early stages of gout, and that gouty disease of these organs is a consequence of the irritation to which they are subjected during the elimination of the excess of sodium urate, and other products of defective metamorphosis. It must of course be admitted that renal disease of independent origin sometimes exists previously to any gouty manifestation, and that defective capacity of the kidneys will promote the development and accelerate the course of the disorder. I now propose to consider those hepatic and renal diseases which, so to speak, follow in the train of gout, and may be regarded as more or less direct effects of the dyscrasia.

That the liver is rarely healthy in gout is a point of common observation; but it is at least doubtful whether there are any organic affections of this organ directly chargeable to the gouty diathesis. I have already referred to the symptoms of hepatic congestion often present in gouty subjects; but those are for the most part of a temporary character, are much relieved by proper diet and treatment, and frequently disappear when the articular inflammation comes on.

On the other hand, a paroxysm of gout is sometimes attended by bilious vomiting and purging and other signs of hepatic disorder. Dr. Murchison states that adult persons of a gouty constitution are especially liable to attacks of inflammation of the *biliary ducts*, generally as a result of extension of catarrh of the stomach and duodenum, and preceded by symptoms referable to these parts. In some cases the symptoms of inflammation of the biliary ducts become very severe; the frequent vomiting, the emaciation, and the persistent jaundice have sometimes led to the suspicion of cancer. Under treatment by purgatives, colchicum, and alkalies, the symptoms gradually pass off. This affection is generally noticed in persons descended from gouty ancestors, but presenting no decided manifestations beyond occasional twinges of pain in some of the smaller joints. In other instances the jaundice and other symptoms referable to the liver appear on the subsidence of an acute attack of articular gout. A case of this kind has recently come before me. A lady, aged forty-four, of gouty family history, had had several acute attacks in the right toe-joint; during the intervals she had suffered much from flatulence and other symptoms of dyspepsia. When called in to see her, I found her

suffering from jaundice and considerable pain in the hepatic region. A week previously an acute attack in the joint had suddenly abated, in consequence (as she thought) of a chill, and the hepatic symptoms appeared two days afterwards.

A gouty form of cirrhosis of the liver has been supposed to occur, but the evidence on this point cannot be regarded as quite conclusive. The two conditions not unfrequently co-exist; but their union in such cases is probably due to the operation of the same causes. Trousseau's description of *gouty chronic hepatitis* is exactly applicable to ordinary cirrhosis. He states that it "is characterised by pains in the right hypochondrium; by an increase or diminution in the volume of the liver, rendered appreciable by palpation and percussion; by jaundice, or at least by a sub-icteric tint of the skin. At the autopsy, the substance of the organ is often found exceedingly hard, granular, like cirrhosis, and (according to Lieutaud) charged with calcareous concretions."* The excessive use of alcohol, especially in the form of raw spirits, is universally recognised as the *ordinary* cause

* "Lectures on Clinical Medicine," New Syd. Soc. Trans., vol. iv, p. 381.

of cirrhosis; but Dr. Budd* has suggested "that there may be other substances, among the immense variety of matters taken into the stomach, or among the products of faulty digestion, which, on being absorbed into the portal blood, cause, like alcohol, adhesive inflammation of the liver." This view is certainly deserving of consideration. It is well known that a temporary enlargement of the gland occurs during digestion, and is relieved by an increased secretion and flow of bile. When the congestion frequently exceeds the normal limit (whether the cause be alcohol or articles of food of an irritant nature) it may easily become the starting-point for structural disease. It would seem therefore not improbable that, just as gout may arise from over-supply of food, with little if any excess of alcohol, so hepatic cirrhosis may sometimes own a similar origin. The fact that this disease has been observed in animals (the cow and pig) is sufficient to prove that it is not always due to the use of alcohol.

Biliary concretions are not often noticed in gouty

* "Diseases of the Liver," third edition, 1857, p. 151. Professor Thierfelder doubts whether cirrhosis of the liver ever results from gouty dyscrasia on the ground that the complaint is not common in gouty subjects, and is not attended by deposits of uric acid in the diseased organ. Ziemssen's Cyclopædia, vol. ix, p. 172.

subjects. Dr. Prout, however, thought that a tendency to the formation of gall-stones of cholesterin is frequently associated with a tendency to lithic-acid deposits in the urine. This view was supported by Dr. Budd (p. 369). "It is probable that in London the habit of drinking porter, which frequently leads to lithic-acid deposits and to the most inveterate forms of gout in persons who inherit no disposition to them, may also frequently lead to the formation of gall-stones." The fact appears to be that gout and biliary calculi often own a similar causation, *e.g.*, errors in diet, sedentary habits, &c. It has been stated that uric acid is sometimes found in gall-stones, and, if such were actually the case, we might infer that a close relation existed between such concretions and gout. Frerichs, however, states that "uric acid has been found in abundance in a cylindrical concretion described as a gall-stone, but there were some doubts as to the place of origin of this concretion." Stöckhardt and Faber some years ago discovered uric acid in concretions of the same nature. It must not, however, be forgotten how readily concretions in collections are confounded with one another, and wrongly described.* Frerichs thinks

* "Diseases of the Liver," vol. ii, New Syd. Soc. Trans., p. 497.

that the co-existence of biliary concretions with urinary calculi must be regarded as an entirely accidental circumstance. It is certain that no *special* diathesis is associated with the formation of gall-stones, for they occur in persons of extremely various constitutions, and more frequently in women than in men.

I now proceed to consider those renal disorders which are directly due to the gouty dyscrasia, and I shall first endeavour to trace the influence which imperfect assimilation and hepatic disorder exercise upon their development.

The connection between the urinary secretion and disorder of the functions of the liver is shown by the frequent occurrence of albuminuria in certain cases of hepatic derangement. I have noticed this coincidence in not a few patients, men in middle life, of active literary habits, but somewhat free livers. These cases exhibit the ordinary symptoms of gouty dyspepsia, as already described, but no acute attacks of articular inflammation; more or less severe eczema is almost always complained of. The albumen occurs sometimes in very minute quantities, such as one-tenth per cent., while in other cases as much as three per cent. is deposited. No tube-casts are discoverable, and the albumen disappears in the course of a few weeks

under the use of suitable diet, purgatives, and alkaline treatment. In no cases of this kind should I think it necessary to prescribe colchicum. I believe that this appearance of albumen in the urine is due to defective metamorphosis of nutritive materials by the liver, and I have little doubt that temporary albuminuria not unfrequently occurs in the absence of any organic disease of the kidney.

Not many years ago it was commonly believed that the occurrence of albumen in the urine was a decisive proof of the existence of organic disease of the kidneys. The discovery that albuminuria could be induced by eating a large number of eggs was sufficient to raise doubts as to the truth of this view, and we now have reason to believe not only that albuminuria may occur in the absence of *renal disease*, but that traces of albumen so often occur in normal urine that this substance may almost be regarded as a regular constituent of that secretion. Cogent evidence in support of this latter view has recently been offered by Professor Senator,* of Berlin. The point is one of great importance, inasmuch as the discovery of

* "Albuminuria in Health and Disease," New Syd. Soc. Trans., p. 15, *et seq.*

albumen in the urine naturally gives rise to the *suspicion* (and probably often to the *conviction*) that the patient is suffering from some form of Bright's disease. Dr. Senator states that he examined for a lengthened period and at different hours of the day the secretion of four persons, all in excellent health, and at one time or other discovered albumen in the urine of each, certainly only in faint traces, which might well have been overlooked had not delicate tests been employed. "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." Dr. Senator cites Leube's* statement to the effect that he found albumen in the urine in 19 instances among 119 healthy soldiers, and also that of Dr. Munn,† who discovered the same condition in 24 out of 200 apparently healthy persons who came before him to be examined for purposes of life-assurance. From these and many other similar experiences, references to which will be found in Dr. Senator's treatise, it would appear

* Virchow's "Archiv.," lxxii, S. 145.

† *New York Medical Record*, March 29, 1879.

warrantable to assume that albumen is often present in normal urine, but only seldom in quantities appreciable by ordinary tests. Dr. George Johnson,* however, while admitting that albuminuria frequently occurs in persons *supposed* to be healthy, maintains that even the occasional presence of the *smallest trace* of albumen is always of pathological import. He alludes to cases in which some obvious cause could be detected, *e.g.*, a previous history of acute nephritis, exposure to cold and wet, as in cold bathing, excessive consumption of food and alcohol, &c. In Dr. Senator's cases, however, there were no histories of this kind.

It would be foreign to my immediate topic to discuss at greater length this subject of physiological albuminuria: I have alluded to it because it is closely connected with another condition which has been termed the "albuminuria of digestion," and often noticed after the use of large quantities of albuminous food. It is doubtless this form of albuminuria which is often present in cases of gouty dyspepsia; some of the albuminous constituents of the food not being converted into urea, and passing out of the body in an unchanged state. The albumen transudes through

* "Latent Albuminuria," *British Medical Journal*, 1879, vol. ii p. 928.

the vessels of the glomeruli, and is not a product of the secretion of the glandular epithelium. As a matter of course its presence is most readily detectible when the urine is scanty and somewhat concentrated, conditions which frequently obtain in these cases of gouty dyspepsia. The subjects of this ailment drink, as a general rule, an insufficient quantity of fluid in the form of water, the place of which in the economy cannot be taken by any other liquid. An insufficient supply of water diminishes the amount of all the important urinary excreta, but facilitates the appearance of "digestion-albuminuria," and especially when at the same time meat and other albuminous foods are taken in excessive quantities. This excretion of albumen, if of frequent occurrence, is very liable to cause irritation of the kidneys. Dr. Senator points out that when egg-albumen as such "finds its way into the blood, it is excreted by the kidneys; but frequently this is not all that happens, for, as 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."* It is worthy of notice, as corroborating the views just expressed, that the albuminuria of Bright's disease is always increased after the use of animal food.

A consideration of the facts adduced in the preceding paragraphs will, I think, justify the conclusion that the defective assimilation which exists in gout may ultimately result in irritation and chronic inflammation of the kidney, and may therefore be regarded as one cause of Bright's disease. Dr. Murchison has strongly advocated this view. He states that when the attack (of acute Bright's disease) follows a chill, and there is no history of scarlatina, "it will almost invariably be found that the patients have previously suffered from derangements of the liver with lithæmia, while many have led intemperate lives. Again, we find that functional derangement of the liver resulting in lithæmia, with dyspeptic symptoms such as those which I have described, is a common cause of the contracted, granular, or gouty kidney."

* "On the Hygienic Treatment of Albuminuria," New Syd. Soc. Trans., p. 143. See also a paper by Drs. Brunton and Power, "On the Albuminous Substances which occur in Albuminuria," St. Bartholomew's Hospital Reports, 1877.

Dr. Dickinson, in the concluding portion of his excellent work on Renal Disorders, has expressed the opinion that the albuminuria of dyspepsia is in all probability indicative of renal mischief. "It has been stated that the urine has become temporarily albuminous as the consequence only of dyspepsia; but, knowing as we do how often inconstant or periodic albuminuria together with dyspepsia is the accompaniment of an early stage of the granulating kidney, particularly when this is connected with gout, we can but suspect that when the urine has become albuminous with symptoms of indigestion, the kidneys may not have been perfectly sound."* With regard to this opinion, it must be admitted that the discovery of albumen in the urine is always sufficient to raise apprehensions of renal lesions; but, when the quantity is small and there are no casts, when there are decided symptoms of gouty dyspepsia, and lastly when the albumen entirely disappears under proper treatment, I cannot but think that we are justified in believing that the kidneys are still sound. If, on the other hand, the albumen continues, it may not only be indicative of disease, but, as already mentioned, its

* "On Renal and Urinary Affections," Part III, 1885, pp. 1268–9.

passage through the kidneys will serve to intensify the condition primarily due to its elimination.

I have met with several cases of Bright's disease in men over fifty, of decidedly gouty tendencies, but never having suffered from acute gout. Dr. George Johnson thinks that the granular kidney is often associated with the gouty diathesis, and "is of common occurrence in persons who eat and drink to excess, or who, not being intemperate in food and drink, suffer from certain forms of dyspepsia without the complication of gouty paroxysms." After describing the condition of the urine (which is at first high-coloured, scanty, and deposits urates, and afterwards pale, more copious, and albuminous), he goes on to say, "in such a case probably renal degeneration is a consequence of the long-continued elimination of products of faulty digestion through the kidneys."* Functional disorder of the stomach and liver, as occurs in gout, may thus result in secondary disturbance of the kidney.

Sufficient evidence has, I think, been adduced in support of the view that the gouty diathesis is closely

* "Lectures on Bright's Disease," 1873, p. 64. See also the paper by the same author on "Latent Albuminuria," *British Medical Journal*, 1879, vol. ii, p. 928.

connected with one form of albuminuria, and this condition may either subside, leaving the kidneys sound, or may by its continuance give rise to organic renal disorder.

I now pass on to consider another form of renal lesion intimately connected with gout, viz., the gouty kidney, properly so called. The morbid anatomy of this condition has been already described (see p. 16). Of the two principal varieties, one is characterised mainly by deposits of uric acid and sodium urate scattered throughout the kidney ; while in the second, atrophy of the cortical substance and a granular condition of the kidney (renal cirrhosis) are associated with these infarctions.

These deposits and the cirrhotic state of the kidneys are often found in the later stages of gout, but it is certain that these organs are sometimes affected at an early period. The irritation due to the excretion of imperfectly metamorphosed substances may be the starting-point of a chronic inflammation, even before any deposit of sodium urate takes place in the kidney, and it would also seem highly probable that acute gout sometimes attacks the renal fibrous tissue before it appears in the joints. Cases of this kind have been described by Garrod and Charcot. Acute pain in

the lumbar regions and temporary albuminuria preceding the articular inflammation, are the principal symptoms; and it may reasonably be inferred that these are due to urate-deposits in the tubules and fibrous tissue.

It must be admitted that some authorities on renal pathology deny that these urate-deposits are necessarily connected with the gouty dyscrasia, mainly on the ground that they sometimes occur in the kidneys of persons who have never exhibited any gouty manifestations. Experience certainly teaches us that cirrhosis of the kidney is far from being peculiar to gout, though it is often found in connection therewith; but with regard to these urate-deposits, especially when found in contracted kidneys, there are strong grounds for believing, with Dr. Garrod, that they are nearly always indicative of gouty disorder within the affected organs. In gout, according to the same authority, the greater part of the deposit is interstitial; in non-gouty cases, the tubular structure is especially affected, and the crystals of uric acid and sodium urate are larger, but far less abundantly distributed. It is worthy of note that contracted kidneys, exhibiting whitish streaks of sodium urate in the pyramidal portions, are sometimes found in the bodies of persons

presenting no external manifestations of gout, but whose joints, when carefully examined, are found to be studded over with chalk-like concretions. Dr. Garrod refers to cases in which these infarctions were found in the absence of any visible deposits of chalk-stones, except one or more specks on the cartilage of the ear. There are likewise strong grounds for believing that this condition of the kidney is often present in subjects who have suffered from comparatively few attacks of articular gout.

This view of the relation of these infarctions and the cirrhosed state of the kidney to the gouty dyscrasia was strongly advocated by the late Dr. Todd.* In several of his cases the kidneys were found much contracted and wasted at the expense of the cortical substance, the cones in some places reaching almost the very surface of the organ; "in some of the cones there were *opaque streaks of deposit of urate of soda* taking the direction of the tubes, and probably occupying the canals of some of them." Dr. Todd also believed that renal cirrhosis (without infarctions of urates) is peculiarly apt to be developed

* "Clinical Lectures." Second edition, 1861. Edited by Dr. Beale. See especially Lecture xxviii on the Gouty Kidney.

in the inveterate gouty diathesis, though it may also occur in other states of the system.

From the account given in preceding paragraphs of the state of the kidney often found in connection with the gouty dyscrasia, it would seem advisable to restrict the use of the term "gouty kidney" to those cases in which infarctions of urates are present. The expression "gouty cirrhosis" should likewise be applied only to the granular and contracted kidney of *gouty* subjects. Renal cirrhosis often occurs independently of gout, and moreover, in rare instances gout is accompanied by amyloid and other changes in the kidney.

I do not think it necessary to describe in any detail the symptoms of gouty kidney. They are for the most part those of renal insufficiency. The urine is generally pale and of low specific gravity, and contains a diminished amount of urea and salts; the albumen is usually small in quantity, and at times scarcely detectible. Tube-casts, either granular or hyaline, are always present. The patient is generally anæmic and gradually loses flesh and strength, and the symptoms of dyspepsia become aggravated. Sooner or later there is puffiness of the eyelids, and also slight œdema of the feet and ankles, observable at first only at night; other forms of dropsy, such as ascites and

hydrothorax, are less common. As the disease advances, such indications of ill-health as anæmia, debility, shortness of breath, vomiting, &c., become more decided, but the progress is often very slow. Headache and dimness of vision (due to retinitis) are often present; symptoms of apoplexy or epilepsy occasionally supervene, and these signs of uræmia often usher in a fatal termination. In some cases they subside and recur from time to time, the patients feeling comparatively well during the intervals. An unfavourable termination is, however, always to be looked for, though its advent may generally be retarded by proper treatment.

I shall conclude this account of gouty affections of the kidney by referring to the occurrence of gravel, cystitis, and diabetes in gouty subjects.

Uric acid and urates are frequent deposits in the urine of gouty persons, and as these substances either form or enter into the composition of at least seventy-five per cent. of all calculi, it is reasonable to expect that the subjects of the gouty dyscrasia would be very prone to suffer from calculous disorders. As a matter of fact, gout and calculus sometimes co-exist, and cases are by no means rare in which persons who in early life have suffered from stone in the bladder are

attacked by gout at later periods. It must not, however, be inferred that the frequent appearance of uric acid and urates invariably presages the formation of a calculus, for many persons void these substances for lengthened periods, and yet never suffer from stone. I often meet with instances of this kind among persons hereditarily predisposed to gout. The form which the crystals take probably influences the formation of calculi; spiny clumps of uric acid are the most potent in this respect. Dr. Ord thinks that no calculus is formed of oxalates, urates and uric acid, or phosphates, without the intervention o fsome colloid substance, and that the most active matters in forming calculi are the exudation from the tubes and the mucus from the urinary tracts. The question of the origination of calculi* has not as yet been fully solved, and it would be foreign to my present purpose to discuss it any further; I would just remark, however, that calculi of oxalate of lime, or containing layers of this substance, are sometimes found in gouty subjects. I have at present under my care a lady, aged fifty-six, who has frequent attacks of gout, and occasionally

* For an exhaustive account of the latest views on this subject, see Dr. Dickinson's work on "Renal and Urinary Affections," Part III, p. 860, *et seq.*

passes small calculi of oxalate of lime. With regard to phosphatic calculi, these of course have no connection with gout, but are for the most part secondary formations due to decomposition of the urine.

Irritability of the bladder is a common symptom in gouty subjects, and is generally accompanied by scanty and highly acid urine, depositing uric acid and urates. Sometimes the irritability is attended by increased secretion from the mucous membrane of the urethra and burning pain in this part; and these symptoms occur in some persons shortly before an attack of gout. In other cases the vesical irritation accompanies the paroxysm; while in a third class the irritability is relieved or suspended on the outbreak of an acute attack.* Dr. Todd† has recorded a case in which vesical irritation rapidly supervened after an incision had been made into a gouty foot. He has also described cases of spasm and paralysis of the bladder in gouty subjects.

It would also appear that inflammation of the mucous membrane of the bladder may be due to gout.

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* See Coulson on "Diseases of the Bladder and Prostate Gland," sixth edition, p. 254.

† "Clinical Lectures," second edition, p. 563.

Sir James Paget* states that gouty cystitis may present all the ordinary characters of acute inflammation of the bladder from any other cause. Its gouty origin may be suspected if it has begun suddenly and after the use of indigestible food, or if it has been preceded by excess of urates in the urine. In some cases cystitis has been known to supervene on the disappearance of an eczematous eruption; Sir James Paget thinks that acute eczema of the mucous membrane of the bladder exists in such cases.

A close connection can sometimes be traced between diabetes and gout. In cases of gouty dyspepsia, the urine frequently contains traces of sugar, and among the sufferers from articular gout, diabetes occasionally supervenes and takes the place of the previous disorder, which is apt to recur when the amount of sugar and the quantity of urine become lessened. The two disorders may, however, co-exist. Diabetes occurring in gouty subjects is more amenable to treatment than when this latter dyscrasia is absent. Members of gouty families would seem to be peculiarly liable to diabetes; in some of these cases, regular gouty attacks come on, in others there are only slight symptoms.

* "Clinical Lectures and Essays," second edition, p. 377.

Charcot states that the children of diabetic subjects are predisposed to gout, and cites a remarkable instance of this nature. He mentions also another family in which the father was gouty, while gravel, diabetes, gout, and phthisis occurred among the children. The advocates of the neurotic origin of gout cite the frequent connection between the gouty dyscrasia and glycosuria as evidence in support of their views.

CHAPTER VII.

THE TREATMENT OF GOUT AND OF VARIOUS DISORDERS CONNECTED WITH IT.

The Treatment of the Gouty Diathesis—Evidence that the Gouty Diathesis may be Modified or Removed—Objects to be aimed at in the Treatment—Question of Diet for Gouty Subjects—Animal Food—Drawbacks attending excessive use of Vegetable Food—Quantity of Meat to be allowed—Other articles of food—Acids to be avoided—Farinaceous Food—Alcohol—Milk—Tea, Coffee, and Cocoa—Articles of Diet suitable for Gouty subjects—Question as to Gelatine—Quantity of Food—Importance of Diet-Rules—Printed Forms desirable—Production of Sense of Satiety to be avoided—Exercise for Gouty subjects—Horse Exercise—Walking—Passive Movements—The Zander Method—Fresh Air—Good influence of Sea Air—Mountain Air—Condition of Liver and Stomach—Action of Saline Purgatives—Various Mineral Purgative Waters, Friedrichshall, Pullna, &c.—Carlsbad Salts—Other Purgatives and Hepatic Stimulants—Alkalies—Mineral Waters of Bath, Buxton, Wildbad, Teplitz, Vichy, Baden, Wiesbaden, Aix-la-Chapelle, and Aix-les-Bains—Efficacy of Water as a Drink for Gouty subjects—The simple Thermal Waters—Alkaline Waters—Muriated Saline Waters—Turkish Baths—Avoidance of Excitement and a proper amount of rest necessary for Gouty subjects—The Treatment of an Acute Attack of Gout—Purgatives—Alkalies—Colchicum—

BELLADONNA AS A LOCAL APPLICATION—DIET AND REGIMEN —TREATMENT DURING THE INTERVALS—IODIDE OF POTASSIUM —ALKALIES—MINERAL WATERS—GUAIACUM—TONICS AS IRON, QUININE, &C. — LOCAL TREATMENT IN CHRONIC GOUT— CHALK-STONES AND GOUTY ULCERS—TREATMENT OF GOUTY DISORDERS OF THE LIVER AND KIDNEYS AND OF GOUTY CUTANEOUS AFFECTIONS.

I PROPOSE in this, the concluding chapter of my work, to describe the treatment, first, of the gouty diathesis; secondly, of an acute attack of articular gout; and thirdly, of the more important of those disorders which are the direct results of the gouty dyscrasia.

I. *The Treatment of the Gouty Diathesis.*—Experience teaches us that the gouty diathesis may be decidedly modified or even altogether removed, and that these objects can be effected even after several acute attacks have actually occurred. The case of the late Dr. Gregory, of Edinburgh, has been often cited in proof of this assertion. Dr. Gregory was descended from a decidedly gouty family; before he was thirty years of age he had had several severe attacks of acute gout, and occasional gastric spasms due to the same cause. By taking active exercise, avoiding all excesses, and keeping to a strictly moderate diet (although he did not abstain from animal food) during a period of twenty years, he so completely overcame the disposi-

tion to the disease that all symptoms of it disappeared in the latter part of his life. Dr. Todd, in referring to the above case, very justly remarks that "there is no disease in which the patient can do so much for himself, or in which the prescriptions of the physician are of so little avail without the full and complete co-operation of the patient, as in gout.*

Sufficient evidence has already been adduced in support of the view I have adopted as to the nature and cause of the gouty diathesis, viz., that excessive formation of uric acid is the condition which underlies gouty manifestations of all kinds. It follows that for the rational treatment of this diathesis our object should be to check the production of this *materies morbi* and to promote its elimination from the system. The attainment of these ends is the problem to be solved.

It has been shown in preceding chapters that excessive formation of uric acid and its accumulation in the blood are for the most part due to defective metamorphosis of the albuminous elements of food, and that the liver is the organ which is mainly at fault. In order, therefore, to combat the symptoms of

* "On Gout and Rheumatism," p. 77.

the uric acid diathesis, the diet and the condition of the liver are the first subjects which require attention.

With regard to the diet, an all-important subject in the treatment of any form of gout, I propose only to lay down a few general principles, because in dealing with this disease it is absolutely necessary to make a special study of each patient. Certain maxims, however, apply with greater or less force to all cases. The primary indication is to supply a sufficient amount of those albuminous substances which are readily utilised and metamorphosed in the system.

A certain proportion of albuminous constituents is necessary for the wants of the organism, and those systems of treatment which are based upon almost complete abstinence from *animal* food have never been found serviceable for gouty cases. Vegetable food in quantity sufficient to contain the requisite amount of albumen will furnish at the same time an excessive proportion of carbo-hydrates, which being more easily oxidised, and therefore more readily consumed in the system than the albuminous compounds, will, to a considerable extent, spare or prevent the disintegration and oxidation of the latter. It is moreover probable that vegetable albumen less readily undergoes disintegration than albumen of animal

origin. In gout the disintegrative changes in the albuminates are arrested, and insufficiently oxidised substances remain in the blood. Under ordinary circumstances, when a diet is taken in which animal food preponderates, a larger amount of oxygen is retained in the system than when amylaceous food s in excess.

The result of the above considerations would appear to be that meat is not to be forbidden to gouty patients; though, as a matter of course, certain restrictions must be laid down as to its use. The following are the chief points to be attended to in connection with the diet :—

1. The quantity of the meat to be allowed.
2. The quantity and quality of other articles of food usually taken with meat, and which lessen or prevent the oxidation of albuminous substances.
3. The avoidance of acids and of acid-forming substances, the operation of which is to diminish the alkalinity of the blood and juices of the tissues, and thus to favour the precipitation of the urates and their retention in the system.

The quantity of meat and of albuminates in general to be allowed to subjects of the uric acid diathesis should be in strict proportion to the wants of the

system, and this limitation should never be exceeded. Physiology teaches us that about twelve ounces of meat and two pounds of bread are amply sufficient to compensate the daily losses of the system of a healthy man. For gouty subjects a far smaller proportion of carbo-hydrates is advisable, and of these latter substances, sugar and starch, having a greater affinity for oxygen, would appear to be even more prejudicial than fat.

For the reasons above given, farinaceous food, such as bread, rice, potatoes, &c., should be used very sparingly; pastry of all kinds should be strictly forbidden. A little fruit may be allowed, provided that no undue amount of acid or of saccharine materials is thereby introduced into the system. Both theory and experience teach us that alcohol in any form should be avoided by gouty subjects; it undergoes oxidation in the system and checks metamorphosis. Plain pure water, aërated water, or some of the alkaline acidulous waters, form the best fluids for gouty subjects. In cases, however, in which the digestion is feeble, a small quantity of alcohol is sometimes beneficial. Old whisky or brandy is probably the least injurious form of alcoholic stimulant; a tablespoonful diluted with six ounces of water should be the maximum

quantity allowed. If wine be preferred, sound claret and hock are best suited for gouty patients. One, two, or even three wineglassfuls may be permitted according to circumstances. Imperfectly fermented and effervescing wines, and malt liquors of all kinds, should be strictly forbidden. Acids and all substances likely to undergo acid fermentation must be interdicted. Acids when absorbed into the blood lessen its alkalinity, inasmuch as they combine with the alkalies they meet with, and set free weaker acids. With the object of preventing scorbutic symptoms, I sometimes allow gouty persons, for whom vegetables would be injurious, to take a little lime-juice, well diluted with water.

There is some difference of opinion with regard to the effect upon gouty subjects of a diet composed largely of milk. When large quantities of milk are taken, the general result is an increased production of uric acid, and this fluid would therefore appear to be unsuitable for such patients. Experience, however, shows that some gouty subjects can take one or two pints of milk in the twenty-four hours with apparent benefit. The prejudicial effects of milk would appear to be due to the lactic acid which results from the fermentation of the milk sugar. It is worthy of note

that the administration of lactic acid to gouty subjects causes the attacks to become not only more frequent, but also more severe and obstinate. Moderation should be observed in the use of tea and coffee; the former especially should be avoided whenever flatulent dyspepsia is a prominent symptom. Cocoa is often preferable for these patients. The nibs should be coarsely powdered and boiled for two or three hours; the liquor is then strained, and the fat, which rises to the surface, removed when cold.

From what has been just stated it will be evident that only a few articles of diet can be safely recommended to the subjects of the uric acid diathesis. Mutton, beef, chicken, game, fish, eggs, and green vegetables (including cress, lettuce, &c.), constitute the list of articles from which a selection must be made. A few ounces of stale bread and a small quantity of butter may be allowed. A diet thus restricted will furnish the necessary amount of albuminates, and the articles are easy of digestion and not likely to set up acid fermentation in the stomach. Substances containing much gelatine should be avoided. Gelatine when introduced into the system appears to undergo a decomposition analogous to that of the albuminous compounds; the ingestion

of large quantities is followed by a marked increase in the proportion of urea in the urine, with decided elevation of the specific gravity. In non-gouty subjects the uric acid is not increased in quantity, but it is probable that where metamorphosis is imperfect, other substances, and uric acid among the number, would be formed in place of urea.

It is not sufficient to lay down rules as to the *quality* of the food; the *quantity* is a point of no less importance. I have for some time past been in the habit of supplying my patients with printed "diet-rules." I keep at hand a set of forms on which the hours for meals, and the articles that *may* be taken and those that *must* be avoided, are clearly specified. As a matter of course, modifications, by way of addition or subtraction, are sometimes required, and spaces are therefore left for additions. I attach the greatest importance to these diet-rules; a patient is far more likely to obey instructions contained in a printed form than verbal directions, however emphatically expressed. To the list of articles I have appended a caution with regard to the ill effects of rapid eating. Gouty patients, above all others, should exercise great moderation in the quantity of food taken at each meal. A positive sense of satiety

should not be experienced, for, as justly remarked by Dr. Beaumont, "this is beyond the point of healthful indulgence, and is Nature's earliest indication of an abuse and over-burden of her powers to replenish the system." That which ought to be attained "occurs immediately previous to this, and may be known by the pleasurable sensations of perfect satisfaction, ease, and quiescence of body and mind. It is when the stomach says, *enough;* and it is distinguished from satiety by the difference of sensations—the latter saying *too much.*" When, as not unfrequently happens, corpulence is associated with gout, the quantity of all articles of diet must be uniformly reduced; that of the albuminous kinds should be considerably diminished, for no one becomes corpulent without taking albumen, in some form or other, in excess of the requirements.

Exercise and abundance of fresh pure air come next to diet in the treatment of the gouty diathesis. Exercise promotes tissue-change and the oxidation of the constituents of the body, and many a gouty patient of lazy, indolent habits would get rid of his complaint if he were to become a farm labourer or a postman. Walking exercise for several hours a day should, whenever possible, be enjoined. Horse exercise, "the Palmerstonian cure for gout," as one

German writer terms it, is also very advantageous, and should be tried whenever circumstances will admit. Muscular exercise promotes the consumption to some extent of albuminates, and to a far greater degree of glycogen and sugar. Mental activity in moderation is also desirable, for it too promotes tissue-change. When patients are unable to take active exercise by reason of gouty deposits, stiffness, and pain in the joints, some forms of passive movement, with friction, shampooing, &c., should be recommended. Dr. Zander, of Stockholm, has invented a series of machines by means of which the several muscles of the body can be brought into play, and thus a considerable amount of exercise can be taken without undue fatigue. Shampooing machines for the legs and abdomen, and friction machines for the hands and feet, are also to be found at the Zander Institute.* The amount of exercise, whether natural or artificial, must be carefully adapted to the circumstances of the case; anything approaching to undue fatigue must be avoided. Acute attacks may not

* For details of Dr. Zander's system the reader is referred to a little work entitled "Mechanical Exercise a Means of Cure," being a description of the Zander Institute, London, its History, Appliances, &c. London: J. & A. Churchill. 1883.

unfrequently be traced to over-exertion. In connection with this subject it must be kept in mind that a certain interval should always elapse between exercise and meals. Over-fatigue is a fertile source of indigestion, and to sit down to a meal immediately after exercise is not conducive to normal digestion.

Fresh air is especially indicated for gouty subjects; but exposure to damp and cold should be carefully avoided. Sea-air often causes a marked improvement in these cases, and should always be tried. It promotes in an especial manner the oxidation and transformation of albuminous tissues. The bracing air of mountainous districts is beneficial for many patients; on the other hand, cases of confirmed gout often require a considerable amount of warmth.

Attention to the functions of the skin is all-important for gouty subjects. I shall have more to say on this subject when describing the various baths that may be used.

In gouty subjects the condition of the stomach and liver, and of the whole intestinal tract, is second in importance only to the question of diet. I have already explained that frequent deposits of uric acid and urates in the urine are generally due to hepatic derangement, for the relief of which it is useless to

prescribe merely alkalies and diuretics, remedies which aid the elimination of uric acid, but do not prevent its formation. Various medicines act more or less decidedly upon the liver, and in cases of functional derangement purgatives are very useful. Saline aperients are those which are generally indicated. They act promptly, and not only remove the contents of the bowels, but cause a decided drain from the intestinal vessels and relieve any congestion which exists in the portal system. Opinions differ as to the way in which these medicines produce their purgative effect. Liebig's theory was that an osmotic current takes place *from* instead of *towards* the circulatory system. Active purgation, however, may be produced in animals by injecting certain saline solutions into the veins. It would appear more probable that these solutions act by preventing the absorption of the secretions which are constantly poured out from the mucous membrane of the intestine, and are taken up by the veins and lymphatics. The salts best adapted for cases of lithæmia are the sulphates of soda and magnesia (the latter I often prescribe in combination with sulphate of quinia), the phosphate of soda and the tartarated soda. These may be given in the ordinary manner,

but for many cases the best way of exhibiting them is in the form of some one or other of such mineral waters as Friedrichshall, Püllna, Kissingen, Æsculap, or Hunyadi Yànos. The dose must be regulated according to the circumstances of the case; from two to five or six fluid ounces may be required. It is generally advisable to add an equal quantity of hot water, and the medicine should be taken about half-an-hour before breakfast. The quantity should be sufficient to produce one or two evacuations without griping or much discomfort.

When symptoms of gastric catarrh, fermentation, and acidity exist, Carlsbad water is preferable. The principal salts contained in the water of the Sprudel spring at Carlsbad are the sulphate and carbonate of soda and chloride of sodium. Carlsbad salts obtained by evaporating the water can be procured in the solid form, and when dissolved in a suitable quantity of water are very efficacious. About a teaspoonful should be dissolved in half a pint of boiling water, and when the solution has cooled down to about 120°, two or three ounces should be taken every five minutes. This treatment may be continued for several weeks, if necessary; the solution being taken every morning or every other morning, according to circumstances. The

quantity of the salt should be increased if the bowels are not sufficiently acted upon ; or a small dose (gr. j.) of the extract of aloes may be taken before dinner.

Other purgatives and certain hepatic alteratives are often useful in the treatment of the uric acid diathesis. Small doses of calomel or blue pill, either alone or combined with colocynth or rhubarb, may be given from time to time. When there is marked congestion of the liver a full dose of calomel (three or four grains), followed after two hours by an ounce of Mist. Sennæ Co., or a few ounces of one of the saline waters, will be the best plan of treatment. Various other hepatic stimulants, as podophyllin, euonymin, iridin, and leptandrin, are available for use in chronic cases. Deposits of oxalate of lime are often found associated with lithates, and when this condition exists the nitro-muriatic acid combined with the tincture of nux vomica and henbane, and taken before meals, will often cause both forms of deposit to disappear from the urine. As a general rule, acids are contra-indicated in gouty cases, but where *chronic* congestion and torpor of the liver exist, a course of nitro-muriatic acid is almost always beneficial. When the urine contains much free uric acid, and exhibits a marked acid reaction, alkalies are generally indicated,

and of these some preparation of potash or lithia is to be preferred. The alkalies facilitate the elimination of uric acid, but, unlike remedies which promote the hepatic functions, they do not prevent its formation.* The citrate of potash is a convenient preparation: a few doses ordinarily suffice to diminish the acidity of the urine.

In addition to those which are more or less purgative in their action, certain mineral waters have a widespread reputation in the treatment of the gouty diathesis. Among the most celebrated are the waters of Bath and Buxton in this country, and of Wildbad, Teplitz, Vichy, Baden, Wiesbaden, Aix-la-Chapelle, and Aix-les-Bains on the continent. The first four are simple thermal waters—that is, their natural temperature is high, but they contain only small proportions of mineral matters. The waters of Vichy are decidedly alkaline owing to the presence of carbonate of soda; those of Baden and Wiesbaden belong to the muriated saline class, chloride of sodium being the principal saline constituent. The

* Dr. Murchison attributes to alkalies the power of combating the pathological condition which leads to the undue formation of lithic acid. He regards them as promoters of oxidation and disintegration of tissue. It is, however, by no means certain that alkalies (*e.g.*, liquor potassæ) increase the amount of urea in the urine.

springs of Aix-la-Chapelle and Aix-les-Bains yield water containing sulphuretted hydrogen. The water of the former place contains also a marked quantity of common salt. All these waters are taken internally and also used for baths.

There can be no doubt as to the efficacy of water as a drink for gouty subjects, and as it is generally difficult to persuade people to make use of what is close at hand, it is often expedient to advise a journey and a stay for several weeks at some one or other of these baths. The action of the simple thermal or indifferent waters is neither greater nor less than that of any ordinary pure water heated to the requisite degree. Water taken internally washes out the stomach, augments secretion, promotes the transformation of tissue, removes waste products from the blood, and thus fulfils many of the indications for the treatment of gout. Hot water, especially if taken slowly, is rapidly absorbed by the blood-vessels, and is tolerated without difficulty by the stomach.

When used as baths, the simple thermal waters produce effects similar to those of ordinary warm baths, and depending mainly on the temperature of the water, and the time spent in the bath. The skin is softened and cleansed, its circulation is

accelerated, and on leaving the bath the action of the cutaneous glands is considerably heightened.

The simple alkaline waters, as those of Vichy, have an antacid and diuretic effect, and their internal use serves to render uric acid more soluble and thus promotes its elimination. It is doubtful whether any saline matters are absorbed when these waters are used for baths. Some observers state that the urine becomes alkaline after a bath of Vichy water. The same result, however, has been noticed after an ordinary warm bath, and is probably due to the fact that an increased amount of acid leaves the system through the cutaneous perspiration.

The use of the muriated saline waters (which contain chloride of sodium) promotes tissue-change, checks acid fermentation, and accelerates the osmotic circulation in the bowels. When used as baths, these waters stimulate the skin and improve its nutrition. It is difficult to believe that any definitely good effects can result from the sulphur waters.

As remedies for gout, little if any permanent benefit can be expected to arise from the mere use of any of the above-mentioned waters, though they are often valuable adjuvants to more rational plans of treatment. A course at such places as Vichy, Bath,

or Buxton, will no doubt relieve many of the symptoms, but the improvement will be only of a temporary character. On the other hand, permanent good is likely to result from the internal use of those waters which contain the sulphates and chlorides and a small proportion of the alkalies. These waters are especially indicated in cases in which abundant formation of uric acid is associated with corpulence and signs of plethora. For weakly persons their occasional use will often prove beneficial, but a course of the muriated alkaline waters or of the simple thermal waters is generally more suitable. One advantage connected with a course of treatment by baths and waters is due to the fact that, while it is going on, patients are generally very careful in obeying all directions with regard to diet and regimen. In some cases the good habits thus acquired become permanent. Equal results might doubtless be very often obtained under similar treatment and regimen at home; but such a method fails to commend itself to most people by reason of its simplicity and the comparative facility with which it might be carried out.

After a bath of any kind, the skin should be thoroughly dried and well rubbed with a coarse towel.

The use of a flesh-brush is also very advantageous. Turkish baths are not suitable for the majority of gouty subjects; they are sometimes useful for young and plethoric persons with rough and coarse skins and deficient perspiration. They are also beneficial in a small number of chronic cases, provided that there is no cardiac complication. They should not be too frequently taken, as they are apt to cause debility.

Avoidance of undue excitement and a proper amount of rest are the last points to which I shall refer in connection with the hygiene of the gouty dyscrasia. Nervous exhaustion, from any cause, much intensifies the action of the other factors of gout, and all possible care should therefore be taken to prevent its occurrence. Late hours are especially injurious, and the patient's habits as regards sleep should always be inquired into. Too little sleep is as mischievous as too much; it is a fertile source of nervous exhaustion. In all cases of sleeplessness, the physician should direct his attention to the condition of the stomach, liver, and kidneys: he will often find that the discomfort is due to functional disorder of some one or all of these organs.

I shall add a few more remarks on the medicines

suitable for gouty subjects after a brief description of the treatment of an ordinary acute attack.

II. *The Treatment of an Acute Attack of Articular Gout.*—When called to a patient suffering from an acute attack, I invariably examine the urine for albumen. If this latter be absent, and if there be constipation and signs of congestion of the liver, two or three grains of calomel, followed by a draught containing sulphate and carbonate of magnesia, may be prescribed with advantage. If there be no marked evidence of hepatic congestion, milder remedies will suffice to relieve any constipation that may be present. The saline draught alone, or ten grains of Pil. Colocynth. et Hyoscyami, with perhaps a grain of calomel or a quarter of a grain of Resin. Podophylli will generally produce a free action of the bowels. At the same time I prescribe a mixture with Vin. Colchici ℥ x., and grs. x.—xx. of some alkaline salt, such as the bicarbonate of potash or soda, the carbonate of magnesia, or the citrate of magnesia or potash. This should be taken four times in the twenty-four hours, and continued according to circumstances. If there be much fever, Liq. Ammon. Acetat. ʒij. may be added to each dose.

For the relief of the pain I find great benefit from

the local application of belladonna in the following form:—R. Extract. Belladonnæ ʒij., Glycerini ℥ss., Aquæ ʒij. A sufficient quantity of this mixture is placed upon cotton wool and applied to the affected joint, which should be raised upon a pillow and kept in the position most conducive to the patient's comfort. As a matter of course, while active symptoms continue, the patient should be restricted to such articles of diet as arrowroot, sago, gruel, milk puddings, &c. Seltzer, Apollinaris, or other aërated alkaline water may be freely allowed. When the acute symptoms have subsided, beef-tea, fish, and chicken may be taken in small quantities; the return to ordinary diet should be very gradually effected. Rest and care are essential for some days after the subsidence of the paroxysm. The application of leeches, blisters, or cold in any form to the affected joint is always to be deprecated. Cold applications, indeed, are highly dangerous (see p. 79).

With regard to the use of colchicum in gout, this drug has been proved not to increase the elimination of uric acid, and there is no evidence of lessened formation of this substance under its use. On the other hand, it certainly exerts a specific influence on gouty pain and inflammation, and the results are clearly

not due to its action as a purgative or a sedative. I prefer to use it in small doses (♏ x.—xv. of the Vinum Colchici) at intervals of four or six hours. It is sometimes useful in chronic gout, but far less frequently than in the acute attacks. For weakly subjects, colchicum is almost always contra-indicated. When aperients are required, I sometimes order small doses of the acetous extract in combination with colocynth and henbane.

In the intervals between the paroxysms much may be done to prevent recurrences, proper diet and regimen being far more efficacious in this respect than any form of medicine. The directions already laid down with regard to food, fresh air, exercise, attention to the functions of the skin, bowels, &c., should be fully explained to the patient, and he should be warned that if these are neglected, neither drugs nor mineral waters will prove of any avail. Provided that due attention be paid to these subjects, various medicines will help to combat the gouty tendencies and to prevent further attacks, or at least to diminish their frequency and severity.

Iodide of potassium is often serviceable for these purposes; it is of course especially indicated whenever gout is complicated by lead-poisoning. Its

good effects are often well marked in other cases; it may be advantageously combined with bicarbonate of potash in some bitter infusion. Various alkaline remedies are generally useful, their main action being to facilitate the excretion of uric acid. Potash and lithia are the alkalies which form the most soluble salts with uric acid. These should be given for lengthened periods (four to eight weeks) in small doses, and in very dilute solutions. The mineral waters, already mentioned, may be substituted for these simpler methods of exhibiting the drugs. The ammoniated tincture of guaiacum is a very valuable remedy in many cases of chronic gout. This drug stimulates the action of the skin and kidneys; it is especially adapted for chronic atonic cases. If thought desirable, the iodide of potassium and the bicarbonate of potash may be given in combination with it. Guaiacum should not be given when there are any decided inflammatory symptoms.

Various tonics and stomachics are often required for gouty subjects. Iron is almost always useful whenever there is marked anæmia. I generally prescribe the tincture of the acetate for such cases. The state of the bowels should always be attended to when iron is being administered. An atonic condition of

the stomach may be improved by quinine and other bitters. Quinine is also valuable in checking fermentation in the stomach and intestines.

With regard to the local treatment in chronic gout affecting the joints, the application of the tincture of iodine generally affords the most relief to pain and swelling; mercurial plasters may also be tried. Gentle friction with various liniments, and carefully applied pressure by means of bandages, are often beneficial. The bandages should not, however, be continuously applied; on the other hand, movement should be encouraged from time to time. Œdema of the limbs may be relieved by friction, warm douches, bandages, and the adoption of an elevated position. It is connected sometimes with phlebitis and sometimes with renal disease, and in either case is apt to be very troublesome. Chalk-stones, when prominent, should be protected from injury. Much good may be done by the continuous application of alkaline solutions, and especially of a solution of carbonate of lithia (grs. v. to ʒj.), as recommended by Dr. Garrod. A piece of lint is soaked in this fluid, and applied to the joint and then covered with oiled silk or gutta-percha tissue. The addition of an equal quantity of iodide of potassium increases the efficacy of the

solution, especially when there is much inflammatory thickening round the joint. Under similar circumstances, warm douches, passive movements, shampooing, &c., are likely to be serviceable. The removal of chalk-stones, through incisions into the skin, is fraught with danger; erysipelas is very likely to supervene. Gouty abscesses may be very carefully punctured: if ulcers form, they should be protected from injury, and treated in the ordinary way.

III. *The Treatment of Gouty Disorders of the Liver and Kidneys and of Gouty Cutaneous Affections.*—Gouty inflammation of the biliary ducts should be treated by poultices to the abdomen and warm baths, with a little blue pill and the sulphate of soda or magnesia as a purgative. Alkalies, such as the citrate or bicarbonate of potash, are afterwards indicated. The affection of the biliary ducts is generally preceded by catarrh of the stomach and duodenum. The diet must be carefully regulated.

Gouty dyspepsia is best treated by mild purgatives, such as the Carlsbad salts, and by effervescing alkaline mixtures. With regard to diet, meat should be allowed only once daily, and alcoholic liquids, sugar, and starchy articles of food should be forbidden.

The hygienic treatment of the uric acid diathesis must of course be followed out.

Cirrhosis of the liver occurring in gouty subjects requires the ordinary treatment. With regard to diet, it is not enough to prohibit the use of alcohol, inasmuch as in all probability the disease is sometimes due to the action upon the liver of other irritants, such as ill-digested food, &c.

In the treatment of gouty disorder of the kidneys, the urine should be carefully examined for albumen, tube-casts, and sugar. If the secretion be found normal in these respects, any symptoms referable to the kidneys, such as pain in the loins, frequent micturition, and a highly concentrated and acid state of the urine, will be best treated by saline purgatives, followed by citrate of potash in an effervescing solution. Warm baths, warm fomentations, or mustard plasters to the loins will relieve the pain, and any renal congestion that may be present.

If the urine contains albumen, but no casts, the significance of the albumen is the question to be decided. As mentioned in a previous chapter (see p. 46) albuminuria of this kind not unfrequently co-exists with symptoms of gouty dyspepsia, and is due, I believe, to malassimilation of food. It subsides

under the treatment already indicated; but it must not on that account be regarded as an unimportant symptom, for if neglected it may set up serious disease of the kidneys.

If the albuminuria recur from time to time in a gouty subject, and if casts of the tubes appear, there can be no doubt as to the existence of a renal lesion. The albumen may be in very small quantities, and at times even absent; the casts are either hyaline or granular. Other symptoms, already described, gradually supervene, and the disease generally runs a protracted course.

The treatment is that of the cirrhotic form of Bright's disease; special attention must of course be paid to the dyscrasia to which the renal affection is due. The measures recommended for dealing with the gouty dyscrasia and for chronic gout must be rigorously enforced. Suitable diet, exercise short of fatigue, a proper amount of rest, attention to the state of the bowels, and to that of the skin, will tend to delay the advent of untoward symptoms. Counter-irritation to the kidneys and hot air baths or vapour baths are valuable remedies in this class of cases. Alkalies are generally indicated, but should be used with caution in cases in which the eliminating powers

of the kidneys are defective. Iodide of potassium is sometimes useful, and should always be given when lead-poisoning is suspected. Opium in any form is of course quite out of place. Patients with this condition of the kidneys are peculiarly susceptible to the action of this drug. When the disease is well marked, a nourishing diet and iron are generally indicated. Warmth and rest of body and mind are absolutely essential.

The treatment of gouty cutaneous affections can be summed up in a few words. Eczema and psoriasis always require constitutional remedies; the local treatment is of subordinate importance. The diet and regimen should be carefully attended to; over-indulgence in animal food should be strictly prohibited, and any co-existing dyspepsia must be dealt with as already described. When the skin-affection is associated with plethora, saline purgatives and alkalies are indicated; when there is debility, the digestion should be aided by vegetable bitters and other tonics. Remedies which stimulate the hepatic functions are generally serviceable. The action of the skin should be promoted by vapour baths or hot air baths. Arsenic may be tried should the skin-affection prove very obstinate. As local remedies, in the early stage of

eczema, a lotion containing bromide of potassium grs. xv. to ℥j.) will often relieve the itching; in later) stages, the white precipitate ointment, zinc ointment, and various preparations of tar are likely to be serviceable. It must be borne in mind that eczema in gouty subjects is very apt to recur, especially in the spring of the year.

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